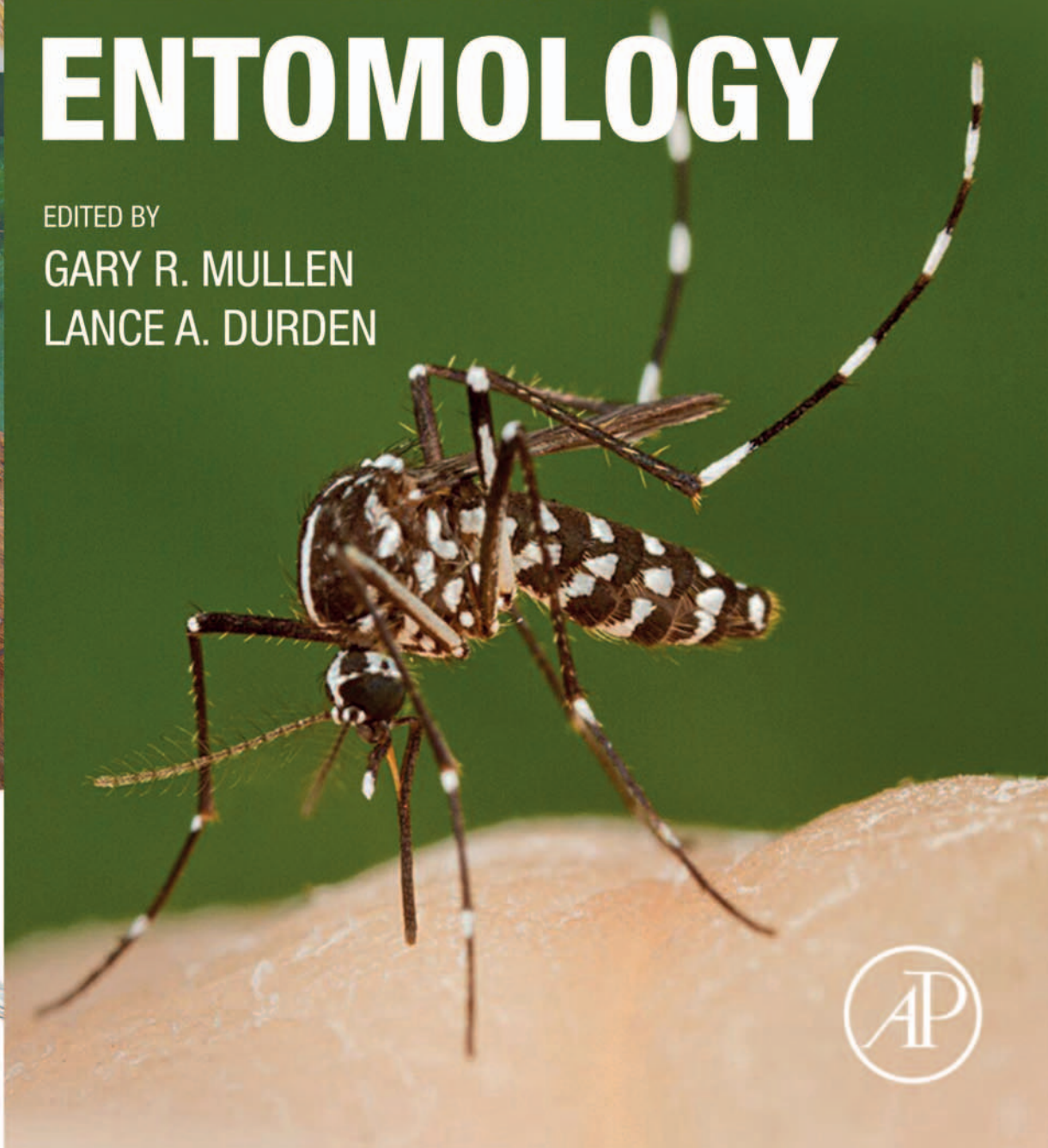
THIRD EDITION

MEDICAL AND VETERINARY ENTOMOLOGY

EDITED BY
GARY R. MULLEN
LANCE A. DURDEN



Medical and Veterinary Entomology

This page intentionally left blank

Medical and Veterinary Entomology

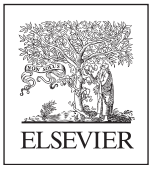
Third Edition

Edited by

Gary R. Mullen
Department of Entomology and Plant Pathology, Auburn University, Auburn, AL, United States

Lance A. Durden
Department of Biology, Georgia Southern University, Statesboro, GA, United States

Jonas G. King, Graphics Assistant
Department of Biochemistry, Molecular Biology, Entomology and Plant Pathology,
Mississippi State University, Starkville, MS, United States



ACADEMIC PRESS
An imprint of Elsevier

Academic Press is an imprint of Elsevier
125 London Wall, London EC2Y 5AS, United Kingdom
525 B Street, Suite 1650, San Diego, CA 92101-4495, United States
50 Hampshire Street, 5th Floor, Cambridge, MA 02139, United States
The Boulevard, Langford Lane, Kidlington, Oxford OX5 1GB, United Kingdom

Notices
Knowledge and best practice in this field are constantly changing. As new research and experience broaden our understanding, changes in research methods, professional practices, or medical treatment may become necessary.

Practitioners and researchers must always rely on their own experience and knowledge in evaluating and using any information, methods, compounds, or experiments described herein. In using such information or methods they should be mindful of their own safety and the safety of others, including parties for whom they have a professional responsibility.

To the fullest extent of the law, neither the Publisher nor the authors, contributors, or editors, assume any liability for any injury and/or damage to persons or property as a matter of products liability, negligence or otherwise, or from any use or operation of any methods, products, instructions, or ideas contained in the material herein.

Library of Congress Cataloging-in-Publication Data
A catalog record for this book is available from the Library of Congress

British Library Cataloguing in Publication Data
A catalogue record for this book is available from the British Library

ISBN: 978-0-12-814043-7

Publisher: Charlotte Cockle
Senior Acquisition Editor: Anna Valutkevich
Editorial Project Manager: Pat Gonzalez
Production Project Manager: Punithavathy Govindaradjane
Designer: Miles Hitchen

Typeset by TNQ Technologies

Contents

Contributors xv
Preface xvii
Acknowledgments xix

1. Introduction

Lance A. Durden and Gary R. Mullen

General Entomology 1
Medical-Veterinary Entomology Literature 2
A Brief History of Medical-Veterinary Entomology 2
Identification and Systematics of Arthropods of Medical-Veterinary Importance 4
Types of Problems Caused by Arthropods 4
- Annoyance 4
- Toxins and Venoms 5
- Allergic Reactions 5
- Invasion of Host Tissues 6

Arthropod-Borne Diseases 6
Food Contaminants 6
Fear of Arthropods 7
Delusional Disorders 7
Formicophilia 8
Host Defenses 8
Minor Arthropod Problems of Medical-Veterinary Interest 9
References and Further Reading 10

2. Morphological Adaptations of Parasitic Arthropods

Nathan D. Burkett-Cadena

Body Shape and Wings 17
Mouthparts 17
Legs 19
Sensory Structures 21
References and Further Reading 22

3. Arthropod Toxins and Venoms

Justin O. Schmidt

Nature of Toxins and Venoms and Their Effects 24
Arthropod Venoms as Antimicrobial, Analgesic, and Other Therapeutic Agents 28
References and Further Reading 29

4. Epidemiology of Vector-Borne Diseases

Christopher M. Barker and William K. Reisen

Components of Transmission Cycles 34
- Host Immunity 35
- The Vertebrate Host 35
- The Arthropod Vector 36

Modes of Transmission 37
- Vertical Transmission 37
- Horizontal Transmission 38

Transmission Cycles 40
Interseasonal Maintenance 41
Vector Incrimination 42
Surveillance 43
Emerging Vector-Borne Diseases 47
- New Tools 49

References and Further Reading 49

5. Forensic Entomology

William L. Krinsky

History 51
Legal Cases Involving Liability 52
- Structural Entomology 52
- Stored-Product Entomology 52
- Occupational Hazards Associated With Arthropods 52
- Veterinary and Wildlife Entomology 52

Legal Cases Involving Homicides, Suspicious and Accidental Deaths, and Abuse 53

Sudden Death With Arthropod Association 53
Automobile-Accident Death 53
Arthropods as Signs of Neglect or Abuse or as Agents of Murder 53
Illicit Drug Transport, Use, and Overdose 53
Suspicious Deaths 54
Stages of Decomposition 54
Insect Succession and Postmortem Interval 55
References and Further Reading 59

6. Cockroaches (Blattaria)

Richard J. Brenner and Richard D. Kramer

Taxonomy 61
Morphology 61
Life History 63
Behavior and Ecology 64
Common Cockroach Species 65
Oriental Cockroach *(Blatta orientalis)* 65
Turkestan Cockroach *(Blatta lateralis)* 65
American Cockroach (*Periplaneta americana*) 65
Australian Cockroach (*Periplaneta australasiae*) 66
Brown Cockroach (*Periplaneta brunnea*) 66
Smokybrown Cockroach (*Periplaneta fuliginosa*) 67
Florida Woods Cockroach (*Eurycotis floridana*) 67
Brown-Banded Cockroach (*Supella longipalpa*) 68
German Cockroach (*Blattella germanica*) 68
Asian Cockroach (*Blattella asahinai*) 69
Surinam Cockroach (*Pycnoscelus surinamensis*) 69
Public Health Importance 70
Pathogenic Agents 70
Intermediate Hosts 72
Cockroach Allergies 73
Veterinary Importance 74
Prevention and Control 75
Sanitation 75
Harborage Elimination 75
Physical Control 75
Biological Control 75
Insect Growth Regulators 76
References and Further Reading 76

7. Lice (Phthiraptera)

Lance A. Durden

Taxonomy 79
Morphology 80
Life History 83
Behavior and Ecology 84
Lice of Medical Importance 86
Human Body Louse (*Pediculus humanus humanus*) 87
Human Head Louse (*Pediculus humanus capitis*) 88
Human Crab Louse (*Pthirus pubis*) 88
Lice of Veterinary Importance 89
Lice of Cattle 89
Lice of Other Livestock Animals 90
Lice of Cats and Dogs 92
Lice of Laboratory Animals 92
Lice of Poultry and Other Birds 93
Public Health Importance 94
Epidemic Typhus 95
Louse-Borne Relapsing Fever 96
Trench Fever 97
Other Pathogens Transmitted by Human Body Lice 98
Lice as Intermediate Hosts of Tapeworms 98
Veterinary Importance 98
Lice of Livestock 98
Lice of Wildlife 101
Lice of Cats and Dogs 101
Lice of Laboratory Animals 102
Lice of Poultry and Other Birds 102
Prevention and Control 102
References and Further Reading 104

8. True Bugs (Hemiptera)

William L. Krinsky

Kissing Bugs (Reduviidae) 109
Taxonomy 109
Morphology 109
Life History 111
Behavior and Ecology 111
Public Health Importance 112
Veterinary Importance 118
Prevention and Control 118
Bed Bugs (Cimicidae) 119
Taxonomy 119
Morphology 119
Life History 120
Behavior and Ecology 121
Public Health Importance 122
Veterinary Importance 123
Prevention and Control 124
References and Further Reading 124

9. Beetles (Coleoptera)

William L. Krinsky

Taxonomy 129
Morphology 129

Life History 130
Behavior and Ecology 130
Public Health Importance 131
Meloidae (Blister Beetles) 133
Oedemeridae (False Blister Beetles) 134
Staphylinidae (Rove Beetles) 134
Tenebrionidae (Darkling Beetles) 135
Dermestidae (Larder Beetles) 137
Scarabaeidae (Scarab Beetles) 137
Coccinellidae (Lady Beetles) 137
Veterinary Importance 138
Ingestion of Toxic Beetles 138
Transmission of Pathogens 138
Intermediate Hosts of Parasites 139
Nest Associates and Ectoparasites 140
Dung Beetles and Biocontrol 140
Prevention and Control 141
References and Further Reading 141

10. Fleas (Siphonaptera)

Lance A. Durden and Nancy C. Hinkle

Taxonomy 145
Morphology 146
Life History 148
Behavior and Ecology 149
Fleas of Medical–Veterinary Importance 150
Human Flea (*Pulex irritans*) 150
Cat Flea (*Ctenocephalides felis*) 151
Dog Flea (*Ctenocephalides canis*) 151
Oriental Rat Flea (*Xenopsylla cheopis*) 151
European Rabbit Flea (*Spilopsyllus cuniculi*) 152
Sticktight Flea (*Echidnophaga gallinacea*) 152
Chigoe (*Tunga penetrans*) 152
Northern Rat Flea (*Nosopsyllus fasciatus*) 153
European Chicken Flea (Hen Flea in Europe) (*Ceratophyllus gallinae*) 153
European Mouse Flea (*Leptopsylla segnis*) 154
Public Health Importance 154
Flea-Associated Allergies 156
Plague 156
Murine Typhus 159
Other Flea-Borne Rickettsial Agents 161
Other Flea-Borne Pathogens 161
Bacteria 161
Viruses 162
Tungiasis 162
Fleas as Intermediate Hosts of Helminths 162
Veterinary Importance 163
Flea Allergy Dermatitis 164
Tungiasis 164
Myxomatosis 164
Squirrel Pox 165
Murine Trypanosomiasis 165
Other Flea-Borne Pathogens and Parasites 165
Fleas as Intermediate Hosts or Vectors of Helminths 165
Prevention and Control 166
References and Further Reading 167

11. Flies (Diptera)

Reid R. Gerhardt and Lawrence J. Hribar

Taxonomy 171
Morphology 173
Life History 177
Behavior and Ecology 177
Families of Minor Medical or Veterinary Interest 178
Tipulidae (Crane Flies) 178
Bibionidae (March Flies) 179
Sciaridae (Dark-Winged Fungus Gnats) 179
Chaoboridae (Phantom Midges) 180
Corethrellidae (Frog-Biting Midges) 180
Chironomidae (Chironomid Midges) 180
Rhagionidae (Snipe Flies) 182
Athericidae (Athericid Flies) 182
Stratiomyidae (Soldier Flies, Latrine Flies) 182
Phoridae (Humpbacked Flies, Scuttle Flies) 183
Syrphidae (Flower Flies, Hover Flies) 184
Piophiliae (Skipper Flies) 184
Drosophilidae (Small Fruit Flies) 185
Chloropidae (Grass Flies, Eye Gnats) 185
Public Health Importance 186
Veterinary Importance 186
Prevention and Control 187
References and Further Reading 188

12. Phlebotomine Sand Flies and Moth Flies (Psychodidae)

Leonard E. Munstermann

Taxonomy 192
Psychodinae 192
Sycoracinae 192
Phlebotominae 193
Morphology 193
Psychodinae 193
Phlebotominae 194
Life History 195
Psychodinae 195
Phlebotominae 195
Behavior and Ecology 196
Psychodinae 196
Phlebotominae 196

Public Health Importance 198
Psychodinae 198
Phlebotominae 198
Vesicular Stomatitis Virus Disease 198
Chandipura Virus Disease 200
Sand Fly Fever 200
Changuinola Virus Disease 201
Bartonellosis 201
Leishmaniasis 202
Veterinary Importance 207
Leishmaniasis 208
Vesicular Stomatitis Virus Disease 208
Prevention and Control 208
Psychodinae 208
Phlebotominae 209
References and Further Reading 210

13. Biting Midges (Ceratopogonidae)

Gary R. Mullen and C. Steven Murphree

Taxonomy 213
Morphology 214
Life History 215
Behavior and Ecology 216
Public Health Importance 218
Oropouche Fever 221
Other Viral Agents 221
Mansonellosis 222
Veterinary Importance 223
Bluetongue Disease 223
Epizootic Hemorrhagic Disease 225
African Horse Sickness 227
Schmallenberg Virus 228
Other Viral Agents 229
Blood Protozoans 229
Equine Onchocerciasis 230
Other Filarial Nematodes 231
Equine Allergic Dermatitis 231
Prevention and Control 232
References and Further Reading 232

14. Black Flies (Simuliidae)

Peter H. Adler and John W. McCreadie

Taxonomy 237
Morphology 238
Life History 240
Behavior and Ecology 240
Public Health Importance 243
Biting and Nuisance Problems 243
Human Onchocerciasis 245
Mansonellosis 250
Other Diseases Related to Black Flies 250
Veterinary Importance 251
Bovine Onchocerciasis 252
Leucocytozoonosis 252
Other Parasites and Pathogens of Veterinary Importance 253
Simuliotoxicosis 253
Prevention and Control 254
Onchocerciasis Control 255
References and Further Reading 256

15. Mosquitoes (Culicidae)

Woodbridge A. Foster and Edward D. Walker

Taxonomy 261
Morphology 264
Life History 269
Behavior and Ecology 272
Genetics 279
Public Health Importance 280
Mosquito Bites 280
Mosquito-Borne Viruses 280
Togaviridae (*Alphavirus*) 283
Flaviviridae (*Flavivirus*) 287
Bunyaviridae (*Orthobunyavirus* and *Phlebovirus*) 297
Malaria 300
Filariasis 305
Veterinary Importance 310
Mosquito-Borne Viruses of Animals 310
Nonhuman Malarias 312
Dog Heartworm 313
Other Filarial Nematodes of Animals 314
Prevention and Control 314
Control of Pathogen Transmission 317
References and Further Reading 317

16. Horse Flies and Deer Flies (Tabanidae)

Bradley A. Mullens

Taxonomy 327
Morphology 329
Life History 330
Behavior and Ecology 332
Public Health Importance 336
Loiasis 336
Tularemia 337
Other Tabanid-Transmitted Human Pathogens 338
Veterinary Importance 338
Surra and Related Trypanosomiases 338
Equine Infectious Anemia 339
Anaplasmosis 339

Elaeophorosis 340
Other Pathogens of Veterinary Importance 340
Prevention and Control 340
References and Further Reading 341

17. Muscid Flies (Muscidae)

Roger D. Moon

Taxonomy 345
Morphology 346
Life History 350
Behavior and Ecology 351
Species of Medical-Veterinary Importance 353
House Fly (*Musca domestica*) 353
Bazaar Fly (*Musca sorbens*) 354
Bush Fly (*Musca vetustissima*) 354
Face Fly (*Musca autumnalis*) 354
Cluster Fly (*Pollenia rudis*) 354
Stable Fly (*Stomoxys calcitrans*) 354
Horn Fly (*Haematobia irritans irritans*) and Buffalo Fly (*Haematobia irritans exigua*) 355
False Stable Fly *(Muscina stabulans*) and Its Relatives 355
Little House Fly (*Fannia canicularis*) and Its Relatives 355
Garbage Flies (*Hydrotaea* spp.) 356
Sweat Flies (*Hydrotaea* spp.) 356
Bird Nest Parasites (*Passeromyia* spp., *Philornis* spp., and *Protocalliphora* spp.) 356
Public Health Importance 356
House Fly (*Musca domestica*) 357
Bazaar Fly (*Musca sorbens*) 357
Bush Fly (*Musca vetustissima*) 358
Face Fly (*Musca autumnalis*) and Cluster Fly (*Pollenia rudis*) 358
Stable Fly (*Stomoxys calcitrans*) 358
False Stable Fly (*Muscina stabulans*) and Its Relatives 359
Little House Fly (*Fannia canicularis*) and Its Relatives 359
Garbage Flies (*Hydrotaea* spp.) 359
Sweat Flies (*Hydrotaea* spp.) 359
Veterinary Importance 359
House Fly (*Musca domestica*) 360
Bush Fly (*Musca vetustissima*) 360
Face Fly (*Musca autumnalis*) 361
Stable Fly (*Stomoxys calcitrans*) 362
Horn Fly (*Haematobia irritans irritans*) and Buffalo Fly (*Haematobia irritans exigua*) 363
Sweat Flies (*Hydrotaea* spp.) 364
Bird Nest Parasites (*Philornis* spp.) 364
Prevention and Control 364
References and Further Reading 366

18. Tsetse Flies (Glossinidae)

William L. Krinsky

Taxonomy 369
Morphology 370
Life History 371
Behavior and Ecology 372
Public Health Importance 374
African Sleeping Sickness 374
Veterinary Importance 377
Nagana 377
Prevention and Control 379
References and Further Reading 381

19. Myiasis (Muscoidea, Oestroidea)

Philip J. Scholl, Douglas D. Colwell, and Ramón Cepeda-Palacios

Taxonomy 384
Morphology 389
Life History 390
Ecology and Behavior 392
Myths 394
Flies Involved in Myiasis 394
Psychodidae (Moth Flies, Drain Flies) 394
Stratiomyidae (Soldier Flies) 394
Phoridae (Humpback Flies) 395
Syrphidae (Flower Flies, Hover Flies, Rat-Tailed Maggots) 395
Piophilidae (Skipper Flies) 395
Neottiophilidae (Nest Skipper Flies) 396
Drosophilidae (Pomace Flies, Vinegar Flies, Fruit Flies, and Wine Flies) 396
Chloropidae (Grass Flies and Australian Frog Flies) 396
Anthomyiidae (Root Maggots) 396
Fanniidae (Faniid Flies) 396
Muscidae (Dung Flies) 397
Tropical Nest Flies 397
Calliphoridae (Blow Flies, Carrion Flies, Floor Maggots, Nest Maggots, and Screwworms) 398
Carrion-Associated Blow Flies 398
Sarcophagidae (Flesh Flies) 401
Oestridae (Bot Flies) 402
New World Skin Bot Flies (Cuterebrinae) 403
Old World Skin Bot Flies (Hypodermatinae) 406
Nose Bot Flies (Oestrinae) 407
Stomach Bot Flies (Gasterophilinae) 410
Other Oestroid Flies 411
Public Health Importance 411
Clinical Use of Maggots 412
Veterinary Importance 413

Prevention and Control 414
Screwworm Eradication Program 415
Cattle Grub Control 416
Myiasis and Molecular Advances 417
References and Further Reading 417

20. Louse Flies, Keds, and Bat Flies (Hippoboscoidea)

Will K. Reeves and John E. Lloyd

Taxonomy 421
Morphology 422
Hippoboscidae 423
Streblidae 424
Nycteribiidae 424
Life History 424
Behavior and Ecology 425
Common Species of Hippoboscids 426
Sheep Ked (*Melophagus ovinus*) 426
Dog fly (*Hippobosca longipennis*) 427
Hippobosca equina 428
Hippobosca variegata 428
Deer Keds (*Lipoptena* and *Neolipoptena* spp.) 428
Lipoptena depressa 428
Lipoptena cervi 428
Lipoptena mazamae 429
Neolipoptena ferrisi 429
Pigeon Fly (*Pseudolynchia canariensis*) 429
Public Health Importance 429
Veterinary Importance 430
Prevention and Control 434
References and Further Reading 435

21. Moths and Butterflies (Lepidoptera)

Gary R. Mullen and Jennifer M. Zaspel

Taxonomy 439
Morphology 440
Spicule Hairs 441
Spine Hairs 443
Life History 443
Behavior and Ecology 443
Urticating Caterpillars 444
Lachryphagous Moths 450
Erebidae 450
Geometridae 450
Crambidae 451
Notodontidae 451
Nolidae 451
Sphingidae 451
Drepanidae 451
Wound-Feeding and Skin-Piercing Moths 452
Public Health Importance 452
Veterinary Importance 454
Caterpillar-Induced Equine Abortion 454
Prevention and Control 455
References and Further Reading 455

22. Ants, Wasps, and Bees (Hymenoptera)

Hal C. Reed and Peter J. Landolt

Taxonomy 459
Morphology 460
Life History 463
Behavior and Ecology 464
Hymenoptera Venoms 465
Ant Venoms 465
Vespid Venoms 466
Honey Bee Venom 466
Ants 466
Fire Ants (*Solenopsis* spp.) 467
Harvester Ants (*Pogonomyrmex* spp.) 469
Pavement Ant (*Tetramorium caespitum*) 470
Pharaoh's Ant (*Monomorium pharaonis*) 470
Wasps 471
Solitary Wasps 471
Mutillidae 471
Pompilidae 471
Sphecidae 471
Social Wasps (Vespidae) 472
Yellowjackets (*Dolichovespula* and *Vespula*) 473
Hornets (*Vespa* spp.) 475
Paper Wasps (*Polistes* spp.) 476
Bees 476
Solitary Bees 476
Halictidae 476
Anthophoridae 477
Social Bees 477
Apidae 477
Bumble Bees (*Bombus* spp.) 477
Honey Bees (*Apis* spp.) 478
Public Health Importance 479
Veterinary Importance 483
Prevention and Control 483
References and Further Reading 484

23. Scorpions (Scorpiones)

Gary R. Mullen and W. David Sissom

Taxonomy 489
Buthidae 490
Pseudochactidae 491
Chaerilidae 491
Chactidae 491
Euscorpiidae 491
Superstitioniidae 491

Typhlochactidae 491
Troglotayosicidae 491
Akravidae 491
Iuridae 492
Caraboctonidae 492
Vaejovidae 492
Bothriuridae 492
Hormuridae 492
Heteroscorpionidae 493
Hemiscorpiidae 493
Urodacidae 493
Diplocentridae 493
Scorpionidae 493
Morphology 494
Life History 496
Behavior and Ecology 497
Public Health Importance 498
Scorpions of Medical Importance 500
Veterinary Importance 501
Prevention and Control 501
References and Further Reading 502

24. Solpugids (Solifugae)

Gary R. Mullen

References and Further Reading 506

25. Spiders (Araneae)

Gary R. Mullen and Richard S. Vetter

Taxonomy 507
Mygalomorph Spiders 507
Actinopodidae 507
Barychelidae 508
Dipluridae 508
Atracidae 508
Macrothelidae 508
Theraphosidae 508
Araneomorph Spiders 508
Agelenidae 508
Araneidae 508
Ctenidae 509
Desidae 509
Dysderidae 509
Eutichuridae 509
Gnaphosidae 509
Lamponidae 509
Lycosidae 509
Miturgidae 510
Oxyopidae 510
Pisauridae 510
Salticidae 510
Segestriidae 510
Sicariidae (Including the Former Loxoscelidae) 510
Theridiidae 510
Thomisidae 511
Trachelidae 511
Morphology 511
Life History 513
Behavior and Ecology 513
Public Health Importance 514
Tarantism 514
Tarantulas 515
Australian Funnel-Web Spiders 517
South American Wandering Spiders 518
Yellow Sac Spiders 518
Hobo Spiders 518
Recluse Spiders 519
Widow Spiders 523
Veterinary Importance 527
Prevention and Control 528
References and Further Reading 529

26. Mites (Acari)

Gary R. Mullen and Barry M. OConnor

Taxonomy 533
Morphology 534
Life History 535
Behavior and Ecology 536
Public Health Importance 536
Mite-Induced Dermatitis 537
Melicharidae 537
Dermanyssidae 537
Macronyssidae 539
Laelapidae 541
Trombiculidae 542
Stored-Products Mites 544
Skin-Invading Mites 547
Sarcoptidae 549
Human Scabies 550
Human Notoedric Mange 552
Mite-Induced Allergies 552
Internal Acariasis 555
Mite-Borne Diseases of Humans 556
Rickettsialpox 556
Tsutsugamushi Disease 557
Intermediate Hosts of Human Parasites 559
Delusory Acariasis and Acarophobia 559
Veterinary Importance 559
Mite-Induced Dermatitis 560
Dermanyssidae 560
Macronyssidae 560
Laelapidae 562
Trombiculidae, Leeuwenhoekiidae, Walchiidae 563
Fur Mites 564
Cheyletidae 564
Myobiidae 565

Listrophoridae 566
Atopomelidae 567
Chirodiscidae 567
Myocoptidae 567
Feather Mites 568
Mange Mites 568
Laminosioptidae 571
Demodecidae 572
Psorergatidae 574
Sarcoptidae 575
Other Sarcoptid Genera 578
Rhyncoptidae 580
Psoroptidae 581
Psoroptic Scab Mites 581
Chorioptic Scab Mites 583
Caparinic Scab Mites 584
Harpirhynchidae 585
Hypoderatidae 585
Mite-Induced Allergies 585
Internal Acariasis 585
Ear Mites 586
Respiratory Mites 588
Entonyssidae 588
Rhinonyssinae 589
Halarachnidae 589
Ereynetidae 590
Trombiculidae, Leeuwenhoekiide, and Walchiidae 591
Lemurnyssidae 591
Turbinoptidae 591
Cytoditidae 591
Pneumocoptidae 592
Gastronyssidae 592
Mite-Borne Diseases 592
Mites as Intermediate Hosts of Tapeworms 593
References and Further Reading 596

27. Ticks (Ixodida)

William L. Nicholson, Daniel E. Sonenshine, Bruce H. Noden, and Richard N. Brown

Taxonomy 603
Family Ixodidae (Hard Ticks) 604
Family Argasidae (Soft Ticks) 605
Family Nuttalliellidae 606
Morphology 606
External Anatomy 606
Internal Anatomy 609
Life History 611
Ixodid Life Cycles 611
Argasid Life Cycles 612
Behavior and Ecology 613
Tick Species of Medical-Veterinary Importance 618
Public Health Importance 626
Human Babesiosis 627
Tick-Borne Encephalitis Complex 628
Colorado Tick Fever 630
Crimean-Congo Hemorrhagic Fever 631
Severe Fever and Thrombocytopenia Syndrome Virus 632
Heartland Virus 632
Bourbon Virus Disease 632
Rocky Mountain Spotted Fever 632
Boutonneuse Fever 635
Other Spotted Fever Group Rickettsiae 635
Human Ehrlichiosis 636
Human Anaplasmosis 637
Human Neoehrlichiosis 637
Q Fever 638
Lyme Disease 638
Tick-Borne Relapsing Fever and *Borrelia miyamotoi* Disease 643
Tularemia 646
Tick Paralysis 647
Tick-Bite Allergies 648
Veterinary Importance 648
Piroplasmoses 649
Diseases Caused by Members of the Family Anaplasmataceae 652
Prevention and Control 659
Personal Protection 659
Acaricides 660
Pheromone-Assisted Control 661
Passive Treatment 661
Hormone-Assisted Control 661
Biological Control 661
Vaccines 661
Management 662
Eradication 663
References and Further Reading 663

28. Molecular Tools Used in Medical and Veterinary Entomology

Dana Nayduch, Rebecca Trout Fryxell, and Pia Untalan Olafson

Molecular Techniques 673
Nonmolecular Techniques 677
Genomics: Sequencing, Cataloging, and Study of an Organism's Genes 678
Genome Projects of Important Arthropods 678

Genome Sequencing 679
Analyzing Gene Expression 684
RNA Analysis 684
Protein Analysis 686
Molecular Tools to Manipulate Arthropods 687
Editing Genomes With CRISPR-Cas9 687
Insect Population Suppression 687
Posttranscriptional Gene Silencing With RNAi 689
Conclusions and Future Directions 689
References and Further Reading 691

Appendix: Arthropod-Related Viruses of Medical and Veterinary Importance 695
Michael J. Turell

Glossary 705

Taxonomic Index 741
Subject Index 757

This page intentionally left blank

Contributors

Peter H. Adler, Department of Plant and Environmental Sciences, Clemson University, Clemson, SC, United States

Christopher M. Barker, Center for Vectorborne Diseases, School of Veterinary Medicine, University of California, Davis, CA, United States

Richard J. Brenner, Director, ATIP Foundation, Arlington, TX, United States; Formerly Assistant Administrator of Technology Transfer, USDA, Agricultural Research Service, Beltsville, MD, United States

Richard N. Brown, Department of Wildlife, Humboldt State University, Arcata, CA, United States

Nathan D. Burkett-Cadena, University of Florida, Florida Medical Entomology Laboratory, Vero Beach, FL, United States

Ramón Cepeda-Palacios, Animal Science and Environmental Conservation Department, Autonomous University of Baja California Sur, La Paz, Mexico

Douglas D. Colwell, Livestock Parasitology, Agriculture and Agri-Food Canada, Lethbridge Research and Development Centre, Lethbridge, AB, Canada

Lance A. Durden, Department of Biology, Georgia Southern University, Statesboro, GA, United States

Woodbridge A. Foster, Department of Evolution, Ecology, and Organismal Biology, and Department of Entomology, The Ohio State University, Columbus, OH, United States

Rebecca Trout Fryxell, Department of Entomology and Plant Pathology, University of Tennessee, Knoxville, TN, United States

Reid R. Gerhardt, Department of Entomology and Plant Pathology, University of Tennessee, Knoxville, TN, United States

Nancy C. Hinkle, Department of Entomology, University of Georgia, Athens, GA, United States

Lawrence J. Hribar, Florida Keys Mosquito Control District, Marathon, FL, United States

Jonas G. King, Department of Biochemistry, Molecular Biology, Entomology and Plant Pathology, Mississippi State University, Starkville, MS, United States

Richard D. Kramer, Board Certified Entomologist (BCE), CEO Innovative Pest Management, Inc., Brookeville, MD, United States

William L. Krinsky, Division of Entomology, Peabody Museum of Natural History, Yale University, New Haven, CT, United States

Peter J. Landolt, Temperate Tree Fruit and Vegetable Research, USDA ARS, Wapato, WA, United States

John E. Lloyd, Department of Renewable Resources, University of Wyoming, Laramie, WY, United States

John W. McCreadie, Department of Biological Sciences, University of South Alabama, Mobile, AL, United States

Roger D. Moon, Department of Entomology, University of Minnesota, St. Paul, MN, United States

Gary R. Mullen, Department of Entomology and Plant Pathology, Auburn University, Auburn, AL, United States

Bradley A. Mullens, Department of Entomology, University of California, Riverside, CA, United States

Leonard E. Munstermann, Yale University School of Public Health, New Haven, CT, United States

C. Steven Murphree, Biology Department, Belmont University, Nashville, TN, United States

Dana Nayduch, USDA-ARS, Arthropod-borne Animal Diseases Research Unit, Manhattan, KS, United States

William L. Nicholson, Rickettsial Zoonoses Branch, Division of Vector-Borne Diseases, National Center for Emerging and Zoonotic Infectious Diseases, Centers for Disease Control and Prevention, Atlanta, GA, United States

Bruce H. Noden, Department of Entomology and Plant Pathology, Oklahoma State University, Stillwater, OK, United States

Barry M. OConnor, Museum of Zoology, University of Michigan, Ann Arbor, MI, United States

Pia Untalan Olafson, USDA-ARS, Livestock Arthropod Pests Research Unit, Kerrville, TX, United States

Hal C. Reed, Department of Biology and Chemistry, Oral Roberts University, Tulsa, OK, United States

Will K. Reeves, U.S. Department of Agriculture, Animal and Plant Health Inspection Service, Biotechnology Resource Services, Western Compliance Assurance Branch, Fort Collins, CO, United States

William K. Reisen, Center for Vectorborne Diseases, School of Veterinary Medicine, University of California, Davis, CA, United States

Justin O. Schmidt, Southwestern Biological Institute, Tucson, AZ, United States; Department of Entomology, University of Arizona, Tucson, AZ, United States

Philip J. Scholl, United States Department of Agriculture, Agricultural Research Service (retired), Oxford, FL, United States

W. David Sissom, Department of Life, Earth and Environmental Sciences, West Texas A&M University, Canyon, TX, United States

Daniel E. Sonenshine, Department of Biological Sciences, Old Dominion University, Norfolk, VA, United States

Michael J. Turell, Virology Division, United States Army Medical Research Institute of Infectious Diseases, Fort Detrick, Frederick, MD, United States (retired)

Richard S. Vetter, Department of Entomology, University of California, Riverside, CA, United States

Edward D. Walker, Department of Microbiology and Molecular Genetics, and Department of Entomology, Michigan State University, East Lansing, MI, United States

Jennifer M. Zaspel, Milwaukee Public Museum, Milwaukee, WI, United States

Preface

It has been 10 years since the publication of the second edition of this book. Significant advances have been made in our knowledge of a plethora of arthropod-related problems and diseases, including changes in their geographic ranges and the emergence of new or previously unrecognized arthropod-borne diseases of medical and veterinary concern. Among notable examples of the latter are Zika and Chikungunya viruses transmitted by mosquitoes and a number of tick-borne pathogens, such as Bourbon, Heartland, and severe fever with thrombocytopenia syndrome viruses, as well as newly recognized forms of ehrlichiosis, rickettsiosis, and babesiosis. A new chapter has been added—Chapter 3: Arthropod Toxins and Venoms—to provide an overview of the structural nature and diversity of chemical compounds that play an important medical-veterinary role, particularly among stinging and biting insects and arachnids. In addition, Chapter 28: Molecular Tools Used in Medical and Veterinary Entomology has been expanded, reflecting the many significant advances and applications of molecular and genetic techniques in recent years, which have become such an integral part of medical-veterinary entomology today.

One of the primary objectives of the first and second editions has been to provide a textbook suitable for teaching courses in medical and veterinary entomology at the college and university levels. In keeping with that goal and the format of the previous editions, the book is organized from an entomological perspective, with each chapter devoted to a particular taxonomic group of insects or arachnids (including spiders, scorpions, solpugids, mites, and ticks). Each chapter includes the following subheadings: Taxonomy, Morphology, Life History, Behavior and Ecology, Public Health Importance, Veterinary Importance, Prevention and Control, and References and Further Reading. The separate sections on public health and veterinary entomology are designed to assist instructors in using this book to teach courses in either medical or veterinary entomology, or courses combining these two related disciplines. The book concludes with an Appendix titled "Arthropod-Related Viruses of Medical and Veterinary Importance" and a Glossary of approximately 1,700 terms. The latter is intended to assist the reader in understanding entomological, medical, and other terms used in the book, with which she or he may not be familiar. We hope this will be helpful to the widest possible range of readers, specialists and nonspecialists alike, in diverse disciplines relating either directly or indirectly to the subject matter**.** The text is illustrated with 538 figures and 22 revised, or new, color maps.

In addition to its value to students as a textbook, this volume is intended for a much broader audience as a comprehensive reference source for biologists in general, entomologists, zoologists, parasitologists, physicians, public health personnel, veterinarians, wildlife biologists, vector biologists, military and armed forces entomologists, the general public, and others looking for a readable, authoritative source of information on this important topic.

We welcome as new contributing authors to the book the following 14 individuals: Justin O. Schmidt (Chapter 3), Christopher M. Barker (Chapter 4), Lawrence J. Hribar (Chapter 11), Leonard E. Munstermann (Chapter 12), C. Steven Murphree (Chapter 13), Douglas D. Colwell and Ramón Cepeda-Palacios (Chapter 19), Will K. Reeves (Chapter 20), Jennifer M. Zaspel (Chapter 21), W. David Sissom (Chapter 23), Bruce H. Noden and Richard N. Brown (Chapter 27), and Rebecca Trout Fryxell and Pia Untalan Olafson (Chapter 28). Together with 24 continuing authors of the second edition and the Graphics Editor, 40 contributors in all, they have helped significantly in revising the respective chapters—providing new perspectives and achieving an appropriate balance between medical and veterinary entomology as closely related disciplines.

The success of the previous two editions is reflected by the widespread adoption of this book for teaching medical and/or veterinary-related courses at colleges and universities throughout the United States and other parts of the world. We hope the third edition will be equally successful in helping to educate and inspire the next generation of medical and veterinary entomologists.

Gary R. Mullen
Lance A. Durden

This page intentionally left blank

Acknowledgments

As with any undertaking of this magnitude, there are many individuals to whom the editors are indebted. Foremost are the contributing authors, whose combined expertise and commitment to promoting medical and veterinary entomology have made the third edition of this book possible. We also owe a debt of gratitude to the many other individuals who contributed so generously of their time in reviewing chapters or select parts of the text, offering suggestions for improving and updating material for the third edition, preparing illustrations, and providing original photographs and new color images for the 538 figures.

We particularly want to recognize the following people who have contributed significantly in preparing the figures, maps, and other illustrations throughout the book: first and foremost, Jonas G. King (Mississippi State University, Starkville, MS) for his invaluable graphics skills and all-important role in ensuring the high-quality color images and maps throughout the chapters; a special thanks to Jillian Masters, an undergraduate student working with Jonas King, for her welcomed talent and generous time assisting with the figures, particularly the maps; and graduate student Anagha Gopakumar, also a member of the King lab group at Mississippi State for her part in preparing illustrations; Lorenza Beati and Lance A. Durden (Georgia Southern University, Statesboro, GA) for preparing the stacked images of the lice (Figs. 7.5–7.7, 7.12–7.16 and 7.19) and fleas (Figs. 10.1, 10.8–10.12, 10.14, 10.17–10.19 and 10.21, and Oriental rat flea on book cover); Nathan D. Burkett-Cadena (University of Florida, Florida Medical Entomology Laboratory, Vero Beach, FL), for providing the original color illustrations that accompany his chapter on morphological adaptations of parasitic arthropods (Figs. 2.1–2.5), preparing original illustrations for the epidemiology chapter (Figs. 4.2 and 4.3), and providing 12 additional figures throughout the book (see below); E. Paul Catts (Washington State University, Pullman, WA), for being able to include in this third edition his superb original line drawings of myiasis-causing flies that he prepared for the first edition, prior to his untimely death in 1996 (Figs. 19.1–19.9, 19.11, 19.13–19.15, 19.17, 19.20, 19.23, 19.27, 19.32, 19.33, 19.36, 19.37 and 19.40); Victoria Rhodes (Missouri Southern State University, Joplin, MO), for her excellent color illustrations, Chapter 28, Figs. 28.1–28.4, and Boxes 28.1–28.3); Margo A. Duncan (Gainesville, FL), for her original illustrations, particularly those in the Lepidoptera chapter (original Fig. 21.4 and redrawn Figs. 21.2, 21.3 and 21.5) that appear in the first, second, and third editions; Rebecca L. Nims (Social Circle, GA), for the outstanding work she did in redrawing from various sources more than half the black-and-white figures for the first and second editions (notably for the mite chapter), virtually all of which have been retained in the third edition; and Philip B. Shevlin (Auburn University, Auburn, AL) for his assistance with preparation of the chemical structures of arthropod toxins and venoms (Figs. 3.3–3.8).

We also acknowledge the following individuals for preparing original illustrations, primarily line drawings, which appear within these pages: Woodbridge A. Foster (The Ohio State University, Columbus, OH; Fig. 15.14); Susan Milna Hope (Mebane, NC; Fig. 16.1); Jonas G. King (Mississippi State University, Starkville, MS; Figs. 14.1 and 18.12); Takumasa Kondo (Palmira, Valle, Colombia; Fig. 11.9); William L. Krinsky (Yale University, New Haven, CT; Figs. 5.3, 8.7 and 8.11); and Blair Sampson (US Department of Agriculture, Agricultural Research Service, Poplarville, MS; Fig. 22.6).

The following individuals have kindly provided original photographs, slides, or digital images for reproduction as figures in this edition, or have contributed in various other ways:

W. V. Adams, Jr. (Louisiana State University, Baton Rouge, LA; Fig. 16.13); Peter H. Adler (Clemson University, Clemson, SC; Fig. 14.7); Roger D. Akre (Washington State University, Pullman, WA; Figs. 22.14, 22.15, 22.24 and 22.31); Karim Aoun and Aïda Bouratbine (Institut Pasteur de Tunis, Tunisia; Fig. 12.14); Stephen Ausmus (US Department of Agriculture; Fig. 22.25); Hans Bänziger (Chaing Mai University, Thailand; Figs. 21.19, 21.20 and 20.22); Martin Bennett (Creative Commons; Fig. 19.21); Robert B. Berry (Orange Breasted Falcon Project, The Peregrine Fund, Wolf, WY; Fig. 17.22); Yehuda Braverman (Kimron Veterinary Institute, Israel; Fig. 12.9); Alberto B. Broce (Kansas State University, Manhattan, KS); Nathan D. Burkett-Cadena (University of Florida, Florida Medical Entomology Laboratory, Vero

Beach, FL; Figs. 5.1, 5.4, 6.4, 7.9, 10.4−10.7, 16.3, 20.6, 25.6 and 26.11); João P. Burini (Pontifica Universidade Católica de São Paulo, Brazil; Figs. 23.2, 23.14, 25.4, 25.5 and 25.10); Lyle Buss (University of Florida, Gainesville, FL; Fig. 21.25); Jerry F. Butler (University of Florida, Gainesville, FL; Figs. 9.3B, 20.5, 21.13, 25.25, 26.40, 26.43 and 27.11); Bonnie Buxton (Philadelphia College of Osteopathic Medicine-Georgia Campus, Suwanee, GA; Fig. 15.42); Brittany E. Campbell (University of Florida, Gainesville, FL; Fig. 8.13); Luis Paulo Carvalho (Federal University of Rondônia-Unir, Porto Velho, Brazil; Fig. 13.14); James Castner (University of Florida, Gainesville, FL; Figs. 6.3, 6.5−6.12 and 21.26); Ronald D. Cave (Indian River Research and Education Center, University of Florida, Fort Pierce, FL; Fig. 19.26); Valerie J. Cervenka (University of Minnesota, St. Paul, MN, Fig. 17.23); Chan Chee Keong (Panang Island, Malaysia; Fig. 9.6); Jillian Cowles (Vail, AZ; Fig. 3.9); Douglas A. Craig (University of Alberta, Edmonton, AB, Canada; Figs. 14.2B and 14.3); Eddie W. Cupp (Owensboro, KY; Figs. 14.12 and 14.14); Aaron T. Dossey (Gainesville, FL; Fig. 1.5); S. Allen Edgar (Auburn University, Auburn, AL; Figs. 15.37 and 15.38); Marc E. Epstein (California Department of Food and Agriculture, Sacramento, CA; Fig. 21.12); John Flannery (Richmond County, NC; Fig. 21.1A); Woodbridge A. Foster (The Ohio State University, Columbus, OH; Figs. 15.11, 15.17, 15.19, 15.26, 15.29 and 15.34); Wes Gaston (US Department of Agriculture, Animal and Plant Health Inspection Service, Wildlife Services; Fig. 19.41); James L. Gathany (Centers for Disease Control and Prevention, Atlanta, GA; Figs. 7.4, 8.3, 11.2, 12.2, 27.5 and 27.17); Carolyn Grissom (Shelbyville, TN; Fig. 25.17); Duane J. Gubler (Duke-National University of Singapore Graduate Medical School, Singapore; Fig. 15.25); Hadel H. Go (American Museum of Natural History, New York, NY; for a taxonomic clarification regarding the Hymenoptera); Robert G. Hancock (University of the Cumberlands, Williamsburg, KY; Fig. 15.23); Elton J. Hansens (Asheville, NC; Figs. 7.8, 10.20, 16.7, 16.10A and 17.12); Mark Harvey (Western Australia Museum, Welshpool, Western Australia; for taxonomic information on the Solifugae); Nancy C. Hinkle (University of Georgia, Athens, GA; Fig. 9.11); Mac Horton (Clemson University, Clemson, SC; Fig. 22.28); Kevin Humphries (Huntsville, AL; Fig. 25.16); Lacy L. Hyche (Auburn University, Auburn, AL; Fig. 21.15); Gregory D. Johnson (Montana State University, Bozeman, MT; Fig. 7.20); Jena Johnson (Jena Johnson Photography and University of Georgia, Athens, GA; Figs. 14.1, 14.5, 14.6, 22.13 and 22.20; book-cover images, Asian tiger mosquito and green-bottle fly); Florian Karolyi (University of Vienna, Austria; Fig. 16.4); Takumasa Kondo (Palmira, Valle, Colombia; Fig. 22.23); William L. Krinsky (Yale University, New Haven, CT; Figs. 5.2, 5.5 and 8.6); Richard C. Lancaster (Agriculture and Agri-Food Canada, Lethbridge, AB; Fig. 13.2); Lloyd L. Lauerman (Alabama State Veterinary Diagnostic Laboratory Auburn, AL; Figs. 13.10−13.12); Matthew S. Lehnert (Kent State University at Stark, North Canton, OH; Figs. 21.1B,C and 21.24); John E. Lloyd, Sr. (University of Wyoming, Laramie, WY; Figs. 7.19, 7.21, 20.3, 20.8 and 20.12−20.14); Amanda D. Loftis (Ross University School of Veterinary Medicine, Basseterre, Saint Kitts, West Indies; Fig. 20.1); Stephen A. Marshall (University of Guelph, Guelph, ON, Canada; Figs. 13.1, 14.2A and 14.4); Sturgis McKeever (Georgia Southern University, Statesboro, GA; Figs. 16.5, 19.18, 21.6−21.10, 21.14, 21.18 and 25.21, and saddleback caterpillar on book cover); Mae Melvin (Centers for Disease Control and Prevention, Atlanta, GA; Fig. 8.8); Hendrick J. Meyer (North Dakota State University, Fargo, ND; Fig. 17.25); Camille Meyers (Courtesy of Peregrine Fund; Fig. 17.22); Steve Mihok (Russell, ON, Canada; Fig. 16.10D); Roger D. Moon (University of Minnesota, St. Paul, MN; Figs. 17.16 and 17.19); Gary R. Mullen (Auburn University, Auburn, AL; Figs. 8.16, 9.3A, 13.5, 13.6, 13.13, 13.14, 17.18, 17.20, 19.19, 21.11, 22.9, 22.19, 22.21, 22.22, 22.29, 22.30, 25.13 and 27.9); Bradley A. Mullens (University of California, Riverside, CA; Figs. 16.8 and 16.10B); Teresa R. Mullens (Riverside, CA; Fig. 16.10C); Leonard E. Munstermann (Yale University, New Haven, CT; Figs. 12.5−12.8 and 12.10); Amy C. Murillo (University of California, Riverside, CA; Figs. 7.17A−F, 7.22, 10.13 and 26.25); Harold D. Newson (Michigan State University, East Lansing, MI; Figs. 15.39 and 15.41); William L. Nicholson (Centers for Disease Control and Prevention, Atlanta, GA; Fig. 27.10; Susan M. Noh (US Department of Agriculture, Pullman, WA; Fig. 27.30); Yoshiro Ohara (Kanazawa Medical University, Uchinada, Ishikawa, Japan; Fig. 16.12); Christopher D. Paddock (Centers for Disease Control and Prevention, Atlanta, GA; Figs. 27.22 and 27.23); Jonathan D. Patterson (Michigan State University, East Lansing, MI; Fig. 15.40); Laverne L. Pechuman (Cornell University, Ithaca, NY; Fig. 16.6); Nicholas Perrault (Creative Commons; Fig. 14.8); Eric Poggenphol (Eric Poggenphol Photography, Northampton, MA; Fig. 14.13); Lorenzo Prendini (American Museum of Natural History, New York; for his contributed expertise on scorpions); Robert J. Raven (Queensland Museum, South Brisbane, Australia; Fig. 25.9); Charles Ray (Auburn University, AU Plant Diagnostic Laboratory, Auburn, AL; for providing records of insects and mites biting humans); David E. Reed (Chanhassen, MN; Fig. 9.4); Hal C. Reed (Oral Roberts University, Tulsa, OK; Figs. 22.2, 22.5, 22.10, 22.16 and 22.18); Lawrence E. Reeves (University of Florida, Gainesville, FL; Fig. 19.24); Jon Richfield (Creative Commons; Fig. 24.1); Mary Elizabeth Hayes Rogers (Waukegan, IL; Fig. 13.18); Christopher Saski (Clemson University,

Genomics and Computational Laboratory, Clemson, SC; for his review and editorial suggestions, Chapter 28); Philip J. Scholl (US Department of Agriculture, Agricultural Research Service, Oxford, FL; Fig. 19.35); Justin O. Schmidt (US Department of Agriculture, Carl Hayden Bee Research Center, Tucson, AZ; Fig. 22.27); Joseph A. Schemanchuk (Department of Agriculture and Agri-Food Canada, Lethbridge, AB, Canada; Figs. 14.15 and 14.16); D. Craig Sheppard (University of Georgia, Athens, GA; Fig. 17.21); W. David Sissom (West Texas A&M University, Canyon, TX; Figs. 23.1, 23.2, 23.6, 23.9 and 23.10−23.13, and scorpion on book cover); Rachel Stone (Wichita State University, Wichita, KS; Fig. 9.9); Daniel R. Suiter (University of Georgia, Griffin, GA; Fig. 6.2); Robert B. Tesh (University of Texas Medical Branch, Galveston, TX; Fig. 8.5); Gerrit Uilenberg (Corsica, France; Figs. 27.28, 27.29, 27.31 and 27.32); Richard Urbanek (US Fish and Wildlife Service; Figs. 14.7 and 14.8); Richard S. Vetter (University of California, Riverside, CA; Figs. 25.12, 25.15, 25.19 and 25.20); P. Kirk Visscher (University of California, Riverside, CA; Fig. 13.7); Jan Votýpka (Czech Academy of Science, Czech Republic; Fig. 14.19); Laurel L. Walters (Lieen-Follican Research, Bishop, CA; Fig. 12.13); Wesley D. Watson (University of North Carolina, Raleigh, NC; Fig. 9.10); Jerry Weintraub (Agriculture and Agri-Food Canada, Lethbridge, AB; Figs. 19.28 and 19.31); Julian White (Toxicology Department, Women's & Children's Hospital, North Adelaide, Australia; Figs. 25.8 and 25.23); Ralph E. Williams (Purdue University, West Lafayette, IN; Fig. 17.17); Germano Woehl, Jr. (Instituto Rã-bugio para Conservação da Biodiversidade, Jaraguá do Sul, Brazil; Fig. 21.16); and Jennifer M. Zaspel (Milwaukee Public Museum, Milwaukee, WI; Figs. 21.21 and 21.23).

Appreciation is also extended to the following institutions and agencies for their courtesy in granting permission to reproduce images, providing information, and otherwise affording helpful assistance to us: American Museum of Natural History, New York, NY; Auburn University College of Veterinary Medicine, Department of Pathobiology, Auburn, AL; Entomological Society of America, Annapolis, MD; The Carter Center, Onchocerciasis Elimination Program for the Americas (OEPA), Atlanta, GA; Centers for Disease Control and Prevention (CDC) and Public Health Image Library (PHIL); Creative Commons; Department of Agriculture and Agri-Food Canada, Lethbridge, AB; Food and Agricultural Organization of the United Nations, Rome, Italy; The Joint Pathology Center (JPC), US Federal Government, Silver Spring, MD; National Geographic Society, Washington, DC; New York Entomological Society, New York, NY; The Natural History Museum, London; Otis Historical Archives (OHA), National Museum of Health and Medicine, Silver Spring, MD; The Rockefeller Foundation, New York, NY; Southeastern Cooperative Wildlife Disease Study (SCWDS), University of Georgia, Athens, GA; University of Florida/IFAS Extension, Gainesville, FL; US Armed Forces Institute of Pathology (AFIP), disestablished in 2011; US Department of Agriculture (USDA), including the Animal and Plant Health Inspection Service (APHIS), Animal Research Service (ARS) at Kerrville, TX, and Laramie, WY, Wildlife Services (WS), and Foreign Animal Disease Diagnostic Laboratory (Plum Island, NY); US National Tick Collection, Georgia Southern University, Statesboro, GA; US Forest Service; US Public Health Service; The Wellcome Collection, London; Wikimedia Commons; and World Health Organization (WHO), Vector Control and Prevention Programme.

It is our special privilege to recognize and thank the following six previous chapter authors of both the first and second editions of this book who are not contributors to the third edition: Raj K. Gupta (Walter Reed Army Institute of Research, Silver Spring, MD); Robert D. Hall (University of Missouri, Columbia, MO); Robert S. Lane (University of California, Berkeley, CA); Louis C. Rutledge (US Army, retired, Mill Valley, CA); Scott A. Stockwell (US Army, retired, Lubbock, TX); also, Gerritt Uilenberg (Corsica, France), as a chapter author of the second edition. Their invaluable contributions continue to be reflected in this new edition of *Medical and Veterinary Entomology.*

At the same time, we are pleased to welcome as new contributing authors the following 12 individuals: Christopher M. Barker, Ramón Cepeda-Palacios, Douglas D. Colwell, Lawrence J. Hribar, Leonard E. Munstermann, C. Steven Murphree, Pia Untalan Olafson, Will K. Reeves, Justin O. Schmidt, W. David Sissom, Rebecca Trout Fryxell, and Jennifer M. Zaspel.

In addition, we honor the memory of the following four chapter authors of the first edition of this book: Roger D. Akre and E. Paul Catts (Department of Entomology, Washington State University, Pullman, WA), Robert Traub (US Army, retired, Bethesda, MD); and John E. "Jack" Lloyd (University of Wyoming, Laramie, WY), a contributor to the first and second editions, who died March 25, 2017, while co-revising his chapter for the third edition. We dedicate this book to their memory and to the many other medical and veterinary entomologists who have devoted their careers to protecting humans and animals from injurious arthropods and vector-borne diseases.

And, finally, we gratefully acknowledge Kristi A. Gomez (Acquisitions Editor), Pat Gonzalez (Editorial Project Manager), Punitha Radjane (Senior Project Manager), Swapna Praveen (Permissions Coordinator), Allison Esposito (Copyeditor), and the fine staff and support personnel at Elsevier, Inc., who have worked so closely with us in preparing and publishing the third edition of this book.

Gary R. Mullen
Lance A. Durden

This page intentionally left blank

Chapter 1

Introduction

Lance A. Durden[1] and Gary R. Mullen[2]

[1]*Department of Biology, Georgia Southern University, Statesboro, GA, United States;* [2]*Department of Entomology and Plant Pathology, Auburn University, Auburn, AL, United States*

Medical entomology is the study of insects, insect-borne diseases, and other associated problems that affect humans and public health. **Veterinary entomology** is the study of insects and insect-related problems that affect domestic animals, particularly livestock and companion animals (dogs, cats, horses, caged birds, etc.). In addition, veterinary entomology includes insect-associated problems affecting captive animals in zoological parks and in wildlife in general. **Medical-veterinary entomology** combines these two disciplines.

Traditionally, the fields of medical and veterinary entomology have included health-related problems involving arachnids (particularly mites, ticks, spiders, and scorpions). This broad approach that encompasses insects and arachnids is followed in this text. Alternatively, the study of health-related problems involving arachnids is called **medical-veterinary arachnology** or, if just mites and ticks are considered, **medical-veterinary acarology**.

Historically, both medical and veterinary entomology have played major roles in the development of human civilization and animal husbandry. Outbreaks of insect-borne diseases of humans have profoundly influenced human history; such diseases include yellow fever, plague, louse-borne typhus, malaria, African trypanosomiasis, Chagas disease, and lymphatic filariasis. Likewise, livestock scourges such as bovine babesiosis, bovine theileriosis, scabies, pediculosis, and botfly infestations, all of which are caused or transmitted by arthropods, have greatly influenced animal production and husbandry practices. Arthropod-related disorders continue to cause significant health problems to humans, domestic animals, and wildlife. At the same time, new strains of known pathogens, as well as previously unrecognized disease agents transmitted by arthropods, are causing newly recognized diseases (e.g., Lyme disease and human granulocytic anaplasmosis) and the resurgence of diseases that had been suppressed for many years (e.g., malaria, Chikungunya fever, and Zika encephalitis). Emerging and resurging arthropod-borne diseases are recognized as a growing health concern by public health and veterinary officials (Wilson and Spielman, 1994; Walker et al., 1996; Gubler, 1998; Winch, 1998; and Gratz, 1999; Marcondes, 2016).

GENERAL ENTOMOLOGY

Basic concepts of entomology such as morphology, taxonomy and systematics, developmental biology, and ecology provide important background information for medical and veterinary entomologists. General entomology books that the reader will find helpful in this regard include those by Gillot (1995), Elzinga (2000), Chapman (1998), Romoser and Stoffolano (1998), Gullan and Cranston (2005), Triplehorn and Johnson (2005), and Pedigo and Rice (2009). References that provide a more taxonomic or biodiversity-oriented approach to general entomology include works by Arnett (2000), Richards and Davies (1994), Bosik (1997), Daly et al. (1998), and Marshall (2006). General insect morphology is detailed in Snodgrass (1993), whereas a useful glossary of general entomology is provided by Torre-Bueno (1962) and was updated and revised by Nichols (1989). An encyclopedia of entomology (Resh and Cardé, 2009) and a dictionary of entomology (Gordh and Headrick, 2001) are also available. Texts on **urban entomology**, the study of insect pests in houses, buildings, and urban areas, which also has relevance to medical-veterinary entomology, have been prepared by Ebeling (1975), Hickin (1985), Mallis et al. (2004), and

Medical and Veterinary Entomology. https://doi.org/10.1016/B978-0-12-814043-7.00001-7

Robinson (1996). General texts on acarology include works by Woolley (1987), Evans (1992), and Krantz and Walter (2009).

MEDICAL-VETERINARY ENTOMOLOGY LITERATURE

Textbooks or monographs pertaining to medical entomology, veterinary entomology, or the combined discipline of medical-veterinary entomology are listed under these headings at the end of this chapter. Most of these publications emphasize arthropod morphology, biology, systematics, and disease relationships, whereas some texts emphasize molecular aspects of medical-veterinary entomology, such as Crampton et al. (1997) and Marquardt et al. (2005). Other works are helpful regarding common names of arthropods of medical-veterinary importance (Pittaway, 1992), surveillance techniques (Bram, 1978), control measures (Drummond et al., 1988), repellents (Debboun et al., 2007; Strickman et al., 2009), or ectoparasites (Andrews, 1977; Marshall, 1981; Kim, 1985; Uilenberg, 1994; Barnard and Durden 1999; Clayton et al., 2015). Publications that devote substantial sections to arthropods associated with wildlife and the pathogens they transmit include those by Davidson et al. (1981), Fowler (1986), Davidson and Nettles (1997), and Samuel et al. (2001).

Several journals and periodicals are devoted primarily to medical and/or veterinary entomology. These include:

- *Journal of Medical Entomology*, published for the Entomological Society of America by Oxford University Press.
- *Medical and Veterinary Entomology*, published by the Royal Entomological Society (UK).
- *Journal of Vector Ecology*, published by the Society of Vector Ecologists.
- *Review of Medical and Veterinary Entomology*, published by CAB International.
- *Annals of Medical Entomology*, published in Bhopal, India.
- *Vector-Borne and Zoonotic Diseases*, published in the United States by Mary Ann Liebert, Inc.

Journals specializing in parasitology, tropical medicine, or wildlife diseases that also publish articles on medical-veterinary entomology include:

- *Parasitology*, published by the British Society for Parasitology.
- *Journal of Parasitology*, published by the American Society of Parasitologists.
- *Parasite-Journal de la Société Française de Parasitologie*, published in France.
- *Advances in Disease Vector Research*, published by Springer-Verlag.
- *Bulletin of the World Health Organization*, published by the World Health Organization.
- *Journal of Wildlife Diseases*, published by the Wildlife Disease Association.
- *Emerging Infectious Diseases*, published by the Centers for Disease Control and Prevention (CDC).
- *American Journal of Tropical Medicine and Hygiene*, published by the American Society of Tropical Medicine and Hygiene.
- *Memorias Do Instituto Oswaldo Cruz*, published in Brazil.

Various Internet websites pertaining to medical-veterinary entomology also can be accessed for useful information. The CDC in Atlanta, Georgia, USA, provides two helpful resources: (1) a pictorial key to arthropods of public health importance, available as downloadable files (PDF), https://www.cdc.gov/nceh/ehs/Publications_Keys.htm; and (2) a comprehensive annual guide titled the "Yellow Book" that details travel medicine updates on a global basis (CDC, 2018). Although all aspects of travel medicine and infectious diseases are covered, much of the information addresses vector-borne diseases.

A BRIEF HISTORY OF MEDICAL-VETERINARY ENTOMOLOGY

Problems caused by biting and annoying arthropods and the pathogens they transmit have been the subject of writers since antiquity (Service, 1978). Homer (mid−eighth century BC), Aristophanes (c. 448−380 BC), Aristotle (384−322 BC), Plautus (c. 254−184 BC), Columella (5 BC−AD 65), and Pliny (AD 23−79) all wrote about the nuisance caused by flies, mosquitoes, lice, and/or bedbugs. However, the study of modern medical-veterinary entomology is usually recognized as beginning in the late 19th century, when blood-sucking arthropods were first proved to be vectors of human and animal pathogens.

Englishman **Patrick Manson** (1844−1922) (Fig. 1.1A) was the first to demonstrate pathogen transmission by a blood-feeding arthropod. Working in China in 1877, he showed that the mosquito *Culex pipiens fatigans* is a vector of *Wuchereria bancrofti*, the causative agent of Bancroftian filariasis. After this landmark discovery, the role of various blood-feeding arthropods in transmitting pathogens was recognized in relatively rapid succession.

In 1891, Americans **Theobald Smith** (1859−1934) (Fig. 1.1B) and **Frederick L. Kilbourne** (1858−1936) implicated the cattle tick, *Rhipicephalus* (*Boophilus*) *annulatus*, as a vector of *Babesia bigemina*, the causative agent of Texas cattle fever (bovine babesiosis). This paved

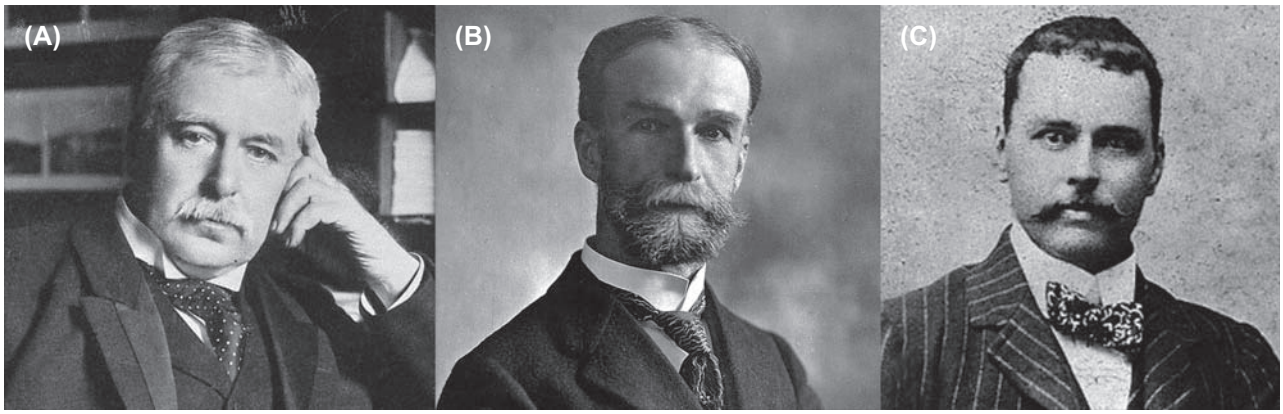


FIGURE 1.1 Historical figures in medical-veterinary entomology. (A) Patrick Manson (1844–1922). (B) Theobald Smith (1859–1934). (C) Ronald Ross (1857–1932). *A and B, Courtesy of The Wellcome Collection, London: C, Courtesy of Wikipedia Commons.*

the way for a highly successful *R. annulatus* eradication program in the United States directed by the US Department of Agriculture (USDA). The eradication of this tick resulted in the projected goal — elimination of indigenous cases of Texas cattle fever throughout the southern United States.

In 1898, Englishman Sir **Ronald Ross** (1857–1932) (Fig. 1.1C), working in India, demonstrated the role of mosquitoes as vectors of avian malarial parasites from diseased to healthy sparrows. Also in 1898, the cyclical development of malarial parasites in anopheline mosquitoes was described by Italian **Giovani Battista Grassi** (1854–1925). In the same year, Frenchman **Paul Louis Simond** (1858–1947), working in Pakistan (then part of India), showed that fleas are vectors of the bacterium that causes plague.

In 1848, American physician **Josiah Nott** (1804–1873) of Mobile, Alabama, published circumstantial evidence that led him to believe that mosquitoes were involved in the transmission of yellow fever virus to humans. In 1881, the Cuban-born Scottish physician **Carlos Finlay** (1833–1915) (Fig. 1.2A) presented persuasive evidence for his theory that what we know today as the yellow fever mosquito, *Aedes aegypti*, was the vector of this virus. However, it was not until 1900 that American **Walter Reed** (1851–1902) (Fig. 1.2B) led the US Yellow Fever Commission at Havana, Cuba, which proved *A. aegypti* to be the principal vector of yellow fever virus.

In 1903, Englishman **David Bruce** (1855–1931) (Fig. 1.2C) demonstrated the ability of the tsetse fly *Glossina palpalis* to transmit, during blood feeding, the trypanosomes that cause African trypanosomiasis.

Other important discoveries continued well into the 20th century. In 1906, American **Howard Taylor Ricketts** (1871–1910) (Fig. 1.3) proved that the Rocky Mountain wood tick, *Dermacentor andersoni*, is a vector of *Rickettsia rickettsii*, the causative agent of Rocky Mountain spotted fever. In 1907, **F. Percival Mackie** (1875–1944) showed that human body lice are vectors of *Borrelia recurrentis*, the spirochete that causes louse-borne (epidemic) relapsing fever. In 1908, Brazilian **Carlos Chagas** (1879–1934) (Fig. 1.4) demonstrated transmission of the agent that

FIGURE 1.2 Historical figures in medical-veterinary entomology. (A) Carlos Finlay (1833–1915). (B) Walter Reed (1851–1902). (C) David Bruce (1855–1931). *A and C, Courtesy of The Wellcome Collection, London: B, Courtesy of Wikipedia Commons.*

FIGURE 1.3 Howard Taylor Ricketts (1871–1910). *Courtesy of The Wellcome Collection, London.*

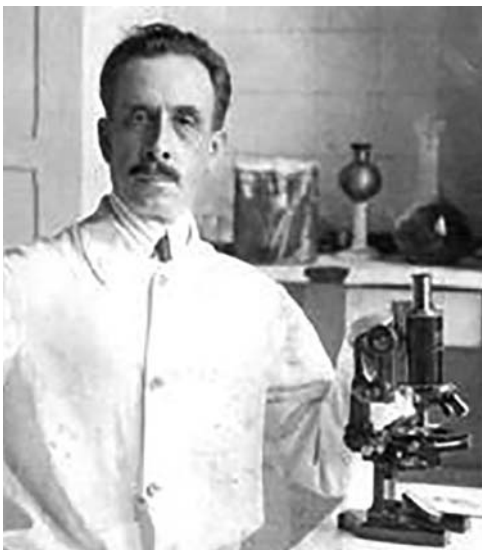


FIGURE 1.4 Carlos Chagas (1879–1934). *Courtesy of Wikipedia Commons.*

causes American trypanosomiasis, later named Chagas disease in his honor, by the conenose bug *Panstrongylus megistus*. In 1909, Frenchman **Charles Nicolle** (1866–1936), working in Tunis, showed that human body lice are vectors of *Rickettsia prowazekii*, the agent of louse-borne (epidemic) typhus.

These important discoveries, as well as others of historic relevance to medical-veterinary entomology, are discussed in more detail in the references listed at the end of this chapter. Because of the chronology of many major discoveries relevant to this topic in the 50-year period starting in 1877, this time has been called the "golden age of medical-veterinary entomology" (Philip and Rozeboom, 1973).

IDENTIFICATION AND SYSTEMATICS OF ARTHROPODS OF MEDICAL-VETERINARY IMPORTANCE

Table 1.1 provides a list of the eight orders of insects and four orders of arachnids that are of particular interest to medical-veterinary entomologists. Accurate identification of these arthropods is an important first step in determining the types of problems they can cause and, subsequently, in implementing control programs.

Although taxonomy and identification are discussed in more detail with respect to arthropod groups treated in the chapters that follow, some publications provide a broader perspective on the classification, taxonomy, and identification of a range of arthropods of medical-veterinary importance. These include two works published by the CDC (1979, 1994), as well as Service (1988), Hopla et al. (1994), Lago and Goddard (1994), and Davis (1995). Also, some medical-veterinary entomology books are very taxonomically oriented, with emphasis on identification—for example, Baker et al. (1956), Smith (1973), Lane and Crosskey (1993), and Walker (1995).

TYPES OF PROBLEMS CAUSED BY ARTHROPODS

Annoyance

Regardless of their role as blood-feeders (hematophages), parasites, or vectors of pathogens, certain arthropods cause severe annoyance to humans or other animals because of their biting behavior. These arthropods include lice, bedbugs, fleas, deer flies, horse flies, tsetse flies, stable flies, mosquitoes, black flies, biting midges, sand flies, chiggers, and ticks. Some arthropods, however, do not bite but instead are annoying because of their abundance, small size, or habit of flying into or around the eyes, ears, and nose. Nonbiting arthropods that cause annoyance include the house fly, chironomid midges, and eye gnats. Large populations of household or filth-associated arthropods such as houseflies and cockroaches also can be annoying. Nuisance arthropods are commonly problems for humans at outdoor recreational areas such as parks, lakes, and beaches.

TABLE 1.1 Principal Orders of Insects and Arachnids of Medical-Veterinary Interest

Order	Common Names
Class Insecta	
Order Blattaria	Cockroaches
Order Phthiraptera	Lice
Order Hemiptera	True bugs: bedbugs, kissing bugs, assassin bugs
Order Coleoptera	Beetles
Order Siphonaptera	Fleas
Order Diptera	Flies: mosquitoes, black flies, no-see-ums, horse flies, deer flies, sand flies, tsetse flies, house flies, stable flies, horn flies, bot flies, blow flies, flesh flies, louse flies, keds, etc.
Order Lepidoptera	Moths and butterflies
Order Hymenoptera	Wasps, hornets, velvet ants, ants, bees
Class Arachnida	
Order Scorpionida	Scorpions
Order Solpugida	Solpugids, sun spiders, camel spiders, barrel spiders
Order Acari	Mites, ticks
Order Araneae	Spiders

Toxins and Venoms

Several terms are used when discussing chemical substances that have adverse effects on humans and other animals. A **poison** is any substance that when taken into the body interferes with normal physiological functions. A **toxin** is a poison of plant or animal origin, which can result in a pathological condition called **toxicosis**. A **venom** is a poisonous mixture of compounds containing one or more toxins, which is produced in venom glands and injected into animal tissues via specialized morphological structures (e.g., stings, modified spines, and chelicerae in arthropods). The act of injecting venom into animal tissues is called **envenomation**.

Toxins produced by arthropods represent a wide range of chemical substances, from simple inorganic or organic compounds to complex alkaloids and heterocyclic compounds. Venoms often contain various pharmacologically active compounds that facilitate the spread and effectiveness of the toxic components. The compounds commonly include amines (e.g., histamine, catecholamines, serotonin), peptides, polypeptides (e.g., kinins), specific proteins, and enzymes (e.g., phospholipase, hyaluronidase, esterases) that vary significantly among different arthropod taxa. Depending on what types of cells or tissues they affect, toxins and venoms can be characterized as, for example, neurotoxins, cytotoxins, or hemotoxins. Frequently they cause symptoms such as pain, itching, swelling, redness, hemorrhaging, or blisters, the severity of which is largely dependent on the particular types and amounts of toxins involved.

For further information and details about arthropod toxins and venoms, see Chapter 3.

Allergic Reactions

A relatively wide spectrum of allergic reactions can occur in humans or animals exposed to certain arthropods. Bites or stings by arthropods such as lice, bedbugs, fleas, bees, ants, wasps, mosquitoes, and chiggers can result in allergic host reactions. Contact allergies can occur when certain beetles or caterpillars touch the skin. Respiratory allergies can result from inhaling allergenic airborne particles from cockroaches, fleas, dust mites, and other arthropods. The recirculation of air by modern air-handling systems in buildings tends to exacerbate inhalation of insect allergens. For reviews of arthropod allergens, see Arlian (2002) and Prester (2012).

Humans and other animals usually react to repeated exposure to bites or stings from the same or antigenically related arthropods in one of two ways, depending on the nature of the antigen or venom inoculated and the sensitivity of the host: (1) desensitization to the bites or stings with repeated exposure and (2) allergic reactions that, in extreme cases, can develop into life-threatening anaphylactic shock. However, a distinct five-stage sequence of

reactions typically occurs in most cases in which one is repeatedly bitten or stung by the same, or related, species of arthropod over time. Stage 1 involves no skin reaction but leads to the development of **hypersensitivity**. Stage 2 is a **delayed-hypersensitivity reaction**. Stage 3 is an **immediate-sensitivity** reaction followed by a delayed-hypersensitivity reaction. Stage 4 is an immediate reaction only, whereas stage 5 again involves no reaction (i.e., the victim becomes **desensitized**). These changes reflect the changing host immune response to prolonged and frequent exposure to the same arthropod or to cross-reactive allergens or venoms.

Invasion of Host Tissues

Some arthropods invade the body tissues of their host. Varying degrees of invasion occur, ranging from subcutaneous infestations to invasion of organs such as the lungs and intestines. Invasion of tissues allows arthropods to exploit different host niches and usually involves the immature stages of parasitic arthropods.

The invasion of host tissues by fly larvae, called **myiasis**, is the most widespread form of host invasion by arthropods. Larvae of many myiasis-causing flies move extensively through the host tissues. As the larvae mature, they select characteristic host sites (e.g., stomach, throat, nasal passages, or various subdermal sites) in which to complete the parasitic phase of their development.

Certain mites also invade the skin or associated hair follicles and dermal glands. Others infest nasal passages, lungs and air sacs, cloaca, stomach, intestines, and other parts of the alimentary tract of their hosts. Examples of these mites include scabies mites, follicle mites, nasal mites, lung mites, and a variety of other mites that infest both domestic and wild birds and mammals.

ARTHROPOD-BORNE DISEASES

Table 1.2 lists the principal groups of insects and arachnids involved in arthropod-borne diseases and the associated types of pathogens. Among the wide variety of arthropods that transmit pathogens to humans and other animals, mosquitoes are the most important, followed by ticks. Viruses and bacteria (including rickettsiae) are the most diverse groups of pathogens transmitted by arthropods, followed by protozoa and filarial nematodes. A standardized nomenclature has been proposed for parasitic diseases of animals including those with arthropod vectors (Kassai et al., 1988).

All of the viruses listed in Table 1.2 are arthropod-borne viruses, usually referred to as **arboviruses**, which indicates that they are typically transmitted by insects or other arthropod hosts. The study of arboviruses is termed **arbovirology**. These and related terms are discussed in more detail in Chapter 4 on the epidemiology of vector-borne diseases and in the Appendix devoted to arboviruses.

Pathogens are transmitted by arthropods in two basic ways: either biologically or mechanically. In **biological transmission**, pathogens undergo development or reproduction in the arthropod host. Examples of diseases that involve biological transmission are malaria, African trypanosomiasis, Chagas disease, leishmaniasis, and lymphatic filariasis. In **mechanical transmission**, pathogens are transmitted by arthropods via contaminated appendages (usually mouthparts) or regurgitation of an infectious blood meal. Examples of diseases that involve mechanical transmission are equine infectious anemia and myxomatosis. Biological transmission is by far the more common and efficient mechanism for pathogen maintenance and transmission.

A wide range of life-cycle patterns and degrees of host associations are characterized by arthropod vectors. Some ectoparasites, such as sucking lice, remain on their host for life. Others, such as mosquitoes and most biting flies, have a more fleeting association with the host, with some being associated with the host only during the brief acts of host location and blood-feeding. Between these two extremes is a wide range of host associations exhibited by different arthropod groups.

Literature references on vector-borne diseases, together with their epidemiology and ecology, are provided in the section "Arthropod-borne Diseases" at the end of this chapter.

FOOD CONTAMINANTS

Many arthropods can contaminate or spoil food materials. In addition, to causing direct damage to food resources, arthropods or their parts (e.g., setae, scales, shed cuticles, or body fragments) may be accidentally ingested. This can lead to toxic or allergic reactions, gastrointestinal myiasis, and other disorders. At least one case of millipedes (*Nopoiulus kochii*) infesting human intestines, for several years, has been documented (Ertek et al., 2004).

Insects such as the house fly may alight on food and regurgitate pathogen-contaminated fluids before or during feeding. While feeding they also may defecate, contaminating the food with potential pathogens. Because the alimentary tract of arthropods may harbor pathogenic microorganisms, subsequent consumption of the contaminated food can lead to the transmission of these pathogens to humans or other animals. Similarly, the integument of household pests such as flies and cockroaches (particularly their legs and tarsi) can serve as a contact source of pathogens that may be readily transferred to food items. Some of these arthropods previously may have visited fecal matter, garbage and other decomposing matter, animal

TABLE 1.2 Examples of Arthropod-Borne Diseases of Medical-Veterinary Importance

Arthropod Vectors	Diseases Grouped by Causative Agents
Mosquitoes	VIRUSES: yellow fever, dengue, Rift Valley fever, myxomatosis; Eastern equine encephalomyelitis, Western equine encephalomyelitis, Venezuelan equine encephalomyelitis, St. Louis encephalitis, LaCrosse encephalitis, Japanese encephalitis, Murray Valley encephalitis, Chikungunya fever, Onyong nyong fever, Ross River fever, West Nile fever, Zika encephalitis PROTOZOANS: malaria FILARIAL NEMATODES: Wuchererian filariasis, Bancroftian filariasis, dog heartworm
Black flies	FILARIAL NEMATODES: human onchocerciasis (river blindness), bovine onchocerciasis
Biting midges	VIRUSES: bluetongue disease, epizootic hemorrhagic disease, African horse sickness, leucocytozoonosis, Oropouche fever FILARIAL NEMATODES: equine onchocerciasis, mansonellosis
Sand flies	VIRUSES: sand fly fever, vesicular stomatitis. BACTERIA: Oroya fever (Veruga Peruana) PROTOZOANS: leishmaniasis
Horse flies and deer flies	VIRUSES: equine infectious anemia, hog cholera BACTERIA: tularemia PROTOZOANS: surra (livestock trypanosomiasis) FILARIAL NEMATODES: loiasis, elaeophorosis
Tsetse flies	PROTOZOANS: African trypanosomiasis, nagana
Triatomine bugs	PROTOZOANS: American trypanosomiasis (Chagas disease)
Lice	VIRUSES: swine pox. BACTERIA: epidemic typhus, trench fever, louse-borne relapsing fever
Fleas	VIRUSES: myxomatosis BACTERIA: plague, murine (endemic) typhus, tularemia, cat flea rickettsiosis, cat scratch disease
Ticks	VIRUSES: tick-borne encephalitis, Powassan encephalitis, Colorado tick fever, Crimean-Congo hemorrhagic fever, African swine fever BACTERIA: Lyme disease, Rocky Mountain spotted fever, Boutonneuse fever, tick-borne ehrlichiosis, Q fever, heartwater fever, anaplasmosis, tick-borne relapsing fever, avian spirochetosis, theileriosis (East Coast fever), bovine dermatophilosus PROTOZOANS: babesiosis
Mites	BACTERIA: tsutsugamushi fever (scrub typhus), rickettsialpox

For more comprehensive coverage, see the individual chapters devoted to each arthropod group.

secretions, or alternative potential sources of pathogens, thereby further contributing to health risks.

Additional information on insects and other arthropods that can contaminate food is provided by Olsen et al. (1996) and in reviews by Terbush (1972), Hughes (1976), and Gorham (1975, 1991a, 1991b).

FEAR OF ARTHROPODS

Some people detest arthropods, or infestation by them, to such a degree that they suffer from **entomophobia**, the fear of insects; **arachnophobia**, the fear of spiders and other arachnids; or **acarophobia**, the fear of mites (including ticks). Showing concern or disapproval toward the presence of potentially injurious arthropods is probably a prudent and healthy reaction, but phobic behaviors reflect an unusually severe psychological response. Such persons exhibit more-than-normal fear when they encounter an arthropod, often resorting to excessive or obsessive measures to control the problem (e.g., overtreatment of themselves or their homes with insecticides and other chemical compounds).

DELUSIONAL DISORDERS

A psychological state occurs in which an individual mistakenly believes that he or she is being bitten by, or infested with, parasites. This is variously known as **delusory parasitosis, delusional parasitosis**, **delusions of parasitosis**, **Ekbom syndrome**, and **Eliott disease**. This condition is distinct from simply a fear, or phobia, of insects or other arthropods and represents a more deeply

rooted psychological problem. This delusional condition is most frequently experienced by middle-aged or elderly persons, particularly women, and is one of the more difficult situations in which entomologists may become involved.

Remarkable behavioral traits are sometimes attributed to the parasites by victims. These include descriptions of tiny animals jumping into the eyes when a room is entered or when a lamp is switched on. Some victims have failing eyesight; others may have real symptoms from other conditions such as psoriasis, which may be attributed to imagined parasites. Victims become convinced that the parasites are real, and they often consult a succession of physicians in a futile attempt to secure a diagnosis and satisfactory treatment to resolve the problem. Patients typically produce skin scrapings or samples of household materials (e.g., vacuumed debris from carpets, draperies, and window sills) that they believe contain the elusive parasites.

Victims of delusory disorders often turn to extension entomologists or medical entomologists as a last resort, out of frustration with being unable to resolve their condition through family physicians, allergists, and other medical specialists. Because patients are convinced that arthropods are present, they are usually reluctant to seek counseling or other psychiatric help. Dealing with these cases requires careful examination of submitted specimens, tact, and professional discretion on the part of the entomologist. Additional information on delusory parasitosis is provided by Driscoll et al. (1993), Koblenzer (1993), Kushon et al. (1993), Poorbaugh (1993), Webb (1993a, 1993b), Goddard (1995), and Hinkle (2000).

Morgellons disease is a term, coined in 2002, for a condition generally regarded by the medical community as delusional parasitosis. The name refers to a medical case that occurred in 1674. Also referred to as **unexplained dermopathy** ("unexplained skin disease"), Morgellons can manifest as a range of skin conditions, including crawling, biting, and stinging sensations; granules, fibers, or dark specks in, or emerging from, the skin; and rashes or sores. Some patients diagnosed with Morgellons disease also experience fatigue, joint pain, visual changes, short-term memory loss, or mental confusion. Although published reports and anecdotal accounts have suggested possible involvement, there is no firm evidence to date implicating insects, or other arthropods, as a direct cause. Morgellons disease has been reported worldwide, with a particular focus of attention in certain parts of the United States (e.g., California, Florida, and Texas). For further information about this disorder, see Koblenzer et al. (2006), Murase et al. (2006), Savely et al. (2006), and Pearson et al. (2012).

FORMICOPHILIA

An unusual human psychosexual disorder, called formicophilia, can involve insects. In such cases an individual experiences self-induced sexuoerotic arousal and orgasm when ants, cockroaches, or other small creatures (e.g., snails) are allowed to crawl, creep, or nibble on the body, notably, the genitalia, perianal area, or nipples (Dewaraja and Money, 1986; Dewaraja, 1987).

HOST DEFENSES

Humans and other animals have developed elaborate means to defend themselves against infestation by arthropods and infection by pathogens they may transmit. Both behavioral or immunological responses are used to resist infestation by arthropods. **Behavioral defenses** include evasive, offensive, or defensive action against biting flies such as mosquitoes, black flies, ceratopogonids, stable flies, and horse flies. Grooming and preening by animals (e.g., biting, scratching, or licking) are defensive behaviors used to reduce or prevent infestations by ectoparasites and other potentially harmful arthropods. Host **immunological defenses** against arthropods vary with different arthropods and with respect to previous exposure to the same or antigenically related taxa. Details concerning such host immune responses are beyond the scope of this book, but some general trends are noteworthy. Repeated feeding attempts by the same or antigenically cross-reactive arthropods often lead to fewer arthropods being able to feed successfully, reduced engorgement weights, greater mortality, and decreased fecundity of female arthropods. Widespread arthropod mortality rarely results. For more information concerning the types of host immune responses and cell types involved against various ectoparasites and pathogens, see Wikel (1996b), Boulanger (2018), and other works listed at the end of this chapter.

Many blood-feeding arthropods partially or completely counteract the host immune response by inoculating immunomodulators or immunosuppressive compounds into the bite site. In fact, a wide range of pharmacologically active compounds are known to be released at the bite site by various arthropods (Ribeiro, 1995). These compounds range from **anticoagulants** to prevent the blood from clotting, local **analgesics** to reduce host pain, **apyrase** to prevent platelet aggregation and promote capillary location, and various enzymes and other factors to promote blood or tissue digestion. Some of these compounds are perceived by the host as antigens and may elicit an immune response, whereas others can cause localized or systemic toxic responses and itching.

MINOR ARTHROPOD PROBLEMS OF MEDICAL-VETERINARY INTEREST

In addition to arthropod groups detailed in the chapters that follow, a few arthropods in other groups may have minor, incidental, or occasional significance to human and animal health. These include springtails (Order Collembola), mayflies (Order Ephemeroptera); grasshoppers, locusts, and crickets (Order Orthoptera); walkingsticks (Order Phasmatodea), earwigs (Order Dermaptera), thrips (Order Thysanoptera), bark lice and book lice (Order Pscoptera), caddisflies (Order Trichoptera), millipedes (Class Diplopoda), and centipedes (Class Chilopoda).

Some **walkingsticks**, or stick insects, possess glands that are used to spray defensive fluids at potential predators, such as ants, beetles, rodents, and birds, or when otherwise threatened. A pair of large, elongate glands are located in the anterior part of the thorax, where they open on the anterolateral margins of the pronotum, just behind the head. Two species in the United States that produce and spray defensive secretions are *Anisomorpha buprestoides* (Fig. 1.5) and *A. ferruginea*, called two-striped walkingsticks. They forcefully discharge an emulsion of malodorous, milky fluid, as a fine mist containing a terpene dialdehyde (anisomorphal) as the active ingredient. A Madagascan species, *Parectatosoma mocquerysi*, produces a similar defensive monoterpene compound (parectadial). In Taiwan, *Megacrania alpheus* has paired thoracic glands that secrete five volatile protective compounds including the defensive secretion actinidine (Chow et al., 1986).

A number of cases have been documented in which animals, particularly humans and dogs, have been sprayed in the eyes by these and other walkingstick species, from as far away as 30 cm. The result in severe cases is immediate, sometimes excruciating, pain, with burning and dull aching of the eye(s) for several hours and impaired vision that may persist for one to several days. Recommended treatment is immediate and thorough irrigation of the affected eye(s) with cool water, followed by administration of an analgesic.

FIGURE 1.5 Two-striped walkingstick (*Anisomorpha buprestoides*); a pair, with smaller male atop larger female; a common species in the southeastern United States that sprays a defensive secretion from a pair of thoracic glands, which can severely irritate the eyes of humans, dogs, and other animals. *Photograph by Aaron T. Dossey.*

For more information on the chemical aspects of defensive secretions in walkingsticks, see Meinwald et al. (1962), Happ et al. (1966), Carlberg (1985), Eisner (1965, 2005), and Dossey et al. (2008); for human cases, see Stewart (1937), Albert (1947), Hatch et al. (1993), and Paysse et al. (2001); and for canine cases, see Dziedzyc (1992) and Brutlag et al. (2011).

Although they are not commonly thought of as being of medical or veterinary concern, a number of orthopteran insects can cause harm. These groups include **grasshoppers**, **katydids**, and **crickets**, as reviewed by Hill and Goddard (2012). Because of their strong mandibles used for cutting and chewing plant materials, some of the larger species, such as the North American robust shieldback katydid (*Atlanticus gibbosus*), can inflict painful bites when carelessly handled; other species are capable of drawing blood (e.g., wetas of New Zealand; families Anostostomatidae and Rhaphidophoridae). When orthopterans are threatened or attempt to bite, the brown regurgitant that they produce can enter a cut or bite wound, causing irritation and pain. If this crop fluid comes in contact with the eyes of vertebrates, it can cause immediate discomfort and distress, as reported in cases involving the African migratory locust (*Locusta migratoria*).

Orthopterans also serve as intermediate hosts for a number of parasites, including the nematode *Tetrameres americana*, which commonly infests free-ranging chickens and other gallinaceous birds; the poultry tapeworm *Choanotaenia infundibulum*, which parasitizes certain grasshoppers; and other tapeworms such as *Metroliasthes lucida*, which parasitize turkeys and guinea fowl. In other cases, orthopterans have been implicated in allergic reactions among personnel working in insectaries where locusts are reared and in outbreaks of inhalant allergies and asthma attacks, as reported during plagues of locusts in Sudan and other parts of Africa. Symptoms include rhinitis, bronchitis, and difficulty breathing due to inhalations of microscopic particles of locusts or their dried feces. Although based primarily on circumstantial evidence, some orthopterans also are regarded as potential mechanical vectors of certain animal pathogens (e.g., causative agents of cholera and vesicular stomatitis).

Springtails, or collembolans, have been reported to infest human and pet skin (Scott et al., 1962; Scott, 1966; Beccati et al., 2011). However, in most cases in which springtails are found on, or adhering to, the skin of humans and household pets (e.g., dogs and cats), they are believed to be incidental associations, most likely due to contact of the skin with soil, compost, decomposing plant material, or moist ground debris where springtails typically live. It is

not surprising, therefore, to find them as contaminants of moist dermal lesions and other skin problems, where they can survive for at least short periods of time. This said, the association of collembolans with cases of delusory parasitosis (Altschuler et al., 2004) has led to considerable controversy over the interpretation of this association, which may represent immunological and histaminic reactions to contact with springtails (Christinsen and Bernard, 2008; Lim et al., 2009).

Some **bark lice** (psocids) are known to cause allergies or dermatitis in humans (Li and Li, 1995; Baz and Monserrat, 1999), whereas adult **mayflies** and **caddisflies** can cause inhalational allergies, especially when they emerge in large numbers from lakes, rivers, or streams (Seshadri, 1955). Some North American mayflies and caddisfies are intermediate hosts of trematodes that can be infected with *Neorickettsia risticii*, the causative agent of Potomac horse fever (equine monocytic ehrlichiosis). Accidental ingestion of infected mayflies and caddisflies by horses is a mode of transmission for this pathogen (Madigan et al., 2000).

Thrips, which have tubular mouthparts adapted for sucking plant fluids, occasionally pierce the skin and have been known to imbibe blood (Williams, 1921; Hood, 1927; Bailey, 1936; Arnaud, 1970). When local populations are particularly high, as in the case of the flower thrips (*Frankliniella tritici*), they have been documented causing significant discomfort at outdoor gatherings in the southeastern United States. On rare occasions, **earwigs** have been recorded imbibing blood (Bishopp, 1961). Bishopp further noted that some earwigs have been known to pierce human skin with their pair of caudal pincers (cerci) and may stay attached for an extended period.

Some miscellaneous arthropods inhabit the feathers of birds or the fur of mammals. The exact nutritional requirements of some of these arthropods remain unknown; most of them, however, do not appear to be true ectoparasites. Representatives of two of the three suborders of earwigs (Suborders Arixeniina and Hemimerina) live in mammalian fur. Members of the Arixeniina are associated with Old World bats, whereas members of the Hemimerina are found on African cricetine rodents (Nakata and Maa, 1974). These earwigs may feed on skin secretions or sloughed cells, but their effect on the health of their hosts is poorly understood. Other occasional inhabitants of host pelage, such as various beetles, cheyletid mites, and pseudoscorpions, are predators of ectoparasites and therefore are beneficial to their hosts (Durden, 1987).

A few arthropods that are not mentioned in the following chapters can occasionally serve as intermediate hosts of parasites that adversely affect domestic and wild animals. These include certain springtails and psocids (bark lice) as intermediate hosts of tapeworms (Baz and Monserrat, 1999).

Occasionally, entomologists are asked questions about millipedes and centipedes. Defensive sprays of some millipedes contain hydrochloric acid or hydrogen cyanide, which can chemically burn the skin and can cause long-term skin discoloration (Radford, 1975; Marek and Bond, 2009). Centipedes, especially some of the larger tropical species, can cause envenomation when they "bite" with their claws (maxillipeds), onto which open the ducts of their venom glands (Remington, 1950).

REFERENCES AND FURTHER READING

General Entomology

Arnett, R. H., Jr. (2000). *American insects: A handbook of the insects of America north of Mexico* (2nd ed.). Boca Raton: CRC Press.

Bosik, J. J. (1997). *Common names of insects and related organisms*. Lanham, MD: Entomological Society of America (This list is updated electronically and can be accessed through the website for the Entomological Society of America).

Chapman, R. F. (1998). *The insects: Structure and function* (4th ed.). Cambridge University Press.

Daly, H. V., Doyen, J. T., & Purcell, A. H., III (1998). *Introduction to insect biology and diversity* (2nd ed.). Oxford University Press.

Ebeling, W. (1975). *Urban entomology*. Berkeley: University of California Press, 695 pp.

Elzinga, R. J. (2000). *Fundamentals of entomology* (5th ed.). Upper Saddle River, NJ: Prentice Hall.

Gillott, C. (1995). *Entomology* (2nd ed.). Plenum Press, 744 pp.

Gordh, G., & Headrick, D. H. (2001). *A dictionary of entomology*. New York: CABI Publishing.

Grimaldi, D., & Engel, M. S. (2005). *Evolution of the insects*. Cambridge University Press.

Gullan, P. J., & Cranston, P. S. (2014). *The insects: An outline of entomology* (5th ed.). New York: Wiley.

Mallis, A., Hedges, S. A., & Moreland, D. (2004). *Handbook of pest control: The behavior, life history and control of household pests* (9th ed.). Mallis Handbook & Technical Training Co.

Marshall, S. A. (2006). *Insects: Their natural history and diversity with a photographic guide to insects of Eastern North America*. Richmond Hill, Ontario: Firefly Books.

Nichols, S. W. (1989). *The Torre-Bueno glossary of entomology*. New York: New York Entomological Society and American Museum of Natural History.

Pedigo, L. P., & Rice, M. E. (2009). *Entomology and pest management* (6th ed.). Long Grove, IL: Waveland Press Inc.

Resh, V. H., & Cardé (Eds.). (2009). *Encyclopedia of entomology* (2nd ed.). San Diego: Academic Press.

Richards, O. W. & Davies, R.G. (1994). Imm's general textbook of entomology (10th ed.). Vol. 1. Structure, physiology and development, Vol. 2. Classification and biology, Chapman & Hall.

Robinson, W. H. (1996). *Urban entomology: Insect and mite pests in the human environment*. Chapman & Hall.

Romoser, W. S., & Stoffolano, J. G., Jr. (1998). *The science of entomology* (4th ed.). Boston: WCB/McGraw Hill.

Snodgrass, R. (1993). *Principles of insect morphology*. Cornell University Press.

de la Torre-Bueno, J. R. (1962). *A glossary of entomology*. Brooklyn: Brooklyn Entomological Society.

Triplehorn, C. A., & Johnson, N. F. (2005). *Borror and DeLong's introduction to the study of insects* (7th ed.). Belmont, CA: Thomson, Brooks/Cole.

General Acarology

Evans, G. O. (1992). *Principles of acarology*. Wallingford, UK: CAB International.

Krantz, G. W., & Walter, D. E. (Eds.). (2009). *A manual of acarology* (3rd ed.). Lubbock: Texas Tech University Press.

Woolley, T. A. (1987). *Acarology: Mites and human welfare*. New York: Wiley.

Medical-Veterinary Entomology

Baker, E. W., Evans, T. M., Gould, D. J., Hull, W. B., & Keegan, H. L. (1956). *A manual of parasitic mites of medical or economic importance*. New York: National Pest Control Assoc.

Baker, J. R., Apperson, C. S., & Arends, J. J. (1986). *Insect and other pests of man and animals*. North Carolina State University.

Clayton, D. H., Bush, S. E., & Johnson, K. P. (2015). *Coevolution of life on hosts: Integrating ecology and history*. University of Chicago Press.

Crampton, J. M., Beard, C. B., & Louis, C. (Eds.). (1997). *Molecular biology of insect disease vectors: A methods manual*. Chapman & Hall.

Debboun, M., Frances, S. P., & Strickman, D. (Eds.). (2007). *Insect repellents: Principles, methods and uses*. Boca Raton: CRC Press.

Duvallet, G., Fontenille, D., & Robert, V. (Eds.). (2017). *Entomologie Médicale et Veterinaire*. Marseille: Quae, Versailles and IRD.

Eldridge, B. F., & Edman, J. D. (Eds.). (2003). *Medical entomology: A textbook on public health and veterinary problems caused by arthropods* (2nd ed.). Norwell, MA: Kluwer Academic.

Harwood, R. F., & James, M. T. (1979). *Entomology in human and animal health* (7th ed.). New York: Macmillan.

Hickin, N. E. (1985). *Pest animals in buildings: A world review* (G. Godwin, London; Longman, New York).

Jobling, B. (1987). *Anatomical drawings of biting flies*. London & Wellcome Trust: British Museum (Natural History).

Kettle, D. S. (1995). *Medical and veterinary entomology* (2nd ed.). Wallingford, UK: CAB International.

Kim, K. C. (Ed.). (1985). *Coevolution of parasitic arthropods and mammals*. Wiley.

Lehane, M. (2005). *Biology of blood-sucking insects* (2nd ed.). Cambridge University Press.

Mario Vargas, V. (2001). *Los acaros en la salud humana y animal*. Universidad de Costa Rica: San Juan.

Marquardt, W. C. (Ed.). (2005). *Biology of disease vectors* (2nd ed.). Burlington, MA: Elsevier.

Marquardt, W. C., Demaree, R. S., & Grieve, R. B. (1999). *Parasitology and vector biology* (2nd ed.). San Diego: Harcourt Academic Press.

Marshall, A. G. (1981). *Ecology of ectoparasitic insects*. London: Academic Press.

Nutting, W.B. (Ed.). (1994). Mammalian diseases and arachnids. Vol. I. Pathogen biology and clinical management, Vol. II. Medico-veterinary, laboratory, and wildlife diseases, and control. CRC Press, Boca Raton.

Parish, L. C., Nutting, W. B., & Schwartzman, R. M. (Eds.). (1983). *Cutaneous infestations in man and animals*. New York: Praeger Press.

Patton, W. S. (1931). *Insects, ticks, mites and venomous animals of medical and veterinary importance. Part II: Public health*. Liverpool University Press.

Patton, W. S., & Evans, A. M. (1929). *Insects, ticks, mites and venomous animals of medical and veterinary importance. Part I: Medical*. Liverpool University Press.

Pittaway, A. R. (1992). *Arthropods of medical and veterinary importance: A checklist of preferred names and allied terms*. Tucson: University of Arizona Press.

Robinson, W. H. (2005). *Urban insects and arachnids: A handbook of urban entomology*. Cambridge University Press.

Russell, R. C., Otranto, D. P., & Wall, R. L. (2013). *The encyclopedia of medical and veterinary entomology*. CAB Interntional.

Walker, A. R. (1995). *Arthropods of humans and domestic animals: A guide to preliminary identification*. New York: Chapman & Hall.

Medical Entomology

Alexander, J. O. (1984). *Arthropods and human skin*. Berlin: Springer-Verlag.

Andrews, M. L. A. (1977). *The life that lives on man*. New York: Taplinger Publishing.

Burgess, N. H. R., & Cowan, G. L. O. (1993). *A colour atlas of medical entomology*. Chapman & Hall.

Busvine, J. M. (1980). *Insects and hygiene: The biology and control of insect pests of medical and domestic importance* (3rd ed.). London: Chapman & Hall.

Daniel, M., Stramova, H., Absolonova, V., Dedicova, D., Lhotova, H., Maskova, L., et al. (1992). Arthropods in a hospital and their potential significance in the epidemiology of hospital infections. *Folia Parasitologica, 39*, 159–170.

Furman, D. P., & Catts, E. P. (1982). *Manual of medical entomology*. Cambridge University Press.

Goddard, J. (2012). *Physician's guide to arthropods of medical importance* (6th ed.). Boca Raton: CRC Press.

Goddard, J. (2012). *Public health entomology*. Boca Raton: CRC Press.

Goddard, J. (1998). Arthropods and medicine. *Journal of Agromedicine, 5*, 55–83.

Gordon, R. M., & Lavoipierre, M. M. J. (1962). *Entomology for students of medicine*. Oxford: Blackwell Scientific.

Gratz, N. G. (1999). Emerging and resurging vector-borne diseases. *Annual Review of Entomology, 44*, 51–75.

Gubler, D. J. (1998). Resurgent vector-borne diseases as a global health problem. *Emerging Infectious Diseases, 4*, 442–450.

Harwood, R. F., & James, M. T. (1979). *Entomology in human and animal health* (7th ed.). New York: Macmillan.

Herms, W. B. (1961). *Medical entomology* (5th ed.). New York: Macmillan.

Horsfall, W. R. (1962). *Medical entomology: Arthropods and human disease*. New York: Ronald Press.

James, M. T., & Harwood, R. F. (1969). *Herms' medical entomology* (6th ed.). New York: Macmillan.

Lane, R. P., & Crosskey, R. W. (Eds.). (1993). *Medical insects and arachnids*. Chapman & Hall.

Leclercq, M. (1969). *Entomological parasitology: The relations between entomology and the medical sciences*. Oxford: Pergamon Press.

Marples, M. J. (1965). *The ecology of the human skin*. Springfield, IL: Charles C. Thomas.

Matheson, R. (1950). *Medical entomology*. Ithaca: Comstock Publishing Co.

McClelland, G. A. H. (1992). *Medical entomology: An ecological perspective* (12th ed.). Davis: University of California Press.

Orkin, M., & Maibach, H. I. (Eds.). (1985). *Cutaneous infestations and insect bites*. New York: Marcel Dekker.

Peters, W. (1992). *A colour atlas of arthropods in clinical medicine*. London: Wolfe Publishing Ltd.

Riley, W. A., & Johannsen, O. A. (1938). *Medical entomology: A survey of insects and allied forms which affect the health of man and animals* (2nd ed.). New York: McGraw-Hill.

Service, M. W. (1980). *A guide to medical entomology*. London: Macmillan.

Service, M. W. (2012). *Medical entomology for students* (5th ed.). Cambridge University Press.

Smith, K. G. V. (Ed.). (1973). *Insects and other arthropods of medical importance*. London: British Museum (Natural History).

Strickman, D., Francis, S. P., & Debboun, M. (2009). *Prevention of bug bites, stings, and disease*. Oxford University Press.

Walker, D. H., Barbour, A. G., Oliver, J. H., Jr., Lane, R. S., Dumler, J. S., Dennis, D. T., et al. (1996). Emerging bacterial zoonotic and vector-borne diseases. *Journal of the American Medical Association, 275*, 463−469.

Vector-borne terrestrial diseases. In Wilson, M. E., & Spielman, A. (Eds.), *Ann. NY Acad. Sci.: Vol. 740. Disease in evolution: Gobal changes and emergence of infectious diseases, pp. 123−224*, (pp. 1−503). (1994) (pp. 1−503).

Winch, P. (1998). Social and cultural responses to emerging vector-borne diseases. *Journal of Vector Ecology, 23*, 47−53.

Veterinary Entomology

Axtell, R. C., & Arends, J. J. (1990). Ecology and management of arthropod pests of poultry. *Annual Review of Entomology, 35*, 101−126.

Barker, B. (1999). *Livestock entomology laboratory manual* (2nd ed.). Dubuque, IA: Kendall/Hunt.

Barnard, S. M., & Durden, L. A. (1999). A veterinary guide to the parasites of reptiles. In *Arthropods (excluding mites)* (Vol. 2). Melbourne, FL: Krieger Press.

Bay, D. E., & Harris, R. L. (1988). *Introduction to veterinary entomology*. Bryan, TX: Stonefly Publishing.

Bowman, D. D. (1999). *Georgi's parasitology for veterinarians* (7th ed.). Philadelphia: W. B. Saunders.

Bram, R. A. (1978). *Surveillance and collection of arthropods of veterinary importance*. U.S. Dep. Agric, Agriculture Hdbk. No. 518.

Drummond, R. O., George, J. E., & Kunz, S. E. (1988). *Control of arthropod pests of livestock: A review of technology*. Boca Raton: CRC Press.

Flynn, R. J. (1973). *Parasites of laboratory animals*. Ames: Iowa State University Press, 884 pp.

Foil, L. D., & Foil, C. S. (1990). Arthropod pests of horses. *Compendium: Continuing Education for Veterinarians, 12*, 723−731.

Garros, C., Bouyer, J., Takken, W. & Smallegange, R. C. (2018). Pests and vector-borne diseases in the livestock industry. Ecology and control of vector-borne diseases. Vol. 5. Wageningen Academic Publishers.

Georgi, J. R. (1990). *Parasitology for veterinarians* (5th ed.). Philadelphia: W. B. Saunders.

Guimarães, J. H., Tucci, E. C., & Barros-Battesti, D. M. (2001). *Ectoparasitos de importância veterinária*. Fundação de Amparo á Pesquisa do Estado de São Paulo.

Jones, C. J., & DiPietro, J. A. (1996). Biology and control of arthropod parasites of horses. *Compendium: Continuing Education for Veterinarians, 18*, 551−558.

Lancaster, J. L., & Meisch, M. V. (1986). *Arthropods in livestock and poultry production*. Halstead Press/Wiley.

Mullens, B. A., Hinkle, N. C., Trout Fryxell, R., & Rochon, K. (2018). Past, present, and future contributions and needs for veterinary entomology in the United States and Canada. *American Entomologist, 64*, 20−31.

Soulsby, E. J. L. (1982). *Helminths, arthropods and protozoa of domesticated animals* (7th ed.). London: Bailliere, Tindall and Cassell.

Uilenberg, G. (1994). Ectoparasites of animals and control methods. *Revue Scientifique et Technique international Office of Epizootics, 13*, 979−1387.

Wall, R., & Shearer, D. (1997). *Veterinary entomology*. Chapman & Hall.

Williams, R. E. (2009). *Veterinary entomology: Livestock and companion animals*. Boca Raton: CRC Press.

Williams, R. E., Hall, R. D., Broce, A. B., & Scholl, P. J. (Eds.). (1985). *Livestock entomology*. Wiley.

Wildlife Entomology

Davidson, W. R., Hayes, F. A., Nettles, V. F., & Kellogg, F. E. (1981). *Diseases and parasites of white-tailed deer*. Tallahassee, FL: Misc. Pub. No. 7, Tall Timbers Res. Stn.

Davidson, W. R., & Nettles, V. F. (1997). *Field manual of wildlife diseases in the southeastern United States* (2nd ed.). Athens, GA: Southeastern Cooperative Wildlife Disease Study.

Fowler, M. E. (Ed.). (1986). *Zoo and wild animal medicine* (2nd ed.). Philadelphia: W. B. Saunders.

Fowler, M. E., & Miller, R. E. (Eds.). (1999). *Zoo and wild animal medicine: Current therapy* (4th ed.). Philadelphia: W. B. Saunders.

Samuel, W. M., Pybus, M. J., & Kocan, A. A. (2001). *Parasitic diseases of wild mammals* (2nd ed.). Ames: Iowa State University Press.

History of Medical-Veterinary Entomology

Anon. (1996). History of CDC [CDC's 50th anniversary]. *Morbidity and Mortality Weekly Report, 45*, 526−530.

Augustin, G. (1909). *History of yellow fever*. New Orleans: Searcy & Pfaff.

Bayne-Jones, S. (1964). Communicable diseases, arthropod-borne diseases other than malaria. In *Preventive medicine in World war II* (Vol. VII). Washington DC: Office of the Surgeon General, Dept. of the Army.

Bean, W. B. (1982). *Walter reed - a biography*. Charlottesville: University of Virginia Press.

Bockarie, M. J., Gbakima, A. A., & Barnish, G. (1999). It all began with Ronald Ross: 100 years of malaria research and control in Sierra Leone (1899−1999). *Annals of Tropical Medicine and Parasitology, 93*, 213−224.

Busvine, J. R. (1976). *Insects, hygiene and history*. London: Athlone Press.

Busvine, J. R. (1993). *Disease transmission by insects: Its discovery and 90 years of effort to prevent it*. Berlin: Springer-Verlag.

Calisher, C. H. (1996). From mouse to sequence and back to mouse: Peregrinations of an arbovirologist. *Journal of Vector Ecology, 21*, 192−200.

Cartwright, F. F. (1972). *Disease and history; the influence of disease in shaping the great events in history*. London: Hart-Davis.

Chernin, E. (1983). Sir Patrick Manson: An annotated bibliography and a note on a collected set of his writings. *Reviews of Infectious Diseases, 5*, 353−386.

Chernin, E. (1987). A unique tribute to Theobald Smith, 1915. *Reviews of Infectious Diseases, 9*, 625–635.
Cirillo, V. J. (2011). "Wonders unconceived:" reflections on the birth of medical entomology. *Perspectives in Biology and Medicine, 54*, 381–398.
Collins, W. E. (1976). Fifty years of parasitology: Some fulgent personalities in arthropodology. *The Journal of Parasitology, 62*, 504–509.
Cook, G. C. (1992). *From the Greenwich hulks to old St. Pancras: A history of tropical disease in London*. London: Athlone Press.
Cox, F. E. G. (Ed.). (1996). *The Wellcome Trust illustrated history of tropical diseases*. London: Trustees of the Wellcome Trust.
Cushing, E. C. (1957). *History of entomology in world war II*. Washington DC: Smithsonian Inst.
Delaporte, F. (1991). *The history of yellow fever: An essay on the birth of tropical medicine*. MIT Press.
Desowitz, R. S. (1991). *The malaria capers: More tales of parasites and people, research and reality*. New York: W. W. Norton & Co.
Desowitz, R. S. (1997). *Who gave Pinta to the Santa Maria? Tracing the devastating spread of lethal tropical diseases*. New York: W. W. Norton & Co.
Dolman, C. E. (1982). Theobald Smith (1859-1934), pioneer American microbiologist. *Perspectives in Biology and Medicine, 25*, 417–427.
Eldridge, B. F. (1992). Patrick Manson and the discovery age of vector biology. *Journal of the American Mosquito Control Association, 8*, 215–222.
Ellis, J. H. (1992). *Yellow fever and public health in the New South*. Lexington: University of Kentucky Press.
Gillett, J. D. (1979). Vitamin C, yellow fever and plague; the near misses. *Antenna, Bulletin Royal Entomology Society of London, 3*, 64–70.
Gillett, J. D. (1985). Medical entomology, past, present and future: A personal view. *Antenna, Bulletin Royal Entomology Society of London, 9*, 63–70.
Gorgas, M. D., & Hendrick, B. J. (1924). *William Crawford Gorgas: His life and work*. Garden City, NY: Garden City Publishing Co.
Harwood, R. F., & James, M. T. (1979). Historical review. In *Entomology in human and animal health* (7th ed., pp. 3–10). Macmillan Publishing Co.
Horsman, R. (1987). *Josiah Nott of Mobile: Southerner, physician, and racial theorist*. Baton Rouge: Louisiana State University Press.
Laurence, B. R. (1989). The discovery of insect-borne disease. *Biologist, 36*, 65–71.
Lewis, J. D. (1984). Reminiscences of medical entomology in the last fifty years. *Antenna, Bulletin Royal Entomology Society of London, 8*, 117–122.
Lockwood, J. A. (1987). Entomological warfare: History of the use of insects as weapons of war. *American Entomologist, 33*, 76–82.
Lockwood, J. A. (2009). *Six-legged soldiers: Using insects as weapons of war*. Oxford University Press.
Miller, G. L. (1997). Historical natural history: Insects and the Civil War. *American Entomologist, 43*, 227–245.
Nye, E. R., & Gibson, M. E. (1997). *Ronald Ross: Malariologist and polymath - a biography*. New York: Macmillan/St. Martin's.
Oldstone, M. B. A. (1998). *Viruses, plagues, and history*. Oxford: Oxford University Press.
Packard, R. M. (2007). *The making of a tropical disease: A short history of malaria*. Baltimore: Johns Hopkins University Press.
Peterson, R. K. D. (1995). Insects, disease, and military history. *American Entomologist, 41*, 147–160.
Philip, C. B. (1948). Tsutsugamushi disease (scrub typhus) in World War II. *The Journal of Parasitology, 34*, 169–191.
Philip, C. B., & Rozeboom, L. E. (1973). Medico-veterinary entomology: A generation of progress. In R. F. Smith, T. E. Mittler, & C. N. Smith (Eds.), *History of entomology* (pp. 333–360). College Park: Entomological Society of America.
Schmidt, C. H., & Fluno, J. A. (1973). Brief history of medical and veterinary entomology in the USDA. *Journal of the Washington Academy of Sciences, 63*, 54–60.
Service, M. W. (1978). A short history of medical entomology. *Journal of Medical Entomology, 14*, 603–626.
Slater, L. B. (2009). *War and disease: Biomedical research on malaria in the twentieth century*. Piscatway, NJ: Rutgers University Press.
Sosa, O., Jr. (1989). Carlos J. Finlay and yellow fever: A discovery. *Bulletin of the Entomological Society of America, 35*, 23–25.
Woodward, T. E. (1973). A historical account of the rickettsial diseases with a discussion of unsolved problems. *The Journal of Infectious Diseases, 127*, 583–594.
Young, M. D. (1966). Scientific exploration and achievement in the field of malaria. *The Journal of Parasitology, 52*, 2–8.
Zinsser, H. (1935). *Rats, lice and history*. Boston: Little, Brown & Co.
Zinsser, H. (1936). Biographical memoir of Theobald Smith, 1859-1934. *Biographical Memoirs. National Academy of Sciences, 17*, 261–303 [reprinted in *Rev. Inf. Dis*. 9, 636-654].

IDENTIFICATION AND SYSTEMATICS OF TAXA OF MEDICAL-VETERINARY IMPORTANCE

CDC. (1979). *Introduction to arthropods of public health importance*. Atlanta: Centers for Disease Control.
CDC. (1994). *Pictorial keys: Arthropods, reptiles, birds and mammals of public health importance*. Atlanta: Centers for Disease Control and Prevention.
Davis, G. M. (1995). Systematics and public health. *BioScience, 45*, 705–714.
Hopla, C. E., Durden, L. A., & Keirans, J. E. (1994). Ectoparasites and classification. *Revue Scientifique et Technique Office International des Epizooties, 13*, 985–1017.
Lago, P. K., & Goddard, J. (1994). Identification of medically important arthropods. *Laboratory Medicine, 25*, 298–305.
Service, M. W. (Ed.). (1988). *Biosystematics of haematophagous insects*. Oxford University Press.

TYPES OF PROBLEMS CAUSED BY ARTHROPODS

Annoyance

Burns, D. A. (1987). The investigation and management of arthropod bite reactions acquired in the home. *Clinical and Experimental Dermatology, 12*, 114–120.
Frazier, C. D. (1973). Biting insects. *Archives of Dermatology, 107*, 400–402.
Newson, H. D. (1977). Arthropod problems in recreation areas. *Annual Review of Entomology, 22*, 333–353.

Allergic Reactions

Arlian, L. G. (2002). Arthropod allergens and human health. *Annual Review of Entomology, 47*, 395–433.

Bellas, T. E. (1982). *Insects as a cause of inhalant allergies: A bibliography* (2nd ed.). Canberra: CSIRO Aust. Div. Entomol. Rep. No. 25. CSIRO.

Carlsen, K. C., Carlsen, L. K. H., Buchmann, M. S., Wikstrom, J., & Mehl, R. (2002). Cockroach sensitivity in Norway: A previously unidentified problem? *Allergy, 57*, 529−533.

Feinberg, A. R., Feinberg, S. M., & Benaim-Pinto, C. (1956). Asthma and rhinitis from insect allergens. *Journal of Allergy, 27*, 436−444.

Feingold, B. F., Benjamini, E., & Michaeli, D. (1968). The allergic responses to insect bites. *Annual Review of Entomology, 13*, 137−158.

Frazier, C. A., & Brown, F. K. (1980). *Insects and allergy and what to do about them*. Norman: University of Oklahoma Press.

Henson, E. B. (1966). Aquatic insects as inhalant allergens; a review of American literature. *Ohio Journal of Science, 66*, 529−532.

Heyworth, M. F. (1999). Importance of insects in asthma. *Journal of Medical Entomology, 36*, 131−132.

Levine, M. I., & Lockley, R. F. (Eds.). (1981). *Monograph on insect allergy*. Pittsburgh: Typecraft.

Musken, H., Franz, J. T., Fernandez-Caldas, E., Maranon, F., Masuch, G., & Bergmann, K. C. (1998a). Psocoptera spp. (dust lice): A new source of indoor allergies in Germany. *The Journal of Allergy and Clinical Immunology, 101*, 121.

Musken, H., Franz, J. T., Fernandez-Caldas, E., Masuch, G., Maranon, F., & Bergmann, K. C. (1998b). Psocoptera (dust lice): New indoor allergens? *Allergologie, 21*, 381−382.

Prester, L. (2012). Arthropod allergies in urban homes. *Archives of Industrial Hygiene and Toxicology, 63*(Suppl. 1), 46−56.

Schulman, S. (1967). Allergic responses to insects. *Annual Review of Entomology, 12*, 323−346.

Wirtz, R. A. (1980). Occupational allergies to arthropods - documentation and prevention. *Bulletin of the Entomological Society of America, 26*, 356−360.

Wirtz, R. A. (1984). Allergic and toxic reactions to non-stinging arthropods. *Annual Review of Entomology, 29*, 47−69.

Arthropod-Borne Diseases

Beck, J. W., & Davies, J. E. (1981). *Medical parasitology*. C. V. Mosby Co.

Benenson, A. S. (Ed.). (1995). *Control of communicable diseases manual* (16th ed.). American Public Health Assoc.

Beran, G. W., & Steele, J. (1994). *Handbook of zoonoses: Section a. Bacterial, rickettsial, chlamydial, and mycotic* (2nd ed.). Boca Raton: CRC Press.

Busvine, J. R. (1979). *Arthropod vectors of disease. The Institute of Biology's Studies in biology No. 55*. London: Edward Arnold.

CDC. (2018). *Yellow book: Health information for international travel*. Oxford University Press.

Cook, G. C. (Ed.). (1996). *Manson's tropical diseases* (20th ed.). Orlando: W. B. Saunders.

Dye, C. (1992). The analysis of parasite transmission by bloodsucking insects. *Annual Review of Entomology, 37*, 1−19.

Ewald, P. W. (1983). Host-parasite relations, vectors and the evolution of disease severity. *Annual Review of Ecology, Evolution, and Systematics, 14*, 465−485.

Faust, E. C., Beaver, B. C., & Jung, R. C. (1975). *Animal agents and vectors of human disease* (4th ed.). Philadelphia: Lea & Febiger.

Goddard, J. (1999). *Infectious diseases and arthropods*. Humana Press.

Gratz, N. (2006). *Vector- and rodent-borne diseases in Europe and north America: Distribution, public health burden, and control* (p. 410). Cambridge: Cambridge University Press.

Horsfall, F. L., & Tamm, I. (Eds.). (1965). *Viral and rickettsial infections of man*. Philadelphia: J. B. Lippincott.

Hubbert, W. T., McCullough, W. F., & Schnurrenberger, P. R. (Eds.). (1975). *Diseases transmitted from animals to man* (6th ed.). Charles Thomas.

Jeffrey, H. C., Leach, R. M., & Cowan, G. O. (1991). *Atlas of medical helminthology and protozoology*. New York: Churchill Livingstone.

Kassai, T., Cordero del Campillo, M., Euzeby, J., Gafaar, S., Hiepe, T., & Himonas, C. A. (1988). Standardized nomenclature of animal parasitic diseases (SNOAPAD). *Veterinary Parasitology, 29*, 299−326.

Marcondes, C. B. (Ed.). (2016). *Arthropod-borne diseases*. Switzerland: Springer International Publishing, Cham.

McHugh, C. P. (1994). Arthropods: Vectors of disease agents. *Laboratory Medicine, 25*, 429−437.

Mills, J. N., Childs, J. E., Ksiazek, T. G., Peters, C. J., & Velleca, W. M. (1995). *Methods for trapping and sampling small mammals for virologic testing*. Atlanta: Centers for Disease Control & Prevention.

Monath, T. P. (Ed.). (1988). The arboviruses: epidemiology and ecology. Vol. I. General principles, Vol. II. African horse sickness to dengue, Vol. III. Eastern equine encephalomyelitis to O'nyong virus disease, Vol. IV. Oropouche fever to Venezuelan equine encephalomyelitis, Vol. V. Vesicular stomatitis to yellow fever, CRC Press, Boca Raton.

Moore, C. G., McLean, R. G., Mitchell, C. J., Nasci, R. S., Calisher, C. H., Marfin, A. A., et al. (1993). *Guidelines for arbovirus surveillance programs in the United States*. Fort Collins: Centers for Disease Control & Prevention.

Service, M. W. (1986). *Blood-sucking insects: Vectors of disease*. London: Edward Arnold.

Service, M. W. (Ed.). (1989). *Demography and vector-borne diseases*. Boca Raton: CRC Press.

Snow, K. R. (1974). *Insects and disease*. New York: Wiley.

Strickland, G. T. (Ed.). (1991). *Hunter's tropical medicine* (7th ed.). W. B. Saunders Company.

Theiler, M., & Downs, W. G. (1973). *The arthropod-borne viruses of vertebrates: An account of the Rockefeller Foundation virus program, 1951−1970*. New Haven: Yale University Press.

WHO. (1989). *Geographical distribution of arthropod-borne diseases and their principal vectors*. WHO/VBC/89.967.

WHO. (1995). *Vector control for malaria and other mosquito-borne diseases*. WHO Tech. Rep. Ser. No. 857.

Food Contaminants

Gorham, J. R. (1975). Filth in foods: Implications for health. *Journal of Milk and Food Technology, 38*, 409−418.

Gorham, J. R. (1991a). Insect and mite pests in food: An illustrated key. In *USDA Agric. Handbook No. 655* (Vols. 1 & 2).

Gorham, J. R. (Ed.). (1991b). *Ecology and management of food-industry pests*. Arlington, VA: FDA Tech. Bull. No. 4. AOAC International.

Hughes, A. M. (1976). The mites of stored food and houses. Ministry of Agriculture, Fisheries and food, her Majesty's Stationery Office, London. In A. R. Olsen, T. H. Sidebottom, & S. A. Knight (Eds.), *Fundamentals of microanalytical entomology; a practical guide to detecting and identifying filth in foods*. Boca Raton: CRC Press.

Terbush, L. E. (1972). The medical significance of mites of stored food. *FDA By-Lines, 3*, 57−70.

Delusional Disorders, Phobias, and Formicophilia

Altschuler, D. Z., Crutcher, M., Dulceanu, N., Cervantes, B. A., Terinte, C., & Sorkin, L. N. (2004). Collembola (springtails) (Arthropoda: Hexapoda: Entognatha) found in scrapings from individuals diagnosed with delusory parasitosis. *Journal of the New York Entomological Society, 112*, 87–95.

Beerman, H., & Nutting, W. B. (1984). Arachnid-related phobias: Symbiphobia, preventions, and treatments. In W. B. Nutting (Ed.), *Medico-veterinary, laboratory, and wildlife diseases, and control: Vol. II. Mammalian diseases and arachnids* (pp. 103–112). Boca Raton: CRC Press.

Christiansen, K. A., & Bernard, E. C. (2008). Critique of the article "Collembola (springtails) (Arthropoda: Hexapoda: Entgnatha) found in scrapings from individuals diagnosed with delusory parasitosis". *Entomological News, 119*, 537–540.

Dewarja, R., & Money, J. (1986). Transcultural sexology; formicophilia, a newly named paraphilia in a young Buddhist male. *Journal of Sex and Marital Therapy, 12*, 139–145.

Dewarja, R. (1987). Formicophilia, an unusual paraphlia treated with counseling and behavior therapy. *American Journal of Psychotherapy, 41*, 593–597.

Driscoll, M. S., Rothe, M. J., Grant-Kels, J. M., & Hale, M. S. (1993). Delusional parasitosis - a dermatological, psychiatric, and pharmacological approach. *Journal of the American Academy of Dermatology, 29*, 1023–1033.

Goddard, J. (1995). Analysis of 11 cases of delusions of parasitosis reported to the Mississippi Department of Health. *Southern Medical Journal, 88*, 837–839.

Hinkle, N. C. (2000). Delusory parasitosis. *American Entomologist, 46*, 17–25.

Koblenzer, C. S. (2006). The challenge of Morgellons disease. *Journal of the American Academy of Dermatology, 55*, 920–922.

Koblenzer, C. S. (1993). The clinical presentation, diagnosis and treatment of delusions of parasitosis - a dermatologic perspective. *Bulletin of the Society for Vector Ecology, 18*, 6–10.

Kushon, D. J., Helz, J. W., Williams, J. M., Lau, K. M. K., Pinto, L., & St Aubin, F. E. (1993). Delusions of parasitosis: A survey of entomologists from a psychiatric perspective. *Bulletin of the Society for Vector Ecology, 18*, 11–15.

Murase, J. E., Wu, J. J., & Koo, J. (2006). Morgellons disease: A rapport-enhancing term for delusions of parasitosis. *Journal of the American Academy of Dermatology, 55*, 913–914.

Pearson, M. L., Selby, J. V., Katz, K. A., Cantrell, V., Braden, C. R., Parise, M. E., et al. (2012). Clinical, epidemiologic, histopathalogic and molecular features of an unexplained dermopathy. *PLoS One, 7*, e29908.

Poorbaugh, J. H. (1993). Cryptic arthropod infestations: Separating fact from fiction. *Bulletin of the Society for Vector Ecology, 18*, 3–5.

Robles, D. T., Romm, S., Combs, H., Olson, J., & Kirby, P. (2008). Delusional disorders in dermatology: A brief review. *Dermatology Online Journal, 14*(6), 2.

Savely, V. R., Leitao, M. M., & Stricker, R. B. (2006). The mystery of Morgellons disease: Infection or delusion? *American Journal of Clinical Dermatology, 7*, 1–5.

Shelomi, M. (2013). Evidence of photo manipulation in a delusional parasitosis paper. *The Journal of Parasitology, 99*, 583–585.

Szepietowski, J. C., Salomon, J., Hrehorow, E., Pacan, P., Zalewska, A., & Sysa-Jedrzejowska, A. (2007). Delusional parasitosis in dermatological practice. *Journal of the European Academy of Dermatology and Venereology, 21*, 462–465.

Vloten, W. A. (1998). Delusions of parasitosis. A psychiatric disorder to be treated by dermatologists? An analysis of 33 patients. *British Journal of Dermatology, 138*, 1030–1032.

Walling, H. W., & Swick, B. L. (2007). Psychocutaneous syndromes: A call for revised nomenclature. *Clinical and Experimental Dermatology, 32*, 317–319.

Webb, J. P., Jr. (1993a). Delusions of parasitosis: A symposium; coordination among entomologists, dermatologists, and psychiatrists. *Bulletin of the Society for Vector Ecology, 18*, 1–2.

Webb, J. P., Jr. (1993b). Case histories of individuals with delusions of parasitosis in southern California and a proposed protocol for initiating effective medical assistance. *Bulletin of the Society for Vector Ecology, 18*, 16–25.

HOST DEFENSES

Barriga, O. O. (1981). Immune reactions to arthropods. In O. O. Barriga (Ed.), *The immunology of parasitic infections: A handbook for physicians, veterinarians, and biologists* (pp. 283–317). Baltimore: University Park Press.

Boulanger, N. (Ed.). (2018). *Skin and arthropod vectors*. Academic Press.

Nelson, W. A., Bell, J. F., Clifford, C. M., & Keirans, J. E. (1977). Interaction of ectoparasites and their hosts. *Journal of Medical Entomology, 13*, 389–428.

Nelson, W. A., Keirans, J. E., Bell, J. F., & Clifford, C. M. (1975). Host-ectoparasite relationships. *Journal of Medical Entomology, 12*, 143–166.

Ribeiro, J. M. C. (1995). Blood-feeding arthropods: Live syringes or invertebrate pharmacologists? *Infectious Agents and Disease, 4*, 143–152.

Wikel, S. K. (1982). Immune responses to arthropods and their products. *Annual Review of Entomology, 27*, 21–48.

Wikel, S. K. (1996a). Host immunology to ticks. *Annual Review of Entomology, 41*, 1–22.

Wikel, S. K. (Ed.). (1996b). *The immunology of host-ectoparasitic arthropod relationships*. Wallingford, UK: CAB International.

Wikel, S. K. (1999). Modulating the host immune system by ectoparasitic arthropods. *BioScience, 49*, 311–320.

MINOR ARTHROPOD PROBLEMS OF MEDICAL-VETERINARY INTEREST

Arnaud, P. H., Jr. (1970). Thrips "biting" man. *The Pan-Pacific Entomologist, 46*, 76.

Bailey, S. F. (1936). Thrips attacking man. *The Canadian Entomologist, 68*, 95–98.

Baz, A., & Monserrat, J. (1999). Distribution of domestic Psocoptera in Madrid apartments. *Medical and Veterinary Entomology, 13*, 259–264.

Beccati, M., Gallo, M. G., Chiavassa, E., & Peano, A. (2011). A case of apparent infestation by *Proisotoma* spp. Springtails (Collembola: Isotomidae) in a cat. *Veterinary Dermatology, 23*, 157–161.

Bishopp, F. C. (1961). Injury to man by earwigs. *Proceedings of the Entomological Society of Washington, 63*, 114.

Durden, L. A. (1987). Predator-prey interactions between ectoparasites. *Parasitology Today, 3*, 306–308.

Ertek, M., Aslan, I., Yazgi, H., Torun, H. C., Ayyildiz, A., & Tasyaran, M. A. (2004). Infestation of the human intestine by the millipede, *Nopoiulus kochii*. *Medical and Veterinary Entomology, 18*, 306–307.

Hood, J. D. (1927). A blood-sucking thrips. *Entomologist, 60*, 201.

Hunter-Jones, P. (1966). Allergy to animals: A zoological hazard. *New Scientist, 31*, 615–616.

Li, D.-N., & Li, J.-C. (1995). Report on human dermatitis caused by *Liposcelis divinatorius (Psocoptera)*. *Chinese Journal of Parasitology & Parasitic Diseases, 13*, 283 (In Chinese).

Lim, C. S. H., Lim, S. L., Chew, F. T., Ong, T. C., & Deharveng, L. (2009). Collembola are unlikely to cause human dermatitis. *Journal of Insect Science, 9*, 3.

Madigan, J. E., Pusterla, N., Johnson, E., Chae, J. S., Pusterla, J. B., Derock, E., & Lawler, S. P. (2000). Transmission of *Ehrlichia risticii*, the agent of Potomac horse fever, using naturally infected aquatic insects and helminth vectors: preliminary report. *Equine Veterinary Journal, 32*, 275–279.

Marek, P. E., & Bond, J. E. (2009). A Müllerian mimicry in Appalachian millipedes. *Proceedings of the National Academy of Sciences of the United States of America, 106*, 9755–9760.

Nakata, S., & Maa, T. C. (1974). A review of the parasitic earwigs (Dermaptera: Arixeniina; Hemimerina). *Pacific Insects, 16*, 307–374.

Radford, A. J. (1975). Millipede burns in man. *Tropical & Geographical Medicine, 27*, 279–287.

Remington, C. L. (1950). The bite and habits of a giant centipede *(Scolopendra subspinipes)* in the Philippine islands. *The American Journal of Tropical Medicine and Hygiene, 30*, 453–455.

Scott, H. G. (1966). Insect pests. Part I. *Springtails*. *Modern Maintenance Management, 18*, 19–21.

Scott, H. G., Wiseman, J. S., & Stojanovich, C. J. (1962). Collembola infesting man. *Annals of the Entomological Society of America, 55*, 428–430.

Seshadri, A. R. (1955). An extraordinary outbreak of caddis-flies (Trichoptera) in the Meltrudam township area of Salem district, South India. *South Indian Journal of Entomology, 3*, 337–340.

Williams, C. B. (1921). A blood-sucking thrips. *Entomologist, 54*, 163–164.

Walkingsticks

Albert, R. O. (1947). Another case of injury to the human eye by the walking stick, *Anisomorpha* (Phasmidae). *Entomological News, 58*, 57–59.

Brutlag, A., Hovda, L. R., & Della Ripa, M. A. (2011). Corneal ulceration in a dog following exposure to the defensive spray of a walkingstick insect (*Anisomorpha* spp.). *Journal of Veterinary Emergency and Critical Care, 21*, 382–386.

Carlberg, U. (1985). Chemical defence in *Anisomorpha buprestoides* (Houttuyn in Stoll') (Insecta: Phasmida). *Zoologischer Anzeiger, 215*, 177–188.

Chow, Y. S., & Lin, Y. M. (1986). Actinidine, a defensive secretion of stick insect, *Megacrania alpheus* Westwood (Orthoptera: Phasmatidae). *Journal of Entomological Science, 21*, 97–101.

Dossey, A. T., Walse, S. S., & Edison, A. S. (2008). Developmental and geographical variation in the chemical defense of the walkingstick insect *Anisomorpha buprestoides*. *Journal of Chemical Ecology, 34*, 584–590.

Dziedzyc, J. (1992). Insect defensive spray-induced keratitis in a dog. *Journal of the American Veterinary Association, 200*, 1969.

Eisner, T. (1965). Defensive spray of a phasmid insect. *Science, 148*, 966–968.

Eisner, T., Eisner, M., & Siegler, M. (2005). *Secret Weapons: Defenses of insects, spiders, Scorpions, and other many-legged creatures* (pp. 93–96). Cambridge, MA: Harvard Univ. Press.

Hatch, R. L., Lamsens, S. D., & Perchalski, J. E. (1993). Chemical conjunctivitis caused by spray of *A. buprestoides,* two-striped walking stick. *Journal of the Florida Medical Association, 80*, 758–759.

Meinwald, J., Chadha, M. S., Hurst, J. J., & Eisner, T. (1962). Defense methods of arthropods – IX. Anisomorphal, the secretion of a phasmid insect. *Tetrahedron Letters, 1*, 29–33.

Paysse, E. A., Holder, S., & Coats, D. K. (2001). Ocular injury from the venom of the southern walkingstick. *Ophthalmology, 108*, 190–191.

Stewart, M. A. (1937). Phasmid injury to the human eye. *The Canadian Entomologist, 69*, 84–86.

Orthopterans (Grasshoppers, Katydids, Crickets)

Fink, A., Permin, A., Jensen, V., Bresciani, J., & Magwisha, H. B. (2005). An experimental infection model for *Tetrameres americana* (Cram.). *Parasitology Research, 95*, 179–185.

Freeman, M. A. (1968). Pharmacological properties of the regurgitated crop fluid of the African migratory locust, *Locusta migratoria* L. *Comparative Biochemistry and Physiology, 26*, 1041–1049.

Hill, J. G., & Goddard, J. (2012). Medical and veterinary importance of grasshoppers, katydids, and crickets (Hexapoda: Orthoptera). *Journal of the Mississippi Academy of Sciences, 57*, 172–177.

Jones, B. R., Cullinane, L. C., & Cray, P. R. (1984). Isolation of *Listeria monocytogenes* from a bite in a cat from the common tree weta *(Hemideina crassidens)*. *New Zealand Veterinary Journal, 32*, 79.

McDougald, L. R. (2003). Cestodes and trematodes. In Y. M. Saif (Ed.), *Diseases of poultry*. Ames, IA: Blackwell Publishing.

Nunamaker, R. A., Lockwood, A. J., Stith, C. E., Cambell, C. L., Schell, S. P., Drolet, B. S., et al. (2003). Grasshoppers (Orthoptera: Acrididae) could serve as reservoirs and vectors of vesicular stomatitis virus. *Journal of Medical Entomology, 40*, 957–963.

Chapter 2

Morphological Adaptations of Parasitic Arthropods

Nathan D. Burkett-Cadena
University of Florida, Florida Medical Entomology Laboratory, Vero Beach, FL, United States

There are several independent lineages of arthropods that are parasitic on vertebrates in one or more stages of their life cycles (Fig. 2.1). Many morphological features of these parasitic arthropods are modified in a number of ways as adaptations for parasitic relationships. Common adaptations include those for feeding on vertebrate blood and other body fluids, attaching to and clinging to hosts, and dispersing to new hosts. Morphological structures that play a particularly important role in host associations are body shape, mouthparts, and legs. These structures are often highly modified in parasitic arthropods. Other features that are often modified in parasitic arthropods are the wings, eyes, and various sensory organs.

BODY SHAPE AND WINGS

A recurring theme among ectoparasites is modification of the general body shape to facilitate movement on the host and to enable them to hide in tight spaces when off the host. For parasitic lineages that exhibit long-term, close association with host animals, this usually involves dorsoventral or lateral flattening of the body. Dorsoventral flattening is characteristic of bed bugs and bat bugs, lice, beaver beetles (Leiodidae, genus *Platypsyllus*), parasitic dermapterans (genus *Hemimerus*), louse flies and keds, and ticks. In contrast, lateral flattening is best exemplified by fleas. Blood-feeding arthropods that have only brief contact with hosts, such as skin-piercing moths, mosquitoes, and biting midges, generally do not exhibit lateral or dorsoventral compression of the body.

Wings are indispensable features of many parasitic insects, without which they would not be able to reach their hosts. Tabanids (horse flies), for example, are strong fliers; some species may attain air speeds of 45 km/h or greater. In other parasitic insects, however, wings have become secondarily reduced, or even completely lost, as in fleas, lice, bed bugs, and the sheep ked. Still others, such as hippoboscids of the genus *Lipoptena* that parasitize deer, may have fully functional wings as adults but shed them after reaching a suitable host. In the latter case, the wings break off at a specific location near their base, leaving a wing stump.

MOUTHPARTS

Mouthparts of parasitic arthropods are typically adapted for feeding on host body fluids, particularly blood but also lymph, skin secretions, and tears. They also may be adapted for feeding externally on skin, sloughed skin scales, hair, or feathers. Those arthropods that feed directly on host tissues generally retain the chewing-type mouthparts like those of cockroaches (Fig. 2.2A). Fluid-feeding parasites, on the other hand, have mouthparts adapted for piercing host skin to reach and feed on internal fluids. It is among this group that the mouthparts have become the most modified and specialized. Arthropods that use their mouthparts to lacerate host skin and feed on blood that pools at the bite site as a result of damage to the surrounding blood vessels are called **telmophages**. Examples of telmophages include black flies, biting midges, horse flies, and deer flies. Bites from these insects typically cause more immediate pain and discomfort at the bite site due to puncture wounds and damage to surrounding skin and related tissues, resulting in a greater risk of secondary infections. Hematophagous arthropods with highly specialized piercing−sucking mouthparts that pierce individual capillaries and then feed directly on host blood are called **solenophages**. Examples of solenophages are mosquitoes, bed bugs, kissing bugs, and sucking lice. They represent the more refined blood feeders, with highly modified, styletiform mouthparts that typically leave little or no evidence of an actual puncture of the skin at the bite site.

Medical and Veterinary Entomology. https://doi.org/10.1016/B978-0-12-814043-7.00002-9

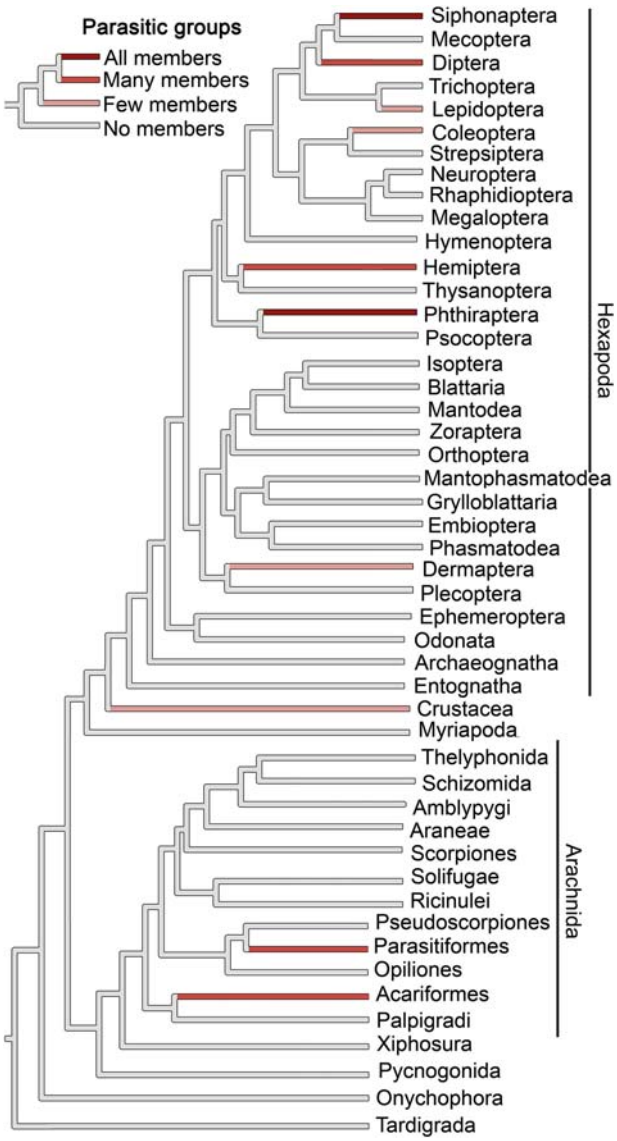


FIGURE 2.1 Arthropod phylogeny illustrating relationships of parasitic lineages. *Composite image redrawn after Regier et al. (2010), Sasaki et al. (2013), and Lozano-Fernandez et al. (2016).*

Biting midges are excellent examples of telmophages. The labrum, mandibles, maxillae, and hypopharynx are bladelike, pointed or serrate apically, and function in lacerating host skin (Fig. 2.2C). The labium serves as a sheath for protecting the other, more delicate, mouthparts. In insects with chewing mouthparts, the labium is analogous to a "lower lip" (Fig. 2.2A) and functions in grasping, manipulating, and retaining food.

Mosquitoes are classic examples of solenophages. The labrum, maxillae, mandibles, hypopharynx, and labium are very elongate, forming a feeding apparatus called the proboscis (Fig. 2.2B). The mosquito labium serves as a protective sheath and a guide for the fascicle, the bundle of thinner and more delicate mouthparts that penetrate the host skin, deliver saliva, and transport host blood. The fascicle is composed of the labrum, maxillae, mandibles, and hypopharynx, all of which are in the form of interconnecting fine stylets. The labrum, which functions as the "upper lip" of insects with chewing mouthparts (Fig. 2.2A), forms the blood-feeding tube in mosquitoes. The mandibles and maxillae, typically used for manipulating and masticating food by arthropods with chewing mouthparts (Fig. 2.2A), are modified in mosquitoes for piercing the host epidermis. The hypopharynx, a tongue-like structure in insects with chewing mouthparts (Fig. 2.2A), is also styletiform in mosquitoes and is used to pierce host tissue. Running the length of the hypopharynx is a channel that delivers saliva to the apical portion of the mouthparts during feeding.

In fleas, the epipharynx (an outgrowth of the body wall unique to fleas) and the maxillae are in the form of stylets and are used to pierce skin (Fig. 2.2D). During blood feeding, the tip of the epipharynx is inserted into a capillary. The pair of maxillae, together with the epipharynx, forms the feeding tube. In fleas the labium is reduced, with only the palps visible externally. The palps help to guide the blood-feeding stylets.

In blood-feeding hemipterans, such as bed bugs and kissing bugs, the maxillae and mandibles are styletiform and held within a sheathlike, segmented labium (Fig. 2.2E). Each of the paired maxillae is curved medially and interlocks with the other on their dorsal and ventral surfaces to form the food canal. The maxillae, mandibles, and labium form the rostrum, which is directed posteriorly and held under the head and thorax of the insect when not in use. The labrum is relatively unmodified and resembles the labrum of insects with chewing mouthparts.

In sucking lice the feeding apparatus is different from that of other parasitic insects. The labrum is highly modified to form a snoutlike structure, called the **haustellum**, which surrounds the other mouthparts (Fig. 2.2F). At the tip of the haustellum are prestomal, or haustellar, "teeth," which are used to anchor the mouthparts to the host. The maxillae, hypopharynx, and labium are modified as stylets for piercing host tissues. The hypopharynx also serves as a salivary canal, while the maxillae form the food canal.

Among the arachnids, ticks represent a highly specialized group of mites that are obligate parasites of terrestrial vertebrates. Ticks feed on host blood during each of their developmental stages (i.e., larvae, nymphs, and adults). The mouthparts are modified for piercing host tissue, anchoring the tick to its host and drawing blood into the alimentary tract. In most groups of mites, the tip of each chelicera is pincer-like, or **chelate**, with a fixed digit and opposable movable digit for grasping and manipulating food (Fig 2.3A). In ticks, however, the chelicerae have lost the terminal chelae and have become modified as short bladelike structures, with serrate tips, adapted for piercing host skin (Fig. 2.3C). The hypostome, a ventral projection of the

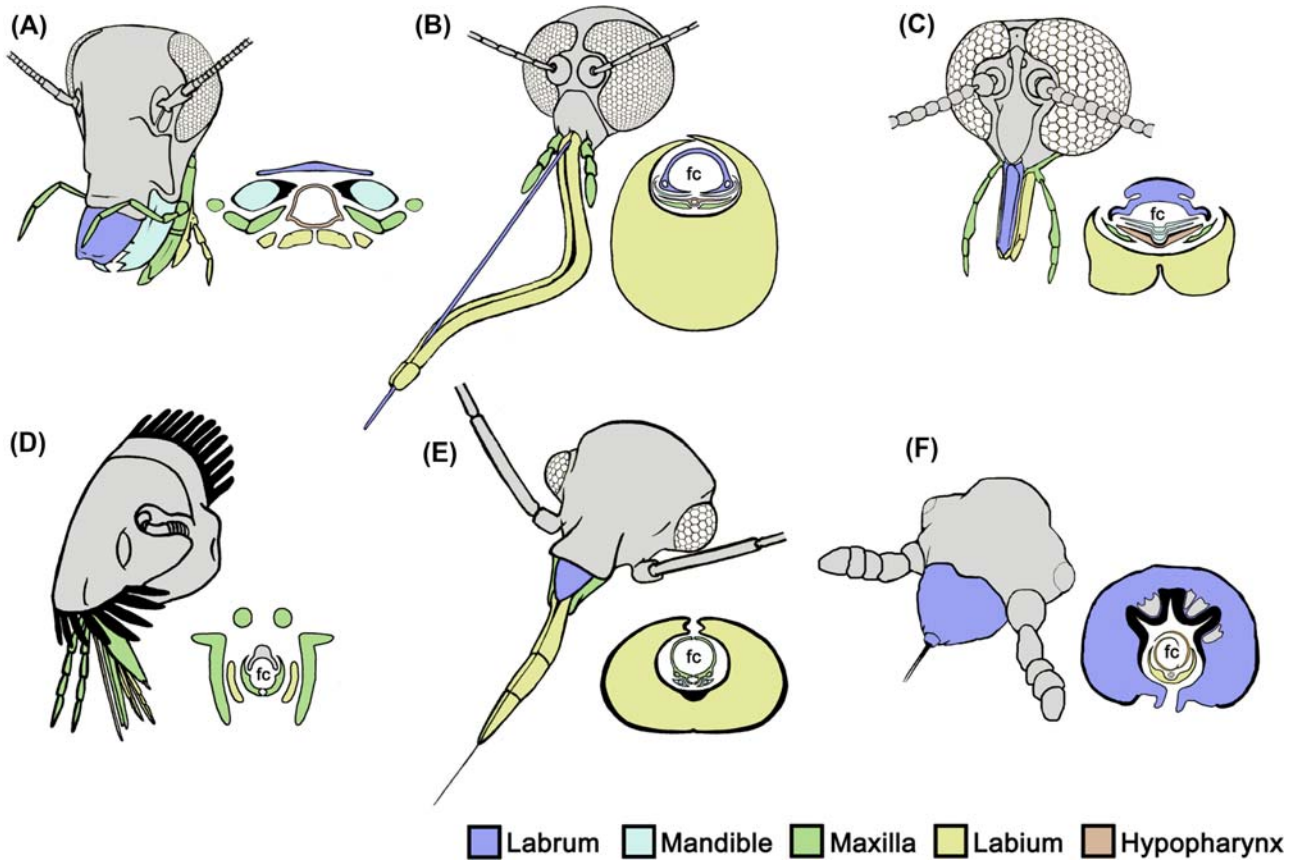


FIGURE 2.2 Head and mouthparts of medically important insects, with cross section of mouthparts. (A) Cockroach, *Periplaneta* (Blattidae); (B) mosquito, *Aedes* (Culicidae) with labium reflexed to show styletiform mouthparts; (C) biting midge, *Culicoides* (Ceratopogonidae); (D) cat flea, *Ctenocephalides felis* (Pulicidae); (E) bed bug, *Cimex* (Cimicidae); (F) human body louse, *Pediculus humanus* (Pediculidae). *fc*, food canal. *Original by Nathan D. Burkett-Cadena.*

fused palpcoxae in mites, is greatly enlarged and serves as an attachment organ in ticks. Rearward projecting teeth on the hypostome serve to anchor the tick securely to its host after it has cut a hole through the skin with its chelicerae. The palps of ticks are relatively unmodified and do not penetrate the skin of the host. In other parasitic mites, including members of the families Dermanyssidae and Macronyssidae, the chelicerae may be long, slender, and retractable and are adapted for piercing host skin. The terminal portion of the chelicera may be pincer-like, as in *Ornithonyssus* (Macronyssidae) (Fig. 2.3B), or serrate, lacking the movable element, as in *Dermanyssus* (Dermanyssidae) (Fig. 2.3B inset).

LEGS

In both insects and arachnids, those taxa that live for extended periods of time on their hosts often have highly enlarged legs and/or the legs bear specialized structures to facilitate attachment to the host and movement amidst the host hair or feathers. Structures for grasping are often coupled with stout, heavily sclerotized and generously muscled appendages. This combination of characters allows ectoparasites not only to obtain access to new hosts but also to avoid being displaced or removed by host grooming.

The insect leg typically consists of five segments (Fig. 2.4A). The basal segment is the ***coxa***, followed by the **trochanter**, **femur**, **tibia**, and **tarsus**. The tarsus is divided into subsegments, or **tarsomeres**, providing flexibility. Claws and other structures, when present, are typically borne on the apical tarsomere. In many ectoparasitic insects the typical leg has become modified to facilitate host attachment and dispersal to new hosts. Adult fleas exhibit perhaps the most widely recognized modification of the legs for getting on and off hosts. The hind legs of fleas are particularly modified to enable them to jump remarkable distances to reach a host or to evade removal by host grooming (Fig. 2.4B). Modifications of the flea hind leg include an enlarged, muscular femur (as in other jumping insects) and an elastic protein in the integument called **resilin**. Resilin, an important structural component of the

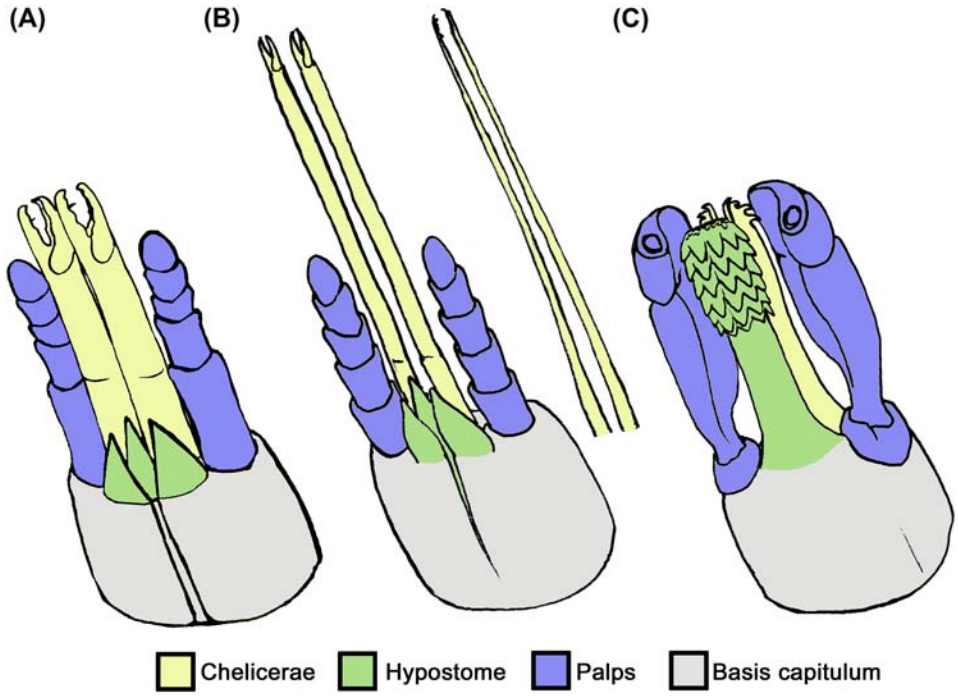


FIGURE 2.3 Gnathosoma of medically important mites, ventral views. (A) *Pneumolaelaps* (Laelapidae); (B) *Ornithonyssus* (Macronyssidae), with inset of chelicerae of *Dermanyssus* (Dermanyssidae); (C) hard tick, *Amblyomma* (Ixodidae). *Original by Nathan D. Burkett-Cadena.*

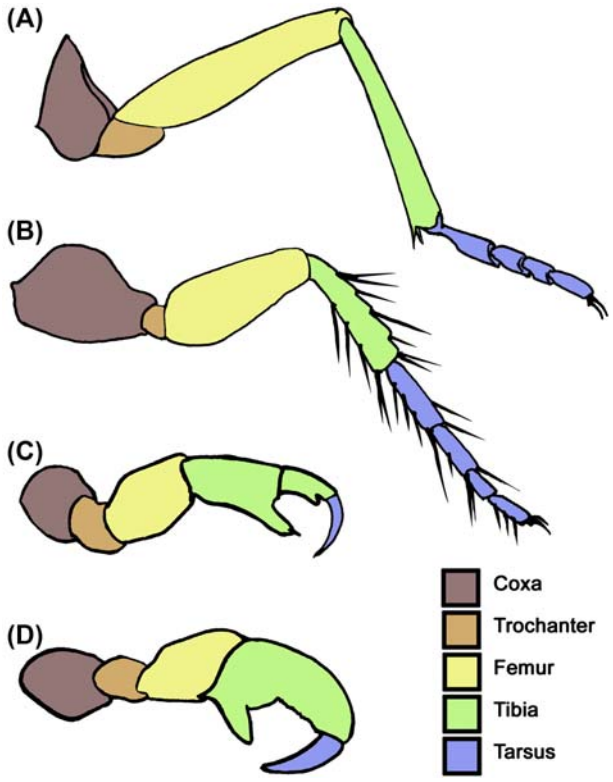


FIGURE 2.4 Legs of medically important insects. (A) Hind leg of blister beetle, *Epicauta* (Meloidae); (B) hind leg of cat flea, *Ctenocephalides felis* (Pulicidae); (C) foreleg of human body louse, *Pediculus humanus* (Pediculidae); (D) hind leg of human pubic louse, *Pthirus pubis* (Pthiridae). *Original by Nathan D. Burkett-Cadena.*

flight mechanism in flying insects, helps to store energy and significantly increase the efficiency of the hind legs in the jumping ability of fleas. Contraction of muscles in the hind legs compresses pads of resilin located at the bases of the hind coxae. A release mechanism causes rapid expansion of the resilin pads, propelling the flea forward and upward during the jump.

Lice are noted for their ability to cling tenaciously to their hosts. The legs of sucking lice are particularly well adapted for grasping host hair. The claws are formed by modifications of the tibia and tarsus, called tibiotarsal claws (Fig. 2.4C and D). The tarsus is generally reduced to form a stout, curved, often sickle-shaped, movable element. This element articulates with a sclerotized projection of the tibia to form an effective grasping structure.

Among the Diptera, hippoboscids (e.g., keds) have legs that are specialized for host attachment. Unlike other blood-feeding dipterans, for which host contact is usually brief, hippoboscids may spend their entire lives on their hosts. The legs are therefore stout and usually spinose, with enlarged tarsal claws. These features enable hippoboscids to hold onto their host and to move about quickly and efficiently amid host pelage.

In mites, each leg typically consists of seven segments (Fig. 2.5A). The basal segment is the coxa, followed by the trochanter, femur, **genu**, tibia, tarsus, and terminal **apotele**. The apotele may bear claws, setae, and/or an empodium, a padlike structure arising between the bases of the claws. In parasitic mites, various modifications of the legs enable

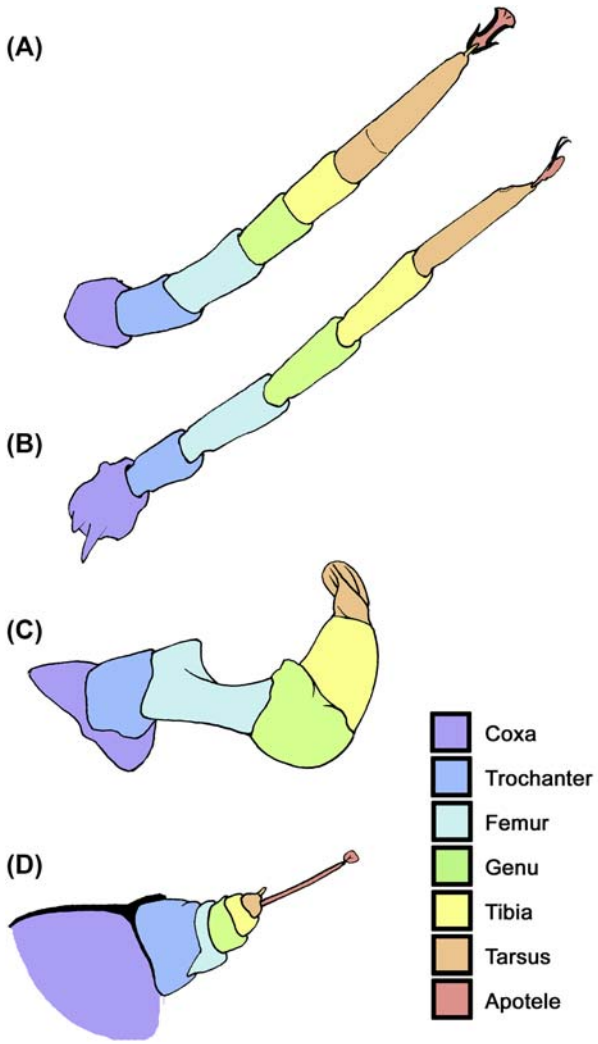


FIGURE 2.5 Legs of medically important mites. (A) Leg I of *Pneumolaelaps* (Laelapidae); (B) leg 1 of hard tick, *Amblyomma* (Ixodidae); (C) leg 3 of fur mite *Trichoecius* (Myocoptidae); (D) leg I of scabies mite, *Sarcoptes* (Sarcoptidae). *Original by Nathan D. Burkett-Cadena.*

these arthropods to locate and attach to their hosts. In ticks, for example, the forelegs have enlarged claws (Fig. 2.5B) that enable them to quickly grasp passing hosts and facilitate holding onto host skin or pelage during feeding and mating. Certain feather mites (e.g., Analgidae) have sucker-like empodia and large spurs on their legs for securing themselves to their avian hosts. Many of the mites that parasitize snakes (e.g., Ixodorhynchidae and Ophionyssidae) also have sucker-like empodia, which facilitate movement on their host and holding onto the smooth surfaces of the body scales. In many other parasitic mites, hind legs, rather than forelegs, are modified for host attachment. Mites in the family Mycoptidae, for example, have legs 3 and 4 enlarged with opposable digits for clasping the fur of their mammalian hosts (Fig. 2.5C). In scabies mites (Sarcoptidae), all four pairs of legs are reduced and have elongate apoteles with terminal suckers (Fig. 2.5D). These structures allow the mites to move about quickly on the surface of the skin and to hold tightly to the epidermis.

SENSORY STRUCTURES

Sensory structures play an integral role in host location and recognition and thus have become highly specialized in many parasitic arthropods. Various sensory structures of parasitic arthropods function to detect motion, vibrations, temperature, moisture, carbon dioxide, and a plethora of chemical substances produced by potential hosts. In combination with each other, these environmental and host-associated cues are often specific for a single host species or group of closely related host animals.

In insects, the antennae and eyes are the primary sensory organs. The antennae of blood-feeding insects, particularly hematophagous dipterans, have receptors that detect chemicals emanating from the skin and present in the exhaled breath of potential hosts. Host substances known to attract mosquitoes include carbon dioxide, lactic acid, octenol, estrogen, fatty acids, and amino acids. In mosquitoes, sensory receptors in the basal segment of the antenna are highly developed to form the **Johnston's organ,** which is specialized for detecting airborne vibrations. Host-seeking female mosquitoes may cue in on vibrations produced by host movements and even vocalizations by hosts such as birds and frogs. Fleas exhibit an interesting modification of the antennae, in which the antenna is short, flattened, and fits into a protective groove on the side of the head (Fig. 2.2D). This allows the antennae to be retracted so as not to become damaged or impede movement as the flea maneuvers amid host hair or feathers.

In fleas and lice, the eyes are generally reduced in size (Fig. 2.2D and F) or may be altogether absent. In some cases, such modifications of the eyes help to prevent damage to the sense organs, while in other cases the reduction of eyes reflects the relative unimportance of vision in the life of the parasite. In many other insects, such as mosquitoes (Fig. 2.2B), biting midges (Fig. 2.2C), and horse flies, the eyes are greatly enlarged, reflecting the more significant role that light perception and vision play in locating, or orienting toward, potential host animals.

Sensory structures are often particularly numerous or well developed on the mouthparts of parasitic arthropods. In many solenophages, for example, these receptors are concentrated near the tip of the proboscis or rostrum (e.g., mosquitoes and bed bugs, respectively) and are used to detect the precise location of capillaries beneath the surface of the skin. In biting midges, sensory receptors for detecting environmental and host cues are concentrated in a specialized pit, in the form of a sensory organ on the enlarged third segment of the maxillary palp (Fig. 2.2C).

In fleas, the dorsal portions of the terminal abdominal segments are modified as a sensory organ, called the **sensilium**. The associated sensory structures are specialized for detecting host-associated cues such as vibrations and temperature gradients.

In mites, chemical and tactile cues are perceived by sensory structures on the pedipalps, legs, and various other parts of the body. Specialized sensory setae with an associated socket-like base, called **trichobothria**, are common in many groups of mites and other arachnids for detecting airborne and substrate vibrations and other tactile cues. In certain groups of mites, the first pair of legs may be unusually long and slender, with numerous receptors, serving as a sensory organ, much like the antennae of insects. In ticks, a complex sensory structure, called **Haller's organ**, is located on the dorsal aspect of the tarsus of the first pair of legs and functions in detection of temperature, air movements, host odors, and other host and environmental cues.

In summary, a number of advantageous morphological modifications are evident in ectoparasitic and blood-feeding arthropods. Recurring adaptations include modifications of the body shape, feeding apparatus, locomotory appendages, and sensory structures. Each modification has allowed parasitic arthropods to more efficiently exploit their vertebrate hosts. A number of excellent works have been produced, such as those by B. Jobling (1987) and R. E. Snodgrass, that examine in detail the morphological adaptations of parasitic arthropods. For further information on general insect and arachnid morphology, see Snodgrass (1993), Chapman (1998), and Krantz and Walter (2009).

REFERENCES AND FURTHER READING

Chapman, R. F. (1998). *The insects: Structure and function* (4th ed.). Cambridge: Cambridge University Press.

Harbach, R. E., & Knight, K. L. (1980). *Taxonomist's glossary of mosquito anatomy*. Biological Research Institute of America, Plexus Publishing, Inc.

Hopkins, G. H., & Rothschild, M. (1953). *An illustrated catalogue of the Rothschild collection of fleas (Siphonaptera) in the British Museum (Natural History)* (Vol. I). London: Tungidae and Pulicidae, British Museum (Natural History).

Jobling, B. (1987). *Anatomical drawings of biting flies*. London: British Museum (Natural History).

Krantz, G. W., & Walter, D. E. (Eds.). (2009). *A manual of acarology* (3rd ed.). Texas Tech University Press.

Labrzycka, A. (2006). A perfect clasp - adaptation of mites to parasitize mammalian fur. *Biological Letters, 43*, 109−118.

Lozano-Fernandez, J., Carton, R., Tanner, A. R., Puttick, M. N., Blaxter, M., Vinther, J., et al. (2016). A molecular palaeobiological exploration of arthropod terrestrialization. *Philosophical Transactions of the Royal Society B, 371*(1699), 20150133.

Marshall, A. G. (1981). *The ecology of ectoparasitic insects*. London: Academic Press.

Regier, J. C., Shultz, J. W., Zwick, A., Hussey, A., Ball, B., Wetzer, R., et al. (2010). Arthropod relationships revealed by phylogenomic analysis of nuclear protein-coding sequences. *Nature, 463*, 1079−1083.

Sasaki, G., Ishiwata, K., Machida, R., Miyata, T., & Su, Z. H. (2013). Molecular phylogenetic analyses support the monophyly of Hexapoda and suggest the paraphyly of Entognatha. *BMC Evolutionary Biology, 13*, 236.

Snodgrass, R. E. (1943). The feeding apparatus of the biting and disease-carrying flies: A wartime contribution to medical entomology. *Smithsonian Miscellaneous Collections, 104*, 1−51.

Snodgrass, R. E. (1944). The feeding apparatus of the biting and sucking insects affecting man and animals. *Smithsonian Miscellaneous Collections, 104*, 1−113.

Snodgrass, R. E. (1946). The skeletal anatomy of fleas (Siphonaptera). *Smithsonian Miscellaneous Collections, 104*, 1−110.

Snodgrass, R. E. (1993). *Principles of insect morphology*. Ithaca, NY: Cornell University Press.

Stojanovich, C. J. (1945). The head and mouthparts of the sucking lice (Insecta: Anoplura). *Microentomology, 10*, 1−46.

Chapter 3

Arthropod Toxins and Venoms

Justin O. Schmidt

Southwestern Biological Institute, Tucson, AZ, United States; Department of Entomology, University of Arizona, Tucson, AZ, United States

Chemicals play many important roles in the lives of insects, arachnids, and other arthropods. Chemicals serve a number of functions, including communication, both within and between or among species; sex attraction and mating; aggregation behavior; finding and capturing prey or other food; defending themselves, or their offspring, nests, and hives, against predators and other natural enemies; alerting members of their species to perceived threats by alarm pheromones; marking trails with trail pheromones; and a variety of other behaviors, particularly among social insects.

From a medical-veterinary perspective, the encounters between arthropods and humans or domestic animals typically involve chemicals used for offense (e.g., subduing and killing prey) and defense (e.g., biting or stinging behavior when disturbed or threatened). The chemicals involved are extremely diverse in their structures and modes of action, causing a wide range of adverse effects, depending on the species involved. The term **toxin** is applied to any specific chemical compound or molecule that causes harm to an organism on contact or when ingested. The term **venom** refers to a toxin or, more typically, a mixture of one or more toxins and other chemicals, involving specialized morphological structures for injecting, spraying, or otherwise directing them to a target. The injection of venom by such specialized structures is called **envenomation**.

Examples of such structures are the modified female reproductive organs and associated muscles and glands forming the sting apparatus in bees, wasps, and ants (Figs. 3.1 and 3.2; see also Figs. 22.1, 22.3, and 22.4); the specialized setae and spines of urticating caterpillars that facilitate toxins penetrating the skin on contact (see Figs. 21.4 and 21.5); the terminal abdominal segment forming the sting in scorpions (see Fig. 23.7); and the modified chelicerae and venom glands of spiders (see Fig. 25.2).

Table 3.1 provides an overview of the more important insects and arachnids of medical and veterinary importance that produce toxins and venoms. They include the insect orders Hymenoptera, Coleoptera, and Lepidoptera and the arachnid orders Scorpiones and Araneae. Also included in the table is information on the geographic occurrence of the listed taxa, the toxic compounds they produce, and the respective effects on vertebrates.

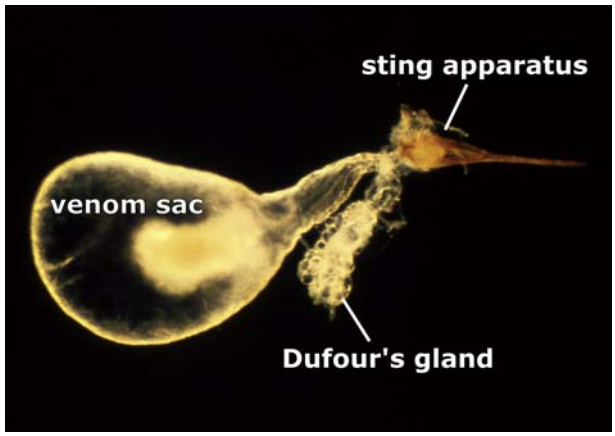


FIGURE 3.1 Venom sac and sting apparatus of the red imported fire ant (Formicidae, *Solenopsis invicta*), dissected from a worker ant. Venom is injected at the sting site via a channel formed by the pair of barbed lancets, at right, which penetrate the skin when an ant stings; the smaller, accessory sac is Dufour's gland, which produces a trail pheromone. *Photograph by Justin O. Schmidt.*

Medical and Veterinary Entomology. https://doi.org/10.1016/B978-0-12-814043-7.00003-0

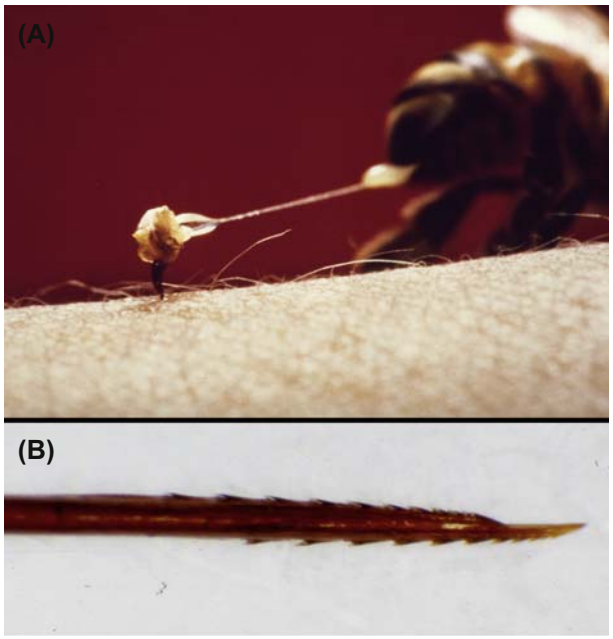


FIGURE 3.2 A honey bee (Apidae, *Apis mellifera*), worker. (A) The bee has just stung a victim, leaving the barbed lancets of the sting apparatus embedded in the skin, after having been torn from the bee's abdomen. (B) Close-up of the pair of barbed lancets, which slide along one another as they penetrate the skin. *Photographs by Justin O. Schmidt.*

NATURE OF TOXINS AND VENOMS AND THEIR EFFECTS

The chemical structures of toxins and venoms vary from small, simple acids (Fig. 3.3) and intermediate-sized compounds (Figs. 3.4 and 3.5) to large, complex molecules (Figs. 3.6–3.8) involving up to 35 or more amino acids. The following examples illustrate the diversity and complexity of chemical structures among toxins and venoms found in insects and arachnids: formic acid of formicine ants and some beetles (Fig. 3.3); acetic acid sprayed as a defensive secretion by the whip-scorpion, or vinegaroon, *Mastigoproctus giganteus* (Fig. 3.3); cantharidin of certain blister beetles, such as *Epicauta* spp. (Fig. 3.4); pederin of certain rove beetles, such as *Paederus* spp. (Fig. 3.4); solenopsin in the venom of fire ants (Fig. 3.5) and poneratoxin of the bullet ant *Paraponera clavata* (Fig. 3.6); melittin of honey bees, such as *Apis mellifera* (Fig. 3.6); scyllatoxin and agitoxin in venom of the deathstalker scorpion, *Leirurus quinquestriatus* (Fig. 3.7); and atracotoxin in venom of the Sydney funnelweb spider, *Atrax robustus* (Fig. 3.8).

Toxins are typically fat soluble and water repellent (hydrophobic), which aid toxins in penetrating the integument (e.g., waxy outer layer of the cuticle in arthropods and vertebrate skin) on contact. Venoms, on the other hand, tend to be more water soluble. They often consist of complex mixtures with other chemicals that facilitate the spread and effectiveness of the toxic components once they penetrate the integument to enter the more aqueous environment within.

The more potent components of arthropod venoms are typically proteins in the form of peptides and polypeptides, consisting of one or more chains of amino-acid residues (Figs. 3.6–3.8). A dazzling variety of venom proteins are known: enzymes such as phospholipases, hyaluronidases, phosphatases, esterases, and proteases, in addition to specific enzymes acting as neurotoxins. These proteins are responsible for much of the damage, and sometimes lethality, that can occur. Venom peptides include hemolysins, such as melittin (Fig. 3.6), that destroy blood cells; kinins, which cause pain and cardiovascular effects; and various neurotoxins. Venoms also can contain cocktails of biogenic amines, including histamine, serotonin, acetylcholine, and epinephrine. These components can cause swelling and other reactions, and sometimes the pain, at sting sites. Neurotoxins are especially important in the venom of dangerous scorpions and spiders. Other toxic compounds are cardiotoxins of milkweed butterflies and certain beetles and quinones and hydroquinones of millipedes.

A number of toxins cause immediate, and often intense, pain. This serves as a powerful deterrent to predators and other perceived threats, including humans and both domestic and wild animals. Although pain is commonly associated with localized swelling or erythema at the bite site, it also can be inflicted without causing other apparent harm. An extreme example of pain acting alone is the tarantula hawk *Pepsis chrysothemis*, a spider wasp (Pompilidae) that stings and paralyzes tarantulas as food for its developing offspring (Fig. 3.9). Its sting is excruciatingly painful, yet 2,000 stings would be required to kill a 110-lb (50-kg) person (Schmidt, 2004).

Although fatalities represent only a small percentage of cases, people and domestic animals can succumb to lethal

TABLE 3.1 Important Toxic and Venomous Insects and Arachnids

Common Names	Scientific Names (Species and Family)	Major Geographic Distribution	Toxic Components and Their Effects on Vertebrates	Toxin or Venom
INSECTS				
Hymenoptera				
Honey bees	*Apis mellifera*; other *Apis* spp. (Apidae)	Worldwide	**Melittin**: inhibits protein kinases and ion transport across cell membranes; increases permeability of cell membranes and cell lysis; cardiotoxic **Phospholipase A_2**: lung congestion and allergic reactions	Venom
Yellowjackets and hornets	*Vespula, Dolichovespula, Vespa* spp. (Vespidae)	Northern Hemisphere (excluding Sub-Saharan Africa)	**Kinins**: hemolysis and pain **Phospholipase A_1**: allergic reactions. **Mastoparans** destroy mast cells	Venom
Paper wasps	*Polistes* spp. (Vespidae)	Worldwide	Similar to yellowjackets and hornets	Venom
Polybine and Old World paper wasps	*Polybia occidentalis, Ropalidia marginata,* and others (Vespidae)	Mostly tropics, worldwide	Similar to yellowjackets and hornets	Venom
Fire ants	*Solenopsis invicta* and other *Solenopsis* spp. (Formicidae)	New World Tropics and Subtropics; invasive to many other areas	Piperidine alkaloids (e.g., **Solenopsin**): inhibit angiogenesis via kinase signaling pathway; cytotoxicity, hemolysis, necrosis, and pain	Venom
Bulldog ants	*Myrmecia pilosula, M. gulosa,* and other *Myrmecia* spp. (Formicidae)	Australia, Tasmania	**Pilosulin**: enzyme that can cause severe allergic reactions, including anaphylaxis	Venom
Bullet ant	*Paraponera clavata* (Formicidae)	New World Tropics; moist regions	**Poneratoxin**: neurotoxic peptide; acts on voltage-gated Na^+ channels in skeletal muscle fibers, causing paralysis; extreme pain	Venom
Harvester ants	*Pogonomyrmex maricopa*; many other *Pogonomyrmex* spp. (Formicidae)	New World; especially warm, arid regions	Phospholipases and other enzymes (e.g., **Barbatolysin**); extreme pain and potential anaphylaxis	Venom
Coleoptera				
Blister beetles	*Epicauta* spp. (Meloidae)	Worldwide	**Cantharidin**: blistering agent; monoterpene that causes release of proteases and other enzymes that destroy adhesion of cells; severe damage to gastrointestinal and urinary tracts if ingested (e.g., horses)	Toxin
Rove beetles	*Paederus* spp. (Staphylinidae)	Worldwide; warm, moist regions	**Pederin**: an amide produced by *Pseudomonas* endosymbionts; prevents cell division; inhibits protein and DNA synthesis, causing vesicating dermatitis	Toxin
Lepidoptera				
Lonomia caterpillars	*Lonomia achelous* and *L. obliqua* (Saturniidae)	South America	Proteolytic enzymes: break down fibrinogen in blood causing hemorrhaging; can lead to heart and kidney failure	Venom

Continued

TABLE 3.1 Important Toxic and Venomous Insects and Arachnids—cont'd

Common Names	Scientific Names (Species and Family)	Major Geographic Distribution	Toxic Components and Their Effects on Vertebrates	Toxin or Venom
Processionary caterpillars	*Thaumetopoea processionea*, *T. wilkinsoni*, and other *Thaumetopea* spp. (Thaumetopoeidae)	Europe, Asia, Africa, and Australian region	**Thaumetopoein**: an urticating toxin; causes dermatitis, rash, conjunctivitis; if inhaled, pharyngitis, respiratory distress, and asthma	Venom
Puss moth caterpillars	*Megalopyge opercularis* and other *Megalopyge* spp. (Megalopygidae)	North America and South America	Protein toxins: cause local and intense radiating pain; edema, welts, pruritus, erythema, and rash	Venom
ARACHNIDS				
Scorpiones				
Brazilian yellow scorpion	*Tityus serrulatus* (Buthidae)	Central America and South America	**Tityus serrulatus toxin 1** (Ts1): peptide neurotoxin acting on voltage-gated sodium channels; causes massive release of neurotransmitters affecting nervous and muscular systems throughout body	Venom
Bark scorpion	*Centruroides sculpturatus* (Buthidae)	Southwestern US and northwestern Mexico	Centruroides toxins: peptide neurotoxins acting on Na^+ channels	Venom
Deathstalker scorpion	*Leiurus quinquestriatus* (Buthidae)	North Africa and Middle East	**Scyllatoxin**: neurotoxin that blocks Ca^{2+}-activated K^+ channels; can severely affect pulmonary and cardiac systems **Agitoxin**: blocks K^+ channels	Venom
Fattail scorpion	*Androctonus australis* (Buthidae)	North Africa and Middle East to India	Androctonus toxins: peptide neurotoxins acting on Na^+ channels	Venom
Araneae				
Black widow spiders	*Latrodectus mactans* and other *Latrodectus* spp. (Theridiidae)	Worldwide	**α-Latrotoxin**: neurotoxin that acts on pre-synaptic nerve terminals, causing massive release and depletion of neurotransmitters; local pain, muscle cramps; paralysis of diaphragm and potential asphyxiation	Venom
Brazilian wandering spider	*Phoneutria nigriventer* (Ctenidae)	South America	Phoneutria toxins: peptide neurotoxins acting on Na^+ channels	Venom
Sydney funnelweb spider	*Atrax robustus* (Hexathelidae)	Australia	**d-Atracotoxin** (robustoxin): neurotoxin that causes repetitive firing and prolonged action potentials; continuous release of acetylcholine; can lead to circulatory and respiratory failure	Venom
Recluse spiders	*Loxosceles reclusa*, *L. laeta*, and other *Loxosceles* spp. (Sicariidae)	Worldwide	**Sphingomyelinase D**: enzyme that breaks down sphingomyelin in plasma membranes, causing cell destruction, inflammation, and necrotic skin lesions	Venom

For the Larger Taxonomic Groups, Only the More Important Species and Representative Taxa are Noted (Ca, Calcium; K, Potassium; Na, Sodium).

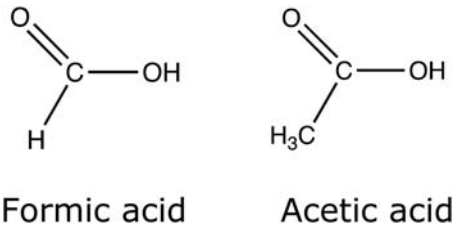
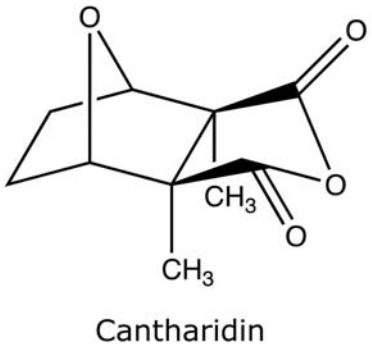


FIGURE 3.3 Two structurally simple toxins found in arthropods: formic acid, typical of some biting ants; and acetic acid, a defensive compound in arachnids called vinegaroons (Thelyphonida; e.g., whip-scorpion, *Mastigoproctus giganteus*).



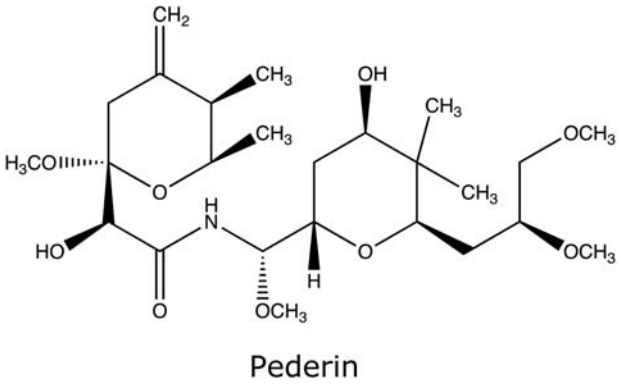


FIGURE 3.4 Toxins that serve as defensive agents in beetles; both can cause dermatitis on contact with skin and injury to the gastrointestinal tract when ingested: cantharidin, in the hemolymph of blister beetles (Meloidae, e.g., *Epicauta* spp.) and pederin, in the hemolymph of certain rove beetles (Staphylinidae, e.g., *Paederus* spp.).

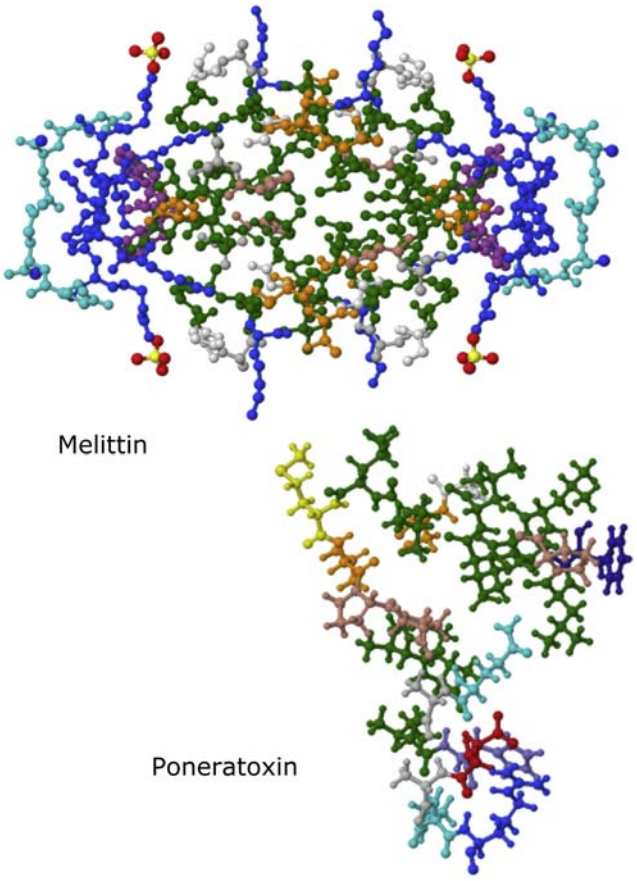


FIGURE 3.6 Two peptide toxins in hymenopteran insects: melittin in honey bees (Apidae, e.g., *Apis mellifera*) with 26 amino acids, and poneratoxin, a 25-residue peptide neurotoxin in bullet ants (Formicidae, e.g., *Paraponera clavata*). *Images from RCSB PDB (www.rcsb.org): melittin, PDB ID 2MLT, Gribskov, M., Wesson, L., Eisenberg, D. (2018) ["To Be Published"]; poneratoxin, PDB ID 1G92, Szolajska, E., Poznanski, J., Ferber, M.L., Michalik, J., Gout, E., Fender, P., Bailly, I., Dublet, B., Chroboczek (2004) Eur. J. Biochem. 271: 2127–2136.*

envenomation by various biting and stinging arthropods such as honey bees (Schmidt, 2018), Asian hornets (Liu et al., 2016), scorpions (Chippaux and Goyffon, 2008), and spiders (Vetter and Isbister, 2008). Livestock, especially horses, can die from ingesting toxic blister beetles (Capinera et al., 1985).

Allergic reactions to stings represent a particular concern to sensitized individuals, with such reactions to insect stings occurring in approximately 1%–4% of human populations. Responses can include dermal rashes and edema at a distance from the sting site; difficulty breathing caused by swelling of the throat and respiratory passages; and a severe drop in blood pressure, followed by faintness

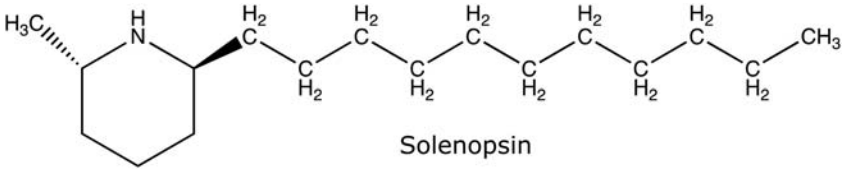


FIGURE 3.5 Solenopsin, a piperidine alkaloid and the primary toxin in venom of fire ants (Formicidae, *Solenopsis* spp.).

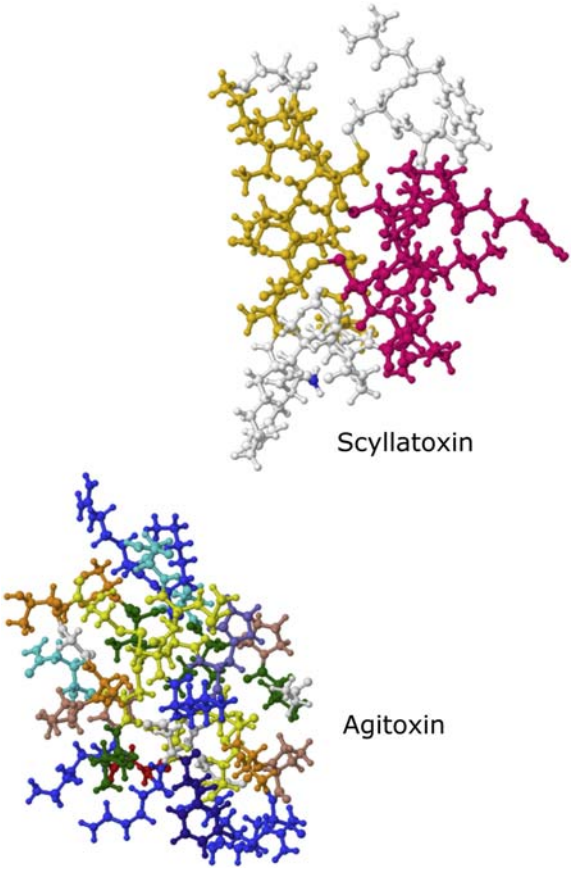


FIGURE 3.7 Two neurotoxins in venom of the deathstalker scorpion *Leiurus quinquestriaus* (Buthidae): scyllatoxin (leiurotoxin I), a 31-residue peptide; and agitoxin, a polypeptide with 38 amino acids, which can severely affect pulmonary and cardiac systems leading to death of sting victims. *Images from RCSB PDB (www.rcsb.org): scyllatoxin, PDB ID 1SCY, Martins, J.C., Van de Ven, F.J., Rorremans, F.A., (1995) J. Mol. Biol. 253: 590–603; agitoxin, PDB ID 1AGT, Krezel, A.M., Kasibhtla, C., Hidalgo, P., MacKinnon, R., Wagner, G. (1995) Protein Sci., 4: 1478–1489.*

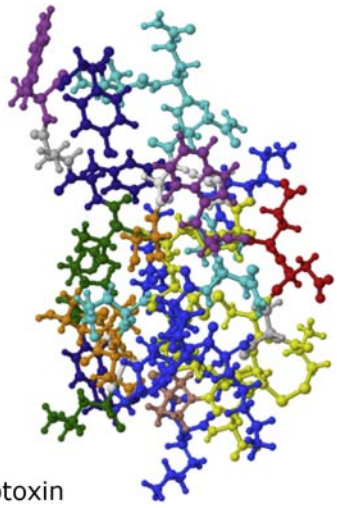


FIGURE 3.8 A potent polypeptide neurotoxin in venom of the Sydney funnelweb spider, *Atrax robustus* (Hexathelidae); δ-atracotoxin (robustoxin) can cause serious circulatory and respiratory failure in human bite cases. *Image from RCSB PDB (www.rcsb.org) PDB ID 1qdp; Pallaghy, P.K., Alewood, D., Alewood, P.F., Norton, R.S. (1997) FEBS Lett. 419: 191–196.*

and loss of consciousness. In extreme cases, anaphylaxis can be fatal, with approximately 60 people per year dying in the United States (Schmidt, 2015). Despite this statistic, the actual per capita incidence is low. Although approximately 7 million people in the United States are allergic to insect stings, only one person per 100,000 allergic cases dies each year.

ARTHROPOD VENOMS AS ANTIMICROBIAL, ANALGESIC, AND OTHER THERAPEUTIC AGENTS

Naturally occurring antimicrobial peptides (AMPs) produced by insects and arachnids have drawn increased interest in recent years as alternatives to conventional antibiotics. Their modes of action, effectiveness, and low resistance rates offer promising activity against bacteria, fungi, viruses, and certain parasites. Melittin from honey bees, for example, has been shown to inhibit bacteria, including the Lyme disease agent *Borrelia burgdorferi*. It also kills yeast (e.g., *Candida albicans*) and suppresses *Mycoplasma* and *Chlamydia* infections. Solenopsin from fire ants has been shown not only to be insecticidal but also to exhibit antibacterial, antifungal, and antiviral activity. Similarly, many antimicrobial peptides have been identified in scorpions and spiders.

In addition to causing pain, peptides in the venoms of many arthropods, particularly scorpions and spiders, can act as analgesics, or painkillers. They typically affect voltage-gated sodium channels of neurons, including primary afferent nerve fibers associated with the pain pathway. Interest has been focused on their potential as therapeutic agents for modulating neural mechanisms involved in chemical, mechanical, and thermal pain, in addition to chronic pain. Venom of the Chinese scorpion (*Mesobuthus martensii*) has been used since ancient times in traditional Chinese medicine for the treatment of pain. Among the insects, peptides in the venom of several hymenopteran families are currently under study as potential analgesics, including some wasps (Sphecidae), tarantula hawks (Pompilidae), and velvet ants (Mutillidae).

Venom peptides have also garnered significant research interest in treating a number of human diseases and other medical conditions. Melittin, from honey bees, has been postulated to have potential for treating epilepsy

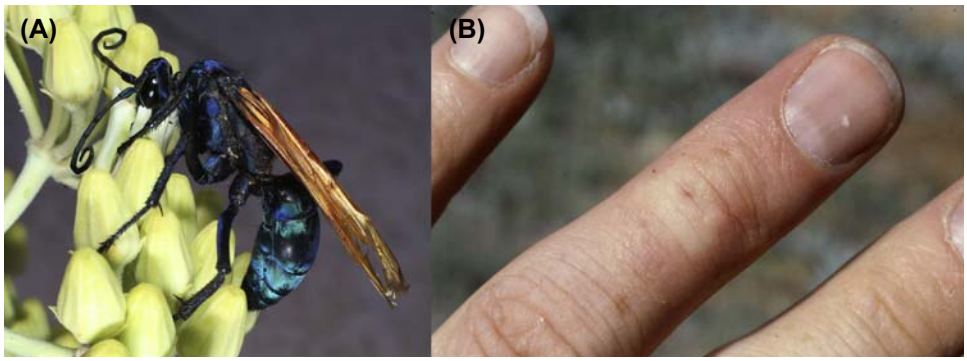


FIGURE 3.9 Tarantula hawk (Pompilidae, *Pepsis chrysothemis*), a wasp that can cause excruciating pain when it stings humans and other animals. (A) Female feeding on milkweed flowers. (B) Sting on human finger; note the relatively unapparent reaction at the sting site, despite extreme pain. *(A) Photograph by Jillian Cowles; (B) Photograph by Justin O. Schmidt.*

and acquired immune deficiency syndrome caused by the Human immunodeficiency virus (HIV). Cantharidin, from blister beetles, has long been used as an aphrodisiac, a topical compound to remove warts, and a treatment for other skin conditions such as molluscum contagiosum. Pederin and its derivatives from staphylinid beetles have been shown to inhibit protein and DNA synthesis and to slow the division of cancer cells. Other antimicrobial peptides in insect venoms have attracted attention as possible alternative treatments of skin, eye, and lung infections.

Scorpion venoms, in particular, are being studied because of the therapeutic potential of specific peptides as anticancer agents. A growing body of literature documents the progress being made in medical research involving breast, prostate, gastric, colorectal, lung, oral, and other cancers. In addition, components of scorpion venoms are drawing attention as possible future agents for treating cell-mediated autoimmune diseases, including multiple sclerosis.

REFERENCES AND FURTHER READING

General References

Beard, R. L. (1960). Insect toxins and venoms. *Annual Review of Entomology, 8*, 1−18.

Bettini, S. (Ed.). (1978). *Arthropod venoms*. Berlin: Springer-Verlag, 977 pp.

Blum, M. S. (1981). *Chemical defenses of arthropods*. New York: Academic Press.

Bücherl, W., & Buckley, E. E. (1971). Venomous animals and their venoms. In *Venomous invertebrates* (Vol. 3). Academic Press, 537 pp.

Capinera, J. L., Gardner, D., & Stermitz, F. R. (1985). Cantharidin levels in blister beetles (Coleoptera: Meloidae) associated with alfalfa in Colorado. *Journal of Economic Entomology, 78*, 763−764.

Chippaux, J.-P., & Goyffon, M. (2008). Epidemiology of scorpionism: A global appraisal. *Acta Tropica, 107*, 71−79.

Eisner, T., Eisner, M., & Siegler, M. (2005). *Secret Weapons: Defense of insects, spiders, scorpions, and other many-legged creatures*. Cambridge, MA: Harvard University Press.

Goddard, J. (1994). Direct injury from arthropods. *Laboratory Medicine, 25*, 365−371.

Meier, J., & White, J. (Eds.). (1995). *Handbook of clinical toxicology of animal venoms and poisons*. Boca Raton: CRC Press, 768 pp.

New, J. J., & German, T. C. (2015). Spiders at the cocktail party: An ancestral threat that surmounts inattentional blindness. *Evolution and Human Behavior, 36*, 163−173.

Nichol, J. (1989). *Bites and stings: The world of venomous animals*. New York: Facts on File, 208 pp.

Papp, C. S., & Swan, L. A. (1983). *A guide to biting and stinging insects and other arthropods* (2nd ed.). Entomography Publications, 211 pp.

Roth, L. M., & Eisner, T. (1962). Chemical defenses of arthropods. *Annual Review of Entomology, 7*, 107−136.

Schmidt, J. O. (2016). *The sting of the wild*. Baltimore, MD: Johns Hopkins University Press.

Schmidt, J. O. (1982). Biochemistry of insect venoms. *Annual Review of Entomology, 27*, 339−368.

Schmidt, J. O. (2015). Allergy to venomous insects. In J. M. Graham (Ed.), *The hive and the honey bee* (pp. 907−952). Hamilton, Illinois: Dadant & Sons.

Tu, A. T. (Ed.). (1984), *Insect poisons, allergens, and other invertebrate venoms: Vol. 2. Handbook of natural toxins*. Marcel Dekker, 732 pp.

Hymenoptera Toxins

Arbiser, J. L., Kau, T., Konar, M., Narra, K., Ramchandran, R., Summers, S. A., et al. (2007). Solenopsin, the alkaloid component of the fire ant (*Solenopsis invicta*), is a naturally occurring inhibitor of phosphatidylinositol-3-kinase signaling and angiogenesis. *Blood, 109*, 560−565.

Camazine, S. (1988). Hymenopteran stings: Reactions, mechanisms and medical treatment. *Bulletin of the Entomological Society of America, 34*, 17−21.

Howell, G., Butler, J., Deshazo, R. D., Farley, J. M., Liu, H. L., Nanayakkara, N. P., et al. (2005). Cardiodepressant and neurologic actions of *Solenopsis invicta* (imported fire ant) venom alkaloids. *Annals of Allergy, Asthma, & Immunology, 94*, 380−386.

Johnson, S. R., Rikli, H. G., Schmidt, J. O., & Evans, S. (2016). A reexamination of poneratoxin from the venom of the bullet ant *Paraponera clavata*. *Peptides, 98*, 51−62.

Klotz, J. H., Deshazo, R. D., Pinnas, J. L., Frishman, A. M., Schmidt, J. O., Suiter, D. R., et al. (2005). Adverse reactions to ants other than imported fire ants. *Annals of Allergy, Asthma, & Immunology, 95*, 418–425.

Liu, Z., X-D, L., et al. (2016). Acute interstitial nephritis, toxic hepatitis and toxic myocarditis following multiple Asian giant hornet stings in Shaanxi Province, China. *Environmental Health and Preventive Medicine*. https://doi.org/10.1007/s12199-016-0516-4.

MacConnell, J. G., Blum, M. S., & Fales, H. M. (1970). Alkaloids and fire ant venom: Identification and synthesis. *Science, 168*, 840–841.

Meyer, M. L. (1996). Chapter 23, Most toxic insect venom. In *Book of insect records*. Gainesville, FL: Department of Entomology & Nematology, University of Florida.

Schmidt, J. O. (1990). Hymenopteran venoms: Striving toward the ultimate defense against vertebrates. In D. L. Evans, & J. O. Schmidt (Eds.), *Insect defense: Adaptations and strategies of prey and predators* (pp. 387–419). Albany: SUNY Press.

Schmidt, J. O. (2004). Venom and the good life in tarantula hawks (Hymenoptera: Pompilidae): How to eat, not be eaten, and live long. *Journal of the Kansas Entomological Society, 77*, 402–413.

Schmidt, J. O. (2018). Clinical consequences of toxic envenomations by Hymenoptera. *Toxicon, 150*, 96–104. https://doi.org/10.1016/j.toxicon.2018.05.013.

Schmidt, J. O., & Blum, M. S. (1978). A harvester ant venom: Chemistry and pharmacology. *Science, 200*, 1064–1066.

Schmidt, P. J., Sherbrooke, W. C., & Schmidt, J. O. (1989). The detoxification of ant (*Pogonomyrmex*) venom by a blood factor in horned lizards (*Phrynosoma*). *Copeia, 1989*, 603–607.

Szolajska, E., Poznanski, J., Ferber, M. L., Michalik, J., Gout, E., Fender, P., et al. (2004). Poneratoxin, a neurotoxin from ant venom. *European Journal of Biochemistry, 271*, 2127–2136.

Yang, S., & Carrasquer, G. (1997). Effect of melittin on ion transport across cell membranes. *Zhonggua Yao Li Xue Bao, 18*, 3–5.

Lepidoptera Toxins

Arocha-Piñango, C. L., Marval, E., & Guerrero, B. (2000). *Lonomia* genus caterpillar toxins: Biochemical aspects. *Biochimie, 82*, 937–942l.

Avilán, L., Guerrero, B., Alvarez, E., & Rodriguez-Acosta, A. (2010). Description of envenomation by the "gusano-pollo" caterpillar (*Megalopyge opercularis*) in Venezuela. *Investigacion Clinica, 51*, 127–132.

Eagleman, D. M. (2008). Envenomation by the asp caterpillar (*Megalopyge opercularis*). *Clinical Toxicology, 46*, 201–205.

Kalender, Y., Kalender, S., Uzunhisarcikli, M., Ogutcu, A., & Açikgoz. (2004). Effects of *Thaumetopoea pityocampa* (Lepidoptera: Thaumetopoeidae) larvae on the degranulation of dermal mast cells in mice; an electron microscopic study. *Folia Biologica (Kraków), 53*, 13–17.

Lamy, M., Pastureaud, M. H., Novak, F., Ducombs, G., Vincendeau, P., Maleville, J., et al. (1986). Thaumetopoein: An urticating protein from the hairs and integument of the pine processionary caterpillar (*Thaumetopoea pityocampa* Schiff., Lepidoptera, Thaumetopoeaidae). *Toxicon, 24*, 347–356.

Moneo, I., Vega, J. M., Caballero, M. L., Vega, J., & Alday, E. (2003). Isolation and characterization of Thap 1, a major allergen from the pine processionary caterpillar *Thaumetopoea pityocampa*. *Allergy, 58*, 34–37.

Pinto, A., Berger, M., Reck, J., Jr., Terra, R., & Guimaraes, J. (2010). *Lonomia obliqua* venom: In vivo effects and molecular aspects associated with the hemorrhagic syndrome. *Toxicon, 56*, 1103–1112.

Stipetic, M. E., Stipetic, M., Rosen, P. B., & Borys, D. J. (1999). A retrospective analysis of 96 "asp" (*Megalopyge opercularis*) envenomation in central Texas during 1996. *Journal of Toxicology - Clinical Toxicology, 37*, 457–462.

Scorpion Toxins

Auguste, P., Hugues, M., Mourre, C., Moinier, D., Tartar, A., & Lazdunski, M. (1992). Scyllatoxin, a blocker of Ca^{2+}-activated K^{+} channels: Structure-function relationships and brain localization of the binding sites. *Biochemistry, 31*, 648–654.

Buisine, E., Wieruszeski, J. M., Lippens, G., Wouters, D., Tartar, A., & Sautiere, P. (1997). Characterization of a new family of toxin-like peptides from the venom of the scorpion *Leiurus quinquestriatus hebraeus*.1H-NMR structure of leiuropeptide II. *The Journal of Peptide Research, 49*, 545–555.

Cologna, C. T., Marcussi, S., Giglio, J. R., Soares, A. M., & Arantes, E. C. (2009). *Tityus serrulatus* scorpion venom and toxins: An overview. *Protein and Peptide Letters, 16*, 920–932.

Curry, S. C., Mance, M. V., Ryan, P. J., Kunkel, D. B., & Northey, W. T. (1983). Envenomation by the scorpion *Centruroides sculpturatus* [review]. *Journal of Toxicology - Clinical Toxicology, 21*, 417–449.

Peigneur, S., Cologna, C. T., Cremonez, C. M., Mille, B. G., Pucca, M. B., Cuypers, E., et al. (2015). A gamut of undiscovered electrophysiological effects produced by *Tityus serrulatus* toxin 1 on Na_v-type isoforms. *Neuropharmacology, 95*, 269–277.

Pucca, M. B., Cerni, F. A., Pinheiro Junior, E. L., Bordon Kde, C., Amorim, F. G., Cordeiro, F. A., et al. (2015). *Tityus serrulatus* venom—A lethal cocktail. *Toxicon, 108*, 272–284.

Valdez-Cruz, N. A., Davila, S., Licia, A., Corona, M., Zamudio, F. Z., Garcia-Valdes, J., et al. (2004). Biochemical, genetic and physiological characterization of venom components from two species of scorpions: *Centruroides exilicauda* Wood and *Centruroides sculpturatus* Ewing. *Biochimie, 86*, 387–396.

Spider Toxins

Grishin, E. V. (1998). Black widow spider toxins: The present and the future. *Toxicon, 36*, 1693–1701.

Murakami, M. T., Gabdoulkhakov, A., Fernandes-Pedrosa, M. F., Tambourgi, D. V., & Anni, R. K. (2005). Structural basis for metal ion coordination and the catalytic mechanism of sphingomyelinase D. *Journal of Biological Chemistry, 280*, 13658–13664.

Nicholson, G. M., & Graudins, A. (2002). Spiders of medical importance in the Asia-Pacific: Atracotoxin, latrotoxin and related spider neurotoxins. *Clinical and Experimental Pharmacology and Physiology, 29*, 785–794.

Südhof, T. C. (2001). Alpha-Latrotoxin and its receptors: Neurexins and CIRL/latrophilins. *Annual Review of Neuroscience, 24*, 933–962.

Szeto, T. H., Birinyi-Strachan, L. C., Smith, R., Connor, M., Christi, M. J., King, G. F., et al. (2000). Isolation and pharmacological characterization of δ-atracotoxin-Hv1b, a vertebrate-selective sodium channel toxin. *Federation of European Biochemical Societies Letters, 470*, 293–299.

Vetter, R. S., & Isbister, G. K. (2008). Medical aspects of spider bites. *Annual Review of Entomology, 53*, 409–429.

Antimicrobial, Analgesic, and Other Therapeutic Activity

Akef, H., Kotb, N., Abo-Elmatty, D., & Salem, S. (2017). Anti-proliferative effects of *Androctonus amoreuxi* scorpion and *Cerastes cerastes* snake venoms on human prostate cancer cells. *Journal of Cancer Prevention, 22*, 40–46.

Al-Asmari, A. K., Riyasdeen, A., Abbasmanthiri, R., Arshadududin, M., & Al-Harthi, F. A. (2016). Scorpion (*Androctonus bicolor*) venom exhibits cytotoxicity and induces cell cycle arrest and apoptosis in breast and colorectal cancer. *Indian Journal of Pharmacology, 48*, 537–543.

Al-Asmari, A. K., Ullah, Z., Al Balowi, A., & Islam, M. (2017). In vitro determination of the efficacy of scorpion venoms as anti-cancer agents against colorectal cancer cells: A nano-liposomal delivery approach. *International Journal of Nanomedicine, 12*, 559–574.

Chen, H., Zhidan, W., Xia, R., Zhaoxia, W., Quing, J., Qiang, G., et al. (2016). Scorpion venom activates natural killer cells in hepatocellular carcinoma via the NKG2D-MICA pathway. *International Immunopharmacology, 35*, 307–314.

Ding, J., Cjhua, P., & Gopalakrishnakone, P. (2014). Scorpion venoms as a potential source of novel cancer therapeutic compounds. *Experimental Biology and Medicine, 239*, 387–393.

Fratini, F., Ciia, G., Turchi, B., & Felicioli, A. (2017). Insects, arachnids and centipedes venom: A powerful weapon against bacteria. A literature review. *Toxicon, 130*, 91–103.

Giovanni, C., Baglioni, M., Baron Toaldo, M., Cescon, M., Bolondi, L., & Gramantiere, L. (2017). Venom from Cuban Blue Scorpion has effect in hepatocellular carcinoma. *Scientific Reports, 7*.

Gomes, A., et al. (2010). Anticancer potential of animal venoms and toxins. *Indian Journal of Experimental Biology, 48*, 93–103.

Heinen, T. E., & Gorina da Veiga, A. B. (2011). Arthropod venoms and cancer. *Toxicon, 57*, 497–511.

Lewis, R., & Garcia, M. (2003). Therapeutic potential of venom peptides. *Nature Reviews, 2*, 790–802.

Li, W., Li, Y., Zhao, Y., Yuan, J., & Mao, W. (2014). Inhibition effects of scorpion venom extracts (*Buthus matensii* Karsch) on the growth of human breast cancer. *African Journal of Traditional, Complementary and Alternative Medicines, 11*, 105–110.

Lubke, L. L., & Garon, C. F. (1997). The antimicrobial agent melittin exhibits powerful in vitro inhibitory effects on the Lyme disease spirochete. *Clinical Infectious Diseases, 25*(Suppl. 1), S48–S51.

Rates, B. R., Verano-Braga, T., et al. (2011). From the stretcher to the Pharmacy's shelf: Drug leads from medically important Brazilian venomous arachnid species. *Inflammation and Allergy - Drug Targets, 10*, 1–9.

Saez, N. J., Senff, S., Jensen, J. E., Er, S. Y., Herzig, V., Rash, L. D., et al. (2010). Spider venom peptides as therapeutics. *Toxins, 2*, 2851–2871.

Sarfo-Poku, C., Eshun, O., & Lee, K. H. (2016). Medical applications of scorpion venom to breast cancer: A mini-review. *Toxicon, 22*, 109–112.

Shen, B., Cao, Z., Li, W., Sabatier, J. M., & Wu, Y. (2017). Treating autoimmune disorders with venom-derived peptides. *Expert Opinion on Biological Therapy, 17*, 1065–1075.

Tong-ngam, P., Roytrakul, S., & Sritanaudomchai, H. (2015). BmKn-2 scorpion venom peptide for killing colorectal cancer cells by apoptosis. *Asian Pacific Journal of Cancer Prevention, 16*, 2807–2811.

Tonk, M., & Vilcinskas, A. (2017). The medical potential of antimicrobial peptides from insects. *Current Topics in Medicinal Chemistry, 17*, 554–575.

Wang, X., & Wang, G. (2016). Insights into antimicrobial proteins from spiders and scorpions. *Protein and Peptide Letters, 23*, 707–721.

Zhang, X. Y., & Zhang, P. Y. (2016). Scorpion venoms in gastric cancer. *Oncology Letters, 12*, 3683–3686.

This page intentionally left blank

Chapter 4

Epidemiology of Vector-Borne Diseases

Christopher M. Barker and William K. Reisen

Center for Vectorborne Diseases, School of Veterinary Medicine, University of California, Davis, CA, United States

Medical and veterinary entomologists play a pivotal role in understanding the epidemiology of vector-borne diseases and are key contributors to multidisciplinary programs that study, monitor, and control vector-borne parasites. Medical entomology plays a prominent role in public health during large-scale outbreaks and periods of war, famine, or natural disasters that disrupt public health programs, displace populations, and increase exposure to vectors. Throughout history, the large-scale movement of populations (e.g., military personnel) into areas endemic for vector-borne diseases has had devastating effects on both the invaders and the local population, because neither was immune to the new parasites to which they were exposed. The recent globalization of commerce, affordable and accessible rapid transportation, and the relaxation of international travel health regulations have combined to increase the spread of parasites and their vectors into new geographical areas, placing previously unexposed populations of humans and other animals at risk of infection.

Globally, outbreaks of many pathogens have been on the increase, and collectively these are known as **emerging infectious diseases**. Vector-borne parasites account for a large proportion of emerging infectious diseases because anthropogenic (human-induced) changes have provided vectors or parasites opportunities to expand their distribution in time or space, or because the parasites have evolved into more virulent or drug-resistant forms.

Specific methods of investigation vary considerably among the vast array of vector-borne parasites, but basic concepts unify the pattern of investigation necessary to understand the epidemiology of vector-borne disease. Inquiry progresses from discovery of the parasite as the causative agent of a disease, to identifying its mode of amplifying transmission among vectors and vertebrate hosts, to monitoring, forecasting, and control. During the discovery period, standard clinical case definitions are established, enabling the tracking of human or animal disease patterns in time and space, and the causative agent is identified, perhaps indicating that an arthropod may be responsible for transmission. Incrimination of the vector(s) requires a combination of field and laboratory investigation that evaluates vector abundance in time and space, host selection patterns, field infection rates, and vector competence. Short-term studies may rapidly determine modes of transmission, but understanding transmission cycles and mechanisms of persistence during unfavorable periods for the parasites typically requires years of careful ecological investigation and laboratory experimentation. Surveillance and control programs are most effective after maintenance, amplification, and epidemic transmission patterns have been described. In practice, discovery rarely progresses in this orderly fashion, and management of health outcomes (e.g., monitoring and management of cases) often precedes the discovery of the causative agent or its modes of transmission.

This chapter provides an introduction to concepts needed to understand the epidemiology and emergence of vector-borne diseases. Epidemiology developed as a science through the investigation of outbreaks of infectious diseases. As a modern discipline, **epidemiology** (etymology: *epi* = upon, *demos* = people, *logos* = study) deals with the natural history and spread of diseases within human and animal populations. Vector-borne diseases result minimally from a triad that includes an arthropod vector, a vertebrate host, and a parasite. The spread of pathogens by arthropods is especially complex, because in addition to interactions between the vertebrate host and the parasite, an arthropod is required for transmission of the parasite to uninfected hosts. Environmental factors such as temperature and rainfall affect these processes by affecting the rate of parasite maturation within the arthropod host as well as arthropod and vertebrate host abundance in time and space.

In medical entomology, a **vector** is an arthropod responsible for the transmission of parasites (not diseases) among vertebrate hosts. Disease is the response of the host to infestation or infection with a parasite outside or inside

Medical and Veterinary Entomology. https://doi.org/10.1016/B978-0-12-814043-7.00004-2

the host's body, respectively. A **parasite** is any organism, including viruses, bacteria, protozoa, helminths, or arthropods, that is dependent on the host for its survival. Parasites may or may not cause disease. When a parasite injures its host and causes disease, it is referred to as a **disease agent,** or **pathogen**. A vector-borne disease, therefore, is an illness caused by a pathogen that is transmitted by an arthropod. **Facultative parasites** have both free-living and parasitic forms, whereas **obligate parasites** are totally dependent on their host(s) to sustain them. **Ectoparasites** live on or outside the host, whereas **endoparasites** live inside the host. When interacting with their hosts, ectoparasites produce an **infestation** that typically remains topical or peripheral, whereas endoparasites produce an **infection** on invasion of host tissues or cells. The manifestation of disease depends on the host—parasite interaction after infection. A host carrying a parasite is said to be infected, whereas an infected host capable of transmitting a parasite is infectious. A host capable of parasite maintenance, particularly in infectious form, without clinical symptoms is a **carrier**.

A complete understanding of the epidemiology of arthropod-borne disease requires knowledge of the biology of parasites, vectors, and hosts and their interactions in different environments. The degree of contact between vectors and vertebrate hosts ranges from intermittent (e.g., mosquitoes) to intimate and continuous (e.g., sucking lice). In many cases, the host provides the vector with food in the form of blood or other tissues, as well as a habitat in which to live. Blood feeding by the vector brings the vector, vertebrate host, and parasite together in time and space, resulting in the potential transmission of parasites from infectious to susceptible vertebrate hosts via the vector. A vector usually must take at least two blood meals during its lifetime to transmit a parasite—the first to acquire the infection and the second to transmit it. Blood meals provide the arthropod with nutrients necessary for metabolism, metamorphosis, and/or reproduction.

The **gonotrophic cycle,** or reproductive cycle of the arthropod, includes the sequence of questing or searching for a host, blood feeding, blood meal digestion, egg maturation, and oviposition. **Parous** females have completed one or more gonotrophic cycles and have a greater probability of being infected with parasites than do **nulliparous** females that have not reproduced and are feeding for the first time. Unlike parasites that are transmitted directly from host to host, parasites transmitted by arthropods generally have replaced free-living or environmentally resistant stages with those that can multiply and develop within the arthropod and be transmitted during the blood-feeding process.

COMPONENTS OF TRANSMISSION CYCLES

The components of a transmission cycle of an arthropod-borne disease are:

- a parasite that can develop and/or multiply within both vertebrate host and vector tissues.
- a vertebrate host (or hosts) that develops a level of infection with the parasite that is infectious to a vector.
- a vector that acquires the parasite from the infectious vertebrate host and is capable of transmission (Fig. 4.1).

Vector-borne parasites have evolved mechanisms for tolerating high constant body temperatures and evading the complex immune systems of the vertebrate hosts as well as for tolerating variable body temperatures and avoiding the very different defensive mechanisms of the arthropod vectors. Parasites that replicate or reproduce asexually such as viruses and bacteria retain the same life form throughout infections of both vertebrate hosts and vectors, whereas more highly evolved parasites that reproduce sexually such as protozoa and helminths have very different life stages in their vertebrate hosts and vectors. Some asexual parasites such as the causative agent of plague, *Yersinia pestis*, may be able to bypass the vector and be transmitted directly from one vertebrate host to another.

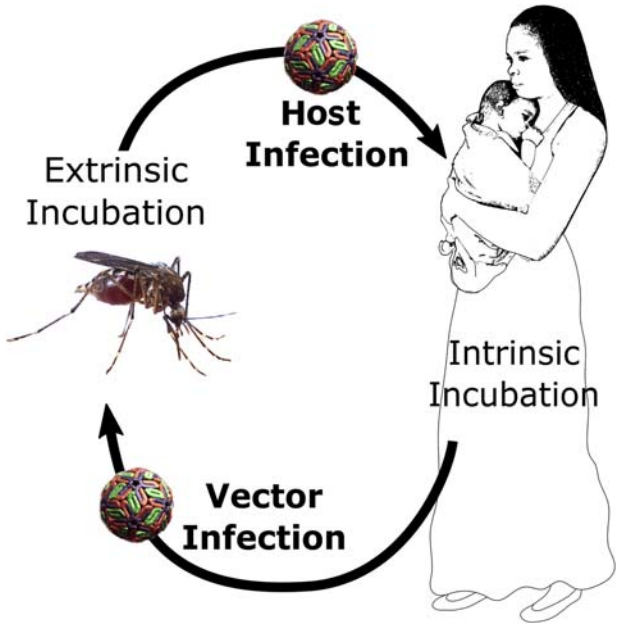


FIGURE 4.1 Components of the transmission cycle of an anthroponosis (e.g., viral disease such as dengue), which does not involve nonhuman vertebrate hosts; with intrinsic incubation of the pathogen in humans and extrinsic incubation in mosquitoes. (Illustration by Jonas King.)

Among sexually reproducing parasites, the host in which gametocyte union occurs is called the definitive host, whereas the host in which asexual reproduction occurs is called the intermediate host. Vertebrates or vectors can serve as either definitive or intermediate host, depending on the life cycle of the parasite. For example, humans are the definitive host for the filarial worm *Wuchereria bancrofti* because adult male and female worms mate within the human lymphatic system, and the mosquito vector *Culex quinquefasciatus* is the intermediate host where larval development of the worms occurs without reproduction. In contrast, humans are the intermediate host of *Plasmodium* protozoans that cause malaria, because only asexual reproduction occurs in the human host; gametocytes produced in the human host unite only in the gut of the definitive host, the *Anopheles* mosquito vector.

Host Immunity

A disease results from the response of the host to infection with the parasite and can occur in either vertebrate or arthropod hosts. Immunity includes all properties of the host that confer resistance to infection and plays an important role in determining host suitability and the extent of disease or illness. Some species or individuals within a population have **natural** (or **innate**) **immunity** and are refractory to infection. Natural immunity does not require that the host have previous contact with the parasite but may be age dependent. For example, humans do not become infected with avian malaria parasites such as *Plasmodium relictum*, even though infective *Culex* mosquito vectors feed frequently on humans. Conversely, mosquitoes do not become infected with measles or polio viruses that infect humans, even though these viruses undoubtedly are ingested by mosquitoes blood feeding on viremic human hosts.

Individuals may acquire immunity after becoming infected with parasites. This **acquired immunity** to the parasite ranges from transient to life-long and may provide partial to complete protection against future infections with the same or related parasites. Partial immunity may allow some parasite development or reproduction within the host and may reduce the severity of disease, whereas complete protection results in clearance of the initial infection and usually prevents immediate reinfection. Acquired immunity may be humoral and result in the rapid formation of antibodies or may be cell-mediated and result in the activation of T cells and macrophages. Antibodies consist of five classes of proteins called **immunoglobulins** that have specific functions in host immunity. **Immunoglobulin G** (**IgG**) is most abundant, comprising more than 75% of the immunoglobulins present in the sera of normal individuals. The IgGs are relatively small proteins, typically reaching high concentration several weeks after infection, and may persist at detectable and protective levels for years. Therefore, parasites such as many arboviruses that induce long-lasting immunity are good candidates for vaccine development. In contrast, **immunoglobulin M** (**IgM**) is a large macroglobulin that appears shortly after infection but decays rapidly relative to IgG. For the laboratory diagnosis of many diseases, serum samples typically are tested during periods of acute illness and again during convalescence 2 to 4 weeks later. A 4-fold increase in parasite-specific IgG antibody concentration in these paired sera provides diagnostic evidence of infection. The presence of elevated concentrations of IgM presumptively implies current or recent infection. **T cells** and **macrophages** are cells that are responsible for the recognition and elimination of parasites. In long-lived vertebrate hosts, acquired immunity may decline over time, eventually making the host susceptible to reinfection.

Clinically, the host response to infection ranges from asymptomatic (i.e., inapparent) to severe. Disease may be acute, occurring during a relatively short interval that begins soon after infection, or chronic, extending over a long period of time after infection. For most parasites, interactions with the vertebrate host are a delicate balance between the need for adequate virulence to amplify parasite concentrations in the host's blood to increase infectiousness to vectors and the need to avoid excessive virulence that could cause adverse effects on the host and result in illness or death that would reduce the host's infectious period. Generally, the susceptibility of the vector to infection dictates the concentration of parasites in the vertebrate host necessary to complete the transmission cycle.

The Vertebrate Host

One or more **primary vertebrate hosts** are essential for the maintenance of parasite transmission, whereas **secondary** or **incidental hosts** are not essential to maintain transmission and may or may not contribute to parasite amplification. **Amplification** refers to the general increase in the number of parasites or prevalence of parasite infection in a given area. An **amplifying host** is permissive to infection, which results in an increase in the number of parasites sufficient to infect susceptible vectors. Amplifying hosts typically do not remain infected for long periods of time and may exhibit disease of varying severity. A **reservoir host** supports parasite development and remains infected and potentially infectious to vectors for long periods but usually does not develop acute disease.

Attributes of a Primary Vertebrate Host Include Accessibility, Susceptibility, and Transmissibility

Accessibility. The vertebrate host must be abundant and fed on frequently by vectors. Adequate numbers of susceptible (i.e., nonimmune) vertebrate hosts must be available to become infected and thereby maintain the parasite population. For persistence of the parasite within the host population, abundance must be above the **critical community size**, which is the minimal number of hosts necessary to ensure an adequate number of susceptible hosts to maintain chains of transmission. Host seasonality, daily activity, and habitat selection determine availability to host-seeking vectors in time and space. For example, the birds that are the primary hosts of eastern equine encephalomyelitis virus (EEEV) generally begin nesting in swamps coincidentally with the emergence of the first spring generation of the mosquito vector, *Culiseta melanura*, thereby bringing EEEV, susceptible avian hosts, and mosquitoes together in time and space. Historically, epidemics of vector-borne diseases have been associated with increases in human accessibility to vectors during wars, natural disasters, environmental changes, or human migrations. Diel activity patterns of primary hosts must coincide with that of vectors, and some parasites themselves exhibit diel periodicity that maximizes potential transmission. For example, larvae (microfilariae) of *W. bancrofti* move to the peripheral circulatory system of the human host during specific hours of the night that coincide with the peak biting period of the mosquito vector, *Culex quinquefasciatus*. Interestingly, this microfilaremic periodicity is altered in Micronesia, where the worms are transmitted by day-biting mosquitoes within the *Aedes scutellaris* group.

Susceptibility. A primary host must be susceptible to infection with a parasite and permissive to its development and reproduction such that the host eventually becomes infectious to vectors. **Dead-end hosts** do not contribute to transmission, either because they do not support a level of infection sufficient to infect vectors or because they become extremely ill and die before the parasite can develop or reproduce to infect additional vectors. Ideal reservoir hosts permit parasites to persist in the peripheral circulatory system (or other suitable tissues) in sufficient numbers for sufficiently long periods to be an effective source for vector infection. Parasites must maintain a delicate balance between their abundance and the duration of infection, which affect their virulence and transmission potential. Strategies vary, and asexual parasites such as viruses and bacteria typically produce intensive infections that produce large numbers of infectious parasites for relatively short periods, during which the host either succumbs to infection or develops protective immunity. In the case of EEEV, for example, 1 mL of blood from an infected bird may contain as many as 10^{10} virus particles during both day and night for a 2- to 5-day period; birds that survive such infections typically develop long-lasting, protective immunity. In contrast, highly evolved parasites produce comparatively few individuals during a longer period. *Wuchereria bancrofti*, for example, maintains comparatively few microfilaria in the blood stream (usually less than 10 microfilariae/ml of blood), which circulate most abundantly in the peripheral blood during periods of the day when the mosquito vectors blood feed. However, because both the worms and the human host are long-lived, transmission is enhanced by repeated exposure of hosts to vector biting rather than by a high parasite load over a period of a few days. Infection with more than 100 microfilariae per female mosquito may prove fatal for the vector; therefore, limiting the number of parasites that infect the vector actually may increase the probability of transmission in this system.

Suitable hosts must either revert to susceptibility to infection or have a relatively high recruitment rate of susceptible hosts into the population through birth or immigration. In the case of malaria, for example, the parasite elicits an immune response that rarely is completely protective, and the host remains parasitemic and susceptible to reinfection. In contrast, encephalitis virus infections of passerine birds typically produce life-long protection, but life expectancy of birds is short and the population replacement rate is high, thereby ensuring a rapid and annual replenishment of susceptible hosts.

The Arthropod Vector

Literally, a **vector** is a "carrier" of a parasite from one host to another. An effective vector generally exhibits characteristics that complement those listed for the vertebrate hosts and include abundance, host fidelity, and vector competence.

Abundance. To be important for transmission of a parasite, a vector species must be sufficiently abundant to ensure frequent feeding on competent hosts. Vectors with small population sizes may play minor roles in transmission but would not provide enough total bites to ensure that the parasite would reliably be transferred between hosts. As an example, *Culex stigmatosoma* is a very competent vector of several bird-borne arboviruses and frequently feeds on birds. The contribution of this species to viral transmission cycles depends on its local abundance, which can be quite high near foulwater habitats in which it breeds and much lower away from these areas.

Host fidelity. A suitable vector feeds frequently on infective vertebrate hosts during periods when stages of the parasite are circulating in the peripheral blood or other tissues accessible to the vector. Host-seeking or biting activity during times or places where primary hosts are

inaccessible or feeding on incompetent hosts will reduce contact with infective hosts and reduce the efficiency of transmission. Patterns of host selection determine the types of parasites to which vectors are exposed. **Anthropophagic** vectors feed selectively on humans and are important in the transmission of parasites that use humans as their primary host. Anthropophagic vectors that readily enter houses to feed on humans or to rest on interior surfaces are termed **endophilic** (literally, "inside loving"). Vectors that rarely enter houses are termed **exophilic** ("outside loving"). **Zoophagic** vectors feed primarily on nonhuman vertebrates. **Mammalophagic** vectors blood feed primarily on mammals and are important in the maintenance of mammalian parasites, whereas **ornithophagic** vectors feed primarily on avian hosts and are important in the maintenance of avian parasites. There is a distinction between vectors attracted to a host (denoted by the suffix "-philic") and those that successfully blood feed on the host (denoted by the suffix "-phagic"). For example, mammalophagic vectors represent the subset of **mammalophilic** vectors that successfully feed on mammalian hosts.

Once infected, the vector must exhibit a high probability of refeeding on one or more susceptible hosts to ensure the transmission of the parasite. Diversion of vectors to nonsusceptible or dead-end hosts dampens transmission effectiveness. The term **zooprophylaxis** (literally, "animal protection") describes the diversion of host-seeking *Anopheles* infected with human malaria parasites from humans to cattle, a dead-end host for the malaria parasites. With zooprophylaxis the dead-end host typically exhibits natural immunity in which host tissues are not conducive to parasites (i.e., do not permit growth or reproduction). Alternatively, transmission to a dead-end host may result in serious illness, because the parasite has not evolved to modulate virulence within the dead-end host. West Nile virus (WNV), for example, can cause serious illness in humans, which are considered to be a dead-end host because they rarely produce a viremia sufficient to infect mosquitoes. In zoonoses such as WNV with complex transmission cycles, *Culex* vector blood feeding on a variety of avian hosts in diverse or complex ecosystems may dampen or dilute amplification transmission, whereas feeding on a reduced variety of highly or moderately competent hosts may enable transmission in simple suburban/urban ecosystems.

Vector competence. The vector must be **competent** (i.e., susceptible to infection and capable of becoming infectious to vertebrate hosts) and must survive long enough for the parasite to complete reproduction and/or development within the vector. Not all arthropods that ingest parasites support parasite replication or development, dissemination, and transmission. For example, the mosquito *Cx. quinquefasciatus* occasionally becomes infected with Western equine encephalomyelitis virus (WEEV); however, because this virus rarely escapes the midgut, this species rarely transmits WEEV. Some arthropods are susceptible to infection under laboratory conditions, but seldom feed in nature on infected vertebrate hosts and/or survive long enough to allow parasite development. *Aedes aegypti*, for example, readily becomes infected with the filarial worm *Brugia malayi* in the laboratory and has been used as a model organism for infection studies, but this mosquito is not considered a vector in nature. Because arthropod vectors are poikilothermic and contact their vertebrate hosts intermittently, parasite transmission rates depend on ambient temperature. Therefore, transmission rates for many parasites are greater at tropical than at temperate latitudes, and transmission at temperate latitudes is most efficient during summer when temperatures are warm. The frequency of host contact and, therefore, the transmission rate also depend on the life history of the vector. For example, Dengue virus (DENV) in the tropics is amplified more rapidly by a mosquito that feeds at 2-day intervals compared with the *Borrelia* spirochetes that cause Lyme disease at temperate latitudes where the hard tick vector has a multi-year life cycle with only one blood meal per life stage per year.

MODES OF TRANSMISSION

The transmission of parasites by vectors may be horizontal or vertical. **Horizontal transmission** describes the passage of parasites between vectors, either by transmission to and from vertebrate hosts or directly between vectors. **Vertical transmission** is the passage of parasites directly to subsequent life stages or generations within vector populations.

Vertical Transmission

Two types of vertical transmission are possible within vector populations: transstadial and transgenerational.

Transstadial transmission is the sequential passage of parasites acquired during one life stage, or stadium, through the molt to the next stage(s) or stadium. Transstadial transmission is essential for the survival of parasites transmitted by mites and hard ticks that blood feed once during each life stage and die after oviposition. Lyme disease spirochetes, for example, that are acquired by larval ticks must be passed transstadially to the nymphal stage before they are transmitted to vertebrates.

Transgenerational transmission is defined as the vertical passage of parasites by an infected vector to its offspring. Some parasites may be maintained transgenerationally for multiple generations, whereas others require horizontal transmission for amplification. Transgenerational transmission normally occurs **transovarially** (through the ovaries) after the parasites infect the ovarian germinal tissue and then are transmitted transstadially to the

next reproductive or blood-feeding stage. Another type of transgenerational transmission is transovum transmission, in which the parasite remains on the surface of the egg after being laid by an infected vector and eventually infects the offspring on eclosion. In true transovarial transmission, most of the progeny are infected, whereas transovum transmission is usually less efficient and results in infection of only a small percentage of the progeny. To be important for parasite persistence, transgenerational transmission in vectors such as mosquitoes must also include transstadial transmission to ensure maintenance of the infection through the reproductive, blood-feeding adult stage.

La Crosse virus (LACV) (Fig. 4.2) is an example of a vertically maintained parasite where the arthropod vector serves as the reservoir. This virus is maintained vertically by transgenerational transmission within clones of infected *Aedes triseriatus* mosquitoes and is amplified by horizontal transmission among squirrels and chipmunks. Because this temperate mosquito is active only during the warmest months of the year, LACV virus spends long periods in infected vectors and relatively short periods in infected vertebrate hosts. Females infected vertically or horizontally transmit their infection transovarially to first-instar larvae. These larvae transmit virus transstadially through the four larval stadia and the pupal stage to the adults. These transgenerationally infected females then take a blood meal and lay infected eggs, often in a tree hole near the one from which they emerged. Venereal transmission of LACV from transgenerationally infected males to uninfected females has been demonstrated in the laboratory and may serve to establish new clones of infected females in nature.

Horizontal Transmission

Horizontal transmission is essential for the maintenance of almost all vector-borne parasites and is accomplished by either anterior (biting) or posterior (defecation) routes. **Anterior-station transmission** occurs when parasites are liberated from the mouth parts or salivary glands during blood feeding (e.g., malaria parasites, encephalitis viruses, filarial worms). **Posterior-station** (or **stercorarian**) **transmission** occurs when parasites remain within the gut and are transmitted via contaminated feces. The trypanosome that causes Chagas disease, for example, develops to the infectious stage within the hindgut and is discharged onto the host skin when the triatomid vector defecates during feeding. Irritation resulting from salivary proteins introduced into the host during feeding causes the host to scratch the bite site and rub the parasite into the wound. Louse-borne relapsing fever and typhus fever rickettsia also use posterior-station modes of transmission.

There are four types of horizontal transmission, depending on the role of the arthropod vector in the life cycle of the parasite: mechanical, multiplicative, cyclodevelopmental, and cyclopropagative.

Mechanical transmission occurs when the parasite is transmitted among vertebrate hosts without amplification or development within the vector, usually by contaminated mouthparts. Arthropods that are associated intimately with their vertebrate hosts and feed at frequent intervals have a greater probability of transmitting parasites mechanically. The role of the arthropod may be little more than an extension of contact transmission between vertebrate hosts.

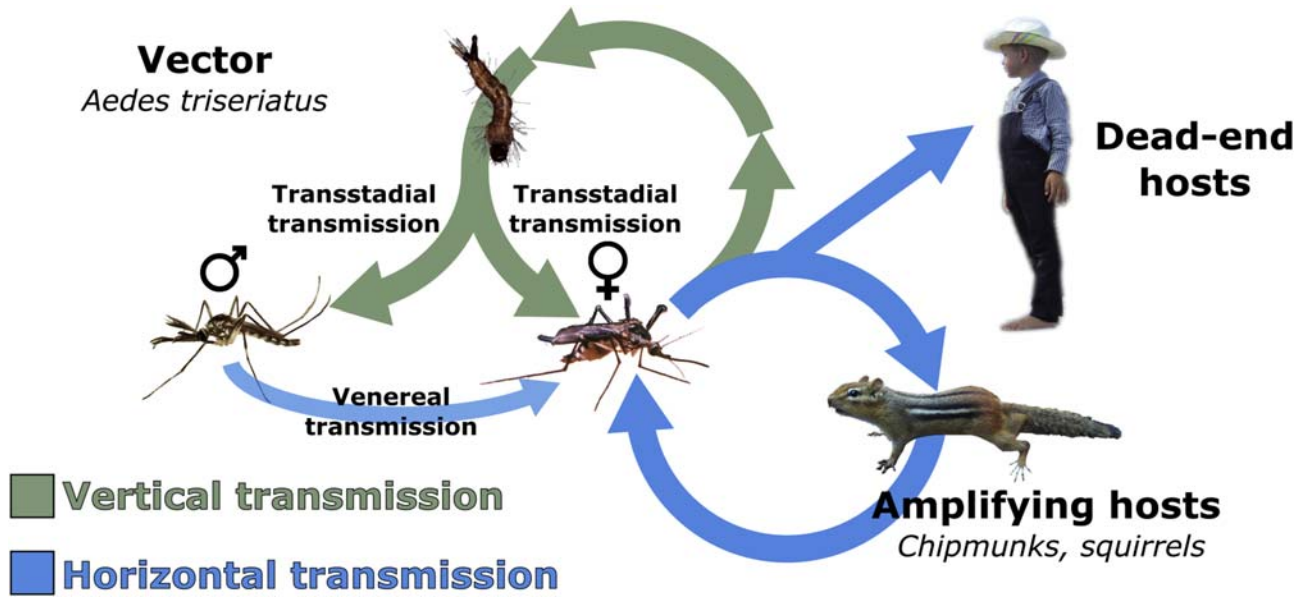


FIGURE 4.2 Modes of transmission of La Crosse encephalitis virus, involving both vertical transmission among mosquitoes (including transovarial, transstadial, and venereal transmission) and horizontal transmission to other host species. Humans are dead-end hosts for this virus. *Modification of original by Nathan D. Burkett-Cadena.*

Eye gnats (*Hippelates* spp.), for example, have sponging mouth parts and feed repeatedly on secretions at the mucous membranes of a variety of vertebrate hosts, making them an effective mechanical vector of the bacteria and viruses that cause conjunctivitis, or "pink eye." Parasites that cause pink eye also may be transmitted from infectious to susceptible hosts via direct contact. For blood-feeding vectors, mechanical transmission may be accomplished by contaminated mouthparts, particularly if the vector is interrupted while blood feeding and then immediately refeeds on a second host in an attempt to complete the blood meal.

In contrast, **nonviremic transmission** is a special form of nonpropagative transmission where the infectious vectors are able to transmit viruses directly to uninfected vectors through concurrent feeding on the same host without the requirement for a systemic infection of the vertebrate host. With tick-borne viruses such as Russian spring-summer encephalitis virus, transmission between co-feeding ticks apparently occurs through the host's skin tissues. With mosquitoes, this has been demonstrated experimentally for WNV when infectious *Culex* mosquitoes inject virus directly into the peripheral circulatory system of small vertebrate hosts such as mice or house finches.

Multiplicative (or **propagative**) **transmission** occurs when the parasite multiplies asexually within the vector and is transmitted only after a suitable incubation period is completed. In this case, the parasite does not undergo metamorphosis (or development) and the form transmitted is indistinguishable from the form ingested with the blood meal. This is exemplified by arboviruses such as WNV, which are not transmitted until the virus infects and escapes the midgut of the mosquito vector, disseminates throughout the hemocoel, and enters and replicates within the salivary glands. With propagative transmission, the number of virions, but not the form of the virus, changes, and the number expectorated by the vector during transmission frequently is much lower than the number ingested with the blood meal.

Venereal transmission is the passage of parasites between male and female vectors during mating and is relatively rare. Venereal transmission usually is limited to transovarially infected males that infect females during insemination, which, in turn, may infect their progeny during fertilization.

Cyclodevelopmental transmission occurs when the parasite develops, but does not multiply, within the vector. Microfilariae of *Wuchereria bancrofti*, for example, are ingested with the blood meal, penetrate the mosquito gut, move to the flight muscles where they molt twice, and then migrate to the mouthparts where they remain until they exit during blood feeding. These filarial worms do not reproduce asexually within the mosquito vector; that is, the number of worms available for transmission is never greater than the number ingested.

Cyclopropagative transmission occurs when the parasite develops and reproduces asexually within the arthropod vector. In the life cycle of the malaria parasite, for example, gametocytes that are ingested with the blood meal unite within the mosquito gut and then change to an invasive form (ookinete) that penetrates the gut and forms an asexually reproducing stage (oocyst) on the outside of the gut wall. After asexual reproduction, this stage ruptures and liberates infective forms (sporozoites) that move to the salivary glands, from where they are transmitted during the next blood meal. Malaria parasites also reproduce asexually within the liver and blood cells of the intermediate human host.

The **extrinsic incubation period** is the time interval from vector ingestion of parasites (infection) to potential parasite transmission (infectiousness), which determines the minimal interval during which the parasite is outside of the vertebrate host. Similarly, the **intrinsic incubation period** is the time from infection of a vertebrate host to potential parasite transmission (infectiousness) from the host to vectors. The intrinsic incubation period is also closely related to the **latent period** from host infection to the onset of symptoms, although infectiousness to vectors often precedes symptoms. Repeated lag periods of consistent duration between clusters of new disease cases at the onset of epidemics were first noticed by early epidemiologists who coined the term "extrinsic incubation." These **serial intervals** actually represent the combined duration of extrinsic incubation in the vector and intrinsic incubation in the host.

The duration of the extrinsic incubation period typically depends on temperature. The rate of parasite development normally increases as a linear degree-day function of ambient temperature between upper and lower thresholds. After being ingested by the mosquito vector, arboviruses such as WNV, for example, must enter and multiply in cells of the midgut, escape the midgut, disseminate throughout the hemocoel, then infect the salivary glands, after which the virus may be transmitted during the mosquito's bite. Under hot summer conditions, this process may be completed within 6 to 7 days, and the vector mosquito, *Culex tarsalis*, is capable of transmitting the virus during the first or second blood meal after becoming infected. In contrast, under cooler spring conditions, transmission may be delayed for more than 2 weeks until the third or fourth blood meal after infection. Some parasites may alter vector behavior by increasing the frequency of vector blood feeding and thereby enhance transmission. The plague bacillus, for example, remains within the gut of the flea vector, *Xenopsylla cheopis,* eventually blocking successful feeding. Regurgitation then occurs during attempted blood feeding, causing vector starvation, more frequent

blood-feeding attempts, and therefore transmission at progressively higher frequency before the vector succumbs to starvation.

TRANSMISSION CYCLES

Transmission cycles vary considerably depending on their complexity and the role of humans as hosts for the parasite. A vector-borne **anthroponosis** is a disease resulting from a parasite that normally infects only humans and one or more anthropophagic vectors (Fig. 4.1). Malaria, dengue fever, some forms of filariasis, and louse-borne typhus are examples of anthroponoses. Humans serve as reservoir hosts for these parasites, which may persist for years as chronic infections. Vectors of anthroponoses selectively blood feed on humans and are associated with domestic or peridomestic environments. Widespread transmission of an anthroponosis with an increase in the number of diagnosed human cases during a specified period of time is called an **epidemic**. When human cases reappear consistently in time and space, transmission is said to be **endemic**.

Vector-borne **zoonoses** are diseases caused by parasites of animals that occasionally infect humans. In most vector-borne zoonoses, humans are not an essential component of the transmission cycle and may not contribute to transmission but rather become infected when bitten by a vector that fed previously on an infectious animal host. Such infections may cause illness, but humans rarely circulate sufficient numbers of parasites to infect vectors and thus are termed **dead-end hosts**. The **enzootic** (literally "in animals") **cycle** is the basic or primary transmission cycle. When levels of enzootic transmission escalate, transmission may become **epizootic**, characterized by a sharp increase in transmission among animals. Spillover transmission from the enzootic cycle to dead-end hosts is called **tangential transmission** (i.e., peripheral to the basic transmission cycle). Often, different vector species are responsible for enzootic, epizootic, and tangential transmission. **Bridge vectors** transmit parasites tangentially between different enzootic and dead-end host species. The degree to which humans are affected by zoonoses may be increased if a secondary **amplification cycle** becomes established among vertebrate hosts inhabiting the peridomestic environment.

Lyme disease, caused by infection with the spirochete *Borrelia burgdorferi*, is an example of a tick-borne zoonosis (Fig. 4.3) that is now epidemic in North America. If left untreated, the spirochete causes serious chronic disease in humans, presenting a variety of symptoms that may include meningoencephalitis, myocarditis, frank arthritis, and fatigue. The vectors are principally ticks in the *Ixodes ricinus* complex including *I. scapularis* in eastern and *I. pacificus* in western North America. Hard ticks require blood meals for both molting and reproduction. Larval ticks acquire *Borrelia* by blood feeding on mice during summer that have infectious spirochetemias (elevated numbers of spirochetes in the blood). The ticks maintain infections during winter, and then pass the infection transstadially to the nymphal stage the following spring (Fig. 4.3). Nymphal ticks subsequently transmit their infection to a variety of hosts, including rodents, squirrels,

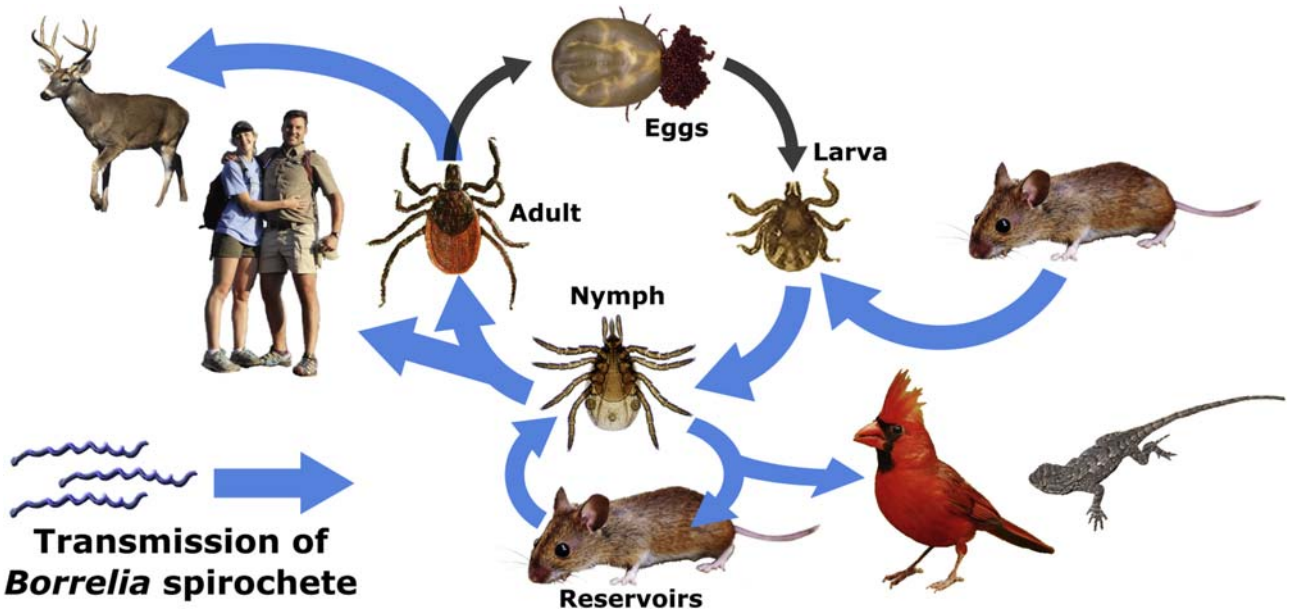


FIGURE 4.3 Components of the transmission cycle of a zoonosis, such as Lyme disease. In this case, the pathogen, a spirochete of the genus *Borrelia*, is maintained by transmission among nonhuman hosts (e.g., woodland mice) but also can be transmitted to humans, deer, and other vertebrates. *Modification of original illustration by Nathan D. Burkett-Cadena.*

lizards, birds, and humans but, if uninfected, may acquire *Borrelia* during blood feeding. Lizards, some birds, and humans are refractory or dead-end hosts, and their infection may actually reduce the rate of *Borrelia* amplification. Infected nymphs also pass their infection transstadially to the adult stage, and adults may transmit to large mammals during blood feeding, although deer also seem to be refractory to infection. There is minimal evidence to support vertical transmission of *Borrelia* to the eggs, and therefore larval ticks and mice seem to be the reservoirs of infection. The changing landscape and reforestation of eastern North America, accompanied by large increases in white-tailed deer and *Peromyscus* mouse populations, and the construction of housing adjacent to or within wooded areas have combined to create epidemiological situations that have led to a widespread rise in Lyme disease incidence over several decades. Infected immature tick populations residing in suburban gardens and lawns greatly increase the risk of transmission to humans.

INTERSEASONAL MAINTENANCE

An important aspect of the ecology of vector-borne parasites is the mechanism(s) by which they persist between active transmission seasons or outbreaks. Parasite transmission typically is most efficient when weather conditions are suitable for vector activity and population growth, and warm temperatures expedite parasite replication and dissemination within the vector. In temperate latitudes, survival during the colder months is a challenge for many parasites, because most vertebrate hosts or arthropod vectors either enter winter dormancy or migrate. Tropical parasites face similar obstacles to persistence, when transmission is interrupted by prolonged periods of less favorable weather or when herd immunity in vertebrate hosts limits horizontal transmission efficiency. The apparent seasonality that is characteristic of most vector-borne parasites may be due to either the periodic amplification of a constantly present parasite or to the consistent reintroduction of parasites after local extinction.

There are several mechanisms of parasite maintenance or reintroduction after periods of unfavorable conditions.

Continued transmission by vectors. During periods of unfavorable weather, vectors may remain active and continue to transmit parasites, although transmission rates may be slowed by cold temperature or low vector abundance. In temperate latitudes with cold winters, transmission may continue at a slow rate, because the frequency of blood feeding and rate of parasite maturation in the vector are diminished. In tropical latitudes, widespread transmission may be terminated during extended dry seasons that reduce vector abundance and survival. In both instances, transmission may be restricted spatially and involve only a small portion of the vertebrate host population. Human infections during adverse periods may be sporadic and highly clustered, sometimes even restricted to members of the same neighborhood or household.

Infected vectors. Many vectors are capable of entering a state of dormancy as immatures or adults before blood feeding. If vertically infected, such vectors typically remain infected for life and therefore may maintain parasites through periods when horizontal transmission is interrupted. California encephalitis virus, for example, is maintained during winter and drought periods within the transovarially infected eggs of its vector, *Aedes melanimon*. Infected eggs of this floodwater mosquito may remain dormant and infected between years and are able to withstand winter cold, summer heat, and extended dry periods. Inundation of eggs during spring or summer produces a cohort of adult mosquitoes that are infected at emergence. Tick-borne parasites, such as Lyme disease, persist through winter within infected immature stages of the tick vector. Similarly, vectors that inhabit the nests of migratory avian hosts such as cliff swallows often remain alive and infected for extended periods until their hosts return.

Infected vertebrate hosts. Parasite maintenance may be accomplished by infected reservoir hosts that either continue to produce stages infective for vectors beyond the acute stage of infection or harbor inactive stages of the parasite that relapse or recrudesce during the season when vectors are blood feeding. Adult filarial worms, for example, continue to produce microfilariae throughout their lifetime, regardless of the population dynamics or seasonality of the mosquito vector. In contrast, some Korean strains of *Plasmodium vivax* malaria overwinter as dormant stages in the liver of the human host and then relapse in spring concurrent with the termination of diapause by the mosquito vector(s).

Alternatively, parasites may become locally extinct during unfavorable weather periods or stochastic fadeout of transmission and then become reintroduced from distant transmission foci. Two possible mechanisms may allow the reintroduction of parasites:

Migratory vertebrate hosts. Many bird species overwinter in the tropics and return to temperate or subarctic breeding sites each spring, potentially bringing with them infections acquired at lower latitudes. It also is possible that the stress of long flights and ensuing reproduction triggers relapses of chronic infections. In addition, many large herbivores migrate seasonally between summer and winter foraging grounds, bringing with them an array of parasites. Rapid long-range human or commercial transportation is another possible mode for vector and parasite introduction. The seasonal movement of agricultural products, livestock, or migratory agricultural workers may drive seasonal patterns of some vector-borne diseases.

Weather fronts. Infected vectors may be carried long distances by prevailing weather fronts. Consistent weather

patterns such as the sweep of the southeastern monsoon from the Indian Ocean across the Indian subcontinent may passively transport infected vectors over hundreds of kilometers. The onset of WEEV activity in north central United States and Canada has been attributed to the passive dispersal of infected mosquitoes by storm fronts, as has the repeated introduction of Japanese encephalitis virus into northern Australia.

VECTOR INCRIMINATION

To understand the epidemiology and control of a vector-borne disease, it is essential to establish which arthropod(s) is the primary vector(s) responsible for parasite transmission. Partial or incomplete vector incrimination has resulted in the misdirection of control efforts at arthropod species that do not play a substantial role in either enzootic maintenance or epidemic transmission. In Southeast Asia, for example, surveys have repeatedly found *Anopheles umbrosus* positive for sporozoites, but later it was learned that these were not of human origin and that this species was not an important target vector for human malaria control. The steps to vector incrimination combine field and laboratory data that estimate field infection rates, vector competence, and vectorial capacity.

Infection rates. The collection of infected arthropods in nature is an important first step in identifying potential vectors, because it indicates that the candidate species feeds on vertebrate hosts carrying the parasite. Vector infection data may be quantified as **infection prevalence,** which is the proportion infected at a specified point in time (i.e., number of vectors infected/number examined). In common use, this quantity is often referred to as the "**infection rate**," although it is not a true rate because it does not quantify a change in infections over time. When the infection prevalence is low and arthropods are tested in groups known as pools, infection prevalence is often estimated as the **minimum infection rate** (number of pools of vectors parasite-positive/total individual vectors tested) with the assumption that only one individual is infected in a positive pool. This number is commonly expressed per 1,000 vectors tested, because of the typically low positivity rate. The theoretical upper limit of the minimum infection rate is determined by pool size. For example, minimum infection rates of vectors tested in pools consisting of 50 individuals each could be no higher than 20 per 1,000 vectors tested even if all pools were positive. Statistical estimates of infection prevalence using pooled samples overcome this limitation by accounting for the possibility that more than one mosquito could be positive in each pool, particularly when prevalence is high.

It is important to distinguish between infected hosts harboring a parasite and infectious hosts capable of transmission. For vector-parasite pairings that involve cyclodevelopmental or cyclopropagative transmission, infectious stages may be distinguished by location in the vector, morphology, or biochemical properties. For propagative transmission typical of viruses or bacteria, it is virtually impossible to distinguish infectious from infected-but-noninfectious vectors in routine monitoring, because the parasite form does not change, although the location of recovery within the vector can be informative. The ability to transmit may be inferred by testing selected body parts such as the cephalothorax, salivary glands, or head. With some tick pathogens, however, parasite movement to the mouthparts does not occur until several hours before detachment.

The **entomological inoculation rate** is the number of potentially infectious bites per host per unit of time. This frequently is determined from the human or host biting rate and the proportion of vectors that are infectious and is calculated as bites per human per night x prevalence of infectious vectors. In malaria, for example, this is the average number of *Anopheles* positive for sporozoites biting a human per time period.

Vector competence is defined as the susceptibility of an arthropod to become infected and transmit a parasite to a vertebrate host. Vector competence typically is determined experimentally by feeding the candidate arthropod vector on a vertebrate host circulating the infective stage of the parasite, incubating the blood-fed arthropod under suitable ambient conditions, refeeding the arthropod on an uninfected susceptible vertebrate host, and then examining this host to determine if it became infected. Because it often is difficult to maintain natural vertebrate hosts in the laboratory and control the concentration of parasites in the peripheral circulatory system, immune-deficient strains of laboratory animals or artificial feeding systems frequently are used to expose the vector to the parasites. **Susceptibility to infection** may be expressed as the percentage of arthropods that became infected among those blood feeding. When the arthropod is fed on a range of parasite concentrations, susceptibility may be expressed as the **median infectious dose**, which is the concentration required to infect 50% of the blood-fed arthropods ($\mathbf{ID_{50}}$). The arthropod's ability to transmit may be expressed as the percentage of feeding arthropods that transmitted the parasite or the percentage of recipient hosts that became infected.

Failure of a blood-fed arthropod to become infected with or transmit a parasite may be attributed to the presence of one or more barriers to infection. For most parasites, the arthropod midgut provides the most important barrier to infection. Often, parasites will replicate in a nonvector species if they are inoculated into the hemocoel, thereby bypassing this **gut barrier**. After penetrating and escaping from the midgut, the parasite then must multiply and/or mature and be disseminated to the salivary glands or

mouthparts. Arthropod cellular or humoral immunity may clear the infection at this point, creating a **dissemination barrier**. Even after dissemination to the salivary glands, the parasite may not be able to infect or be transmitted from the salivary glands due to the presence of salivary gland infection or salivary gland **escape barriers**, respectively. For parasites transmitted at the posterior station, vector competence may be expressed as the percentage of infected vectors passing infectious stages of the parasite in their feces.

Vectorial capacity. The formula for vectorial capacity summarizes the contributions of the vector to parasite transmission. Although developed for the mosquito vectors of malaria parasites and most easily applied to anthroponoses, the components of the model provide a framework to conceptualize how the ecological components of the transmission cycle of many vector-borne parasites interact.

Vectorial capacity is expressed by the formula:

$$C = \frac{ma^2bp^n}{-\ln(p)}$$

where C = vectorial capacity as the average number of future infectious bites that arise from the vectors feeding on a single infectious host per day, m = vectors per human, a = human biting habit (biting rate x proportion feeding on humans), b = vector competence, p = probability of daily survival, and n = extrinsic incubation period in days.

The human **biting rate**, ma, frequently is estimated by human landing counts that involve collecting vectors as they attempt to blood feed and is expressed as bites per unit of time (e.g., 10 mosquitoes per human per night). The human biting habit, a, is the product of vector blood-feeding frequency and the fraction blood feeding on humans. **Feeding frequency** is the length of time between blood meals and frequently is expressed as the inverse of the length of the gonotrophic cycle. Host selection patterns are determined by testing blood-fed vectors to determine what percentage fed on humans or the primary reservoir. Therefore, if the vector blood feeds once every 2 days and 50% of host-seeking vectors feed on humans, $a = (1 \text{ bite}/2 \text{ days}) \times (0.5) = 0.25$. In this example, $ma^2 = 10$ bites/human/night $\times\ 0.25 = 2.5$. The parameter a is repeated, because infected vectors must refeed to transmit.

The probability of the vector surviving through the extrinsic incubation period of the parasite, p^n, requires information on the probability of vector survival, p, and the duration of the extrinsic incubation period, n. The parameter p is estimated either vertically by age-grading the vector population or horizontally by marking cohorts and monitoring their loss rate over time. In Diptera, p may be estimated vertically from the parity rate (proportion of parous females/number examined). In practice, p = (parity rate)$^{1/g}$, where g is the length of the gonotrophic cycle. The extrinsic incubation period may be estimated from ambient temperature from data gathered during vector competence experiments by testing the time from infection to transmission for infected vectors incubated at different temperatures. Continuing our example, if $p = 0.8$ and $n = 10$ days, then the duration of infective life, $\frac{p^n}{-\ln(p)} = \frac{0.8^{10}}{-\ln(0.8)} = 0.48$. In addition, it is useful to also account for vector competence, b. For this example, we will assume that 90% of vectors become infected and 90% of infected females are capable of transmission, so $b = 0.9 \times 0.9 = 0.81$. Therefore, vectorial capacity, $C = 2.5 \times 0.48 \times 0.81$ or 0.97 future parasite transmission events are generated per infective host per day.

SURVEILLANCE

The number of cases of most vector-borne diseases typically varies over both time and space. Surveillance (from the French for "watching over") programs typically monitor these diseases to measure their impact on public or veterinary health. Information on the number of cases can be gathered from morbidity and mortality records maintained by state or national government agencies for the human population. **Morbidity data** are records of illness, whereas **mortality data** are records of the causes of death. These data vary greatly in their quality and timeliness, depending on the accuracy of determining the cause of illness or death and the rapidity of reporting. In the United States, the occurrence of confirmed cases of many vector-borne diseases, including yellow fever, plague, malaria, and encephalitis, are **notifiable diseases**, meaning that the law requires physicians to report them to municipal health authorities. However, infections with many arthropod-borne parasites such as Lyme disease and the mosquito-borne encephalitides frequently are asymptomatic or present variable clinical symptoms and therefore remain largely undiagnosed and underreported. The frequency of case detection and accuracy of reporting systems are dependent on the type of surveillance used and the ability of the medical or veterinary community to recognize suggestive symptoms and request appropriate confirmatory laboratory tests. In addition, some laboratory tests vary in their specificity and sensitivity, complicating the interpretation of laboratory results. Cases may be classified as suspect or **presumptive** based on the physician's clinical diagnosis or as **confirmed** based on a diagnostic rise in specific antibodies or the direct observation (or isolation) of the parasite from the case. Surveillance for clinical cases may be active or passive.

Active surveillance involves active case detection in which health workers seek to identify cases by reviewing hospital records or visiting communities to test suspect cases that fit a predetermined case definition. In malaria

control programs, for example, a field worker visits every household biweekly or monthly and collects blood films from all persons with a current or recent fever. Fever patients are treated with antimalarial drugs presumptively, and these suspect cases are confirmed by detection of malaria parasites in a blood smear. Confirmed cases are revisited and additional medication is administered, if necessary. This surveillance provides population infection rates regardless of case classification criteria.

Most surveillance programs rely on **passive surveillance,** which uses passive case detection to identify clinical human or veterinary cases. In this system, individuals seeking medical attention at primary health care organizations such as physicians' offices, hospitals, and clinics are diagnosed by an attending physician who requests appropriate confirmatory laboratory tests. However, because many arthropod-borne diseases present a variety of nonspecific symptoms (e.g., headache, fever, general malaise, arthralgia), cases frequently may be missed or not specifically diagnosed. In mosquito-borne viral infections, the patient often spontaneously recovers, and cases frequently are listed under fevers of unknown origin or aseptic (or viral) meningitis without a specific diagnosis. In a passive case detection system, it is the responsibility of the attending physician to request laboratory confirmation of suspect clinical cases and then to notify public health officials that a case of a vector-borne disease has been documented.

The reporting system for clinical cases of vector-borne diseases must be evaluated carefully when interpreting surveillance data. This evaluation should take into account the disease, its frequency of producing clinically recognizable symptoms, the official case definition, the sensitivity and specificity of confirmatory laboratory tests, and the type and extent of the reporting system. Usually programs that focus on the surveillance of a specific disease and use active case detection provide the most reliable epidemiological information. In contrast, broad-based community health care systems that rely on passive case detection typically produce the least reliable information, especially for relatively rare vector-borne diseases with nonspecific symptoms. Unfortunately, the extent of diagnosis and therefore the sensitivity of surveillance are increasingly dependent on the extent of medical insurance coverage and the physician's decision to pursue a definitive diagnosis.

Diseases that are always present or reappear consistently at a similar level during a specific transmission season are classified as **endemic**. The number of cases in a population is expressed as incidence or prevalence. Population is defined as the number of individuals at risk from infection in a given geographical area at a given time. **Prevalence** is the proportion of a population infected with a parasite at a single point in time, which typically is estimated using a cross-sectional survey and expressed as the percentage of the population tested that was found to be infected. **Incidence** is the number of new cases per unit of population per unit of time. Incidence data may be inferred from changes in prevalence from consecutive surveys or estimated from a longitudinal study that follows a cohort of individuals over time.

The level of parasite endemicity in a population may be graded as **hypoendemic** (low), **mesoendemic** (medium), or **hyperendemic** (high), depending on the incidence of infection and/or the immune status of the population. In malaria surveys, for example, the percentage of children with palpable spleens (evidence of parasite infection) and the annual parasite incidence are used to characterize the level of endemicity. In endemic disease, the percentage of individuals with sera positive for IgG antibodies typically increases steadily as a function of age or residence history, whereas in hypoendemic disease with intermittent transmission, the rise in seroprevalence with age is not monotonic, and certain age groups often express elevated positivity rates. The occurrence of an above-normal number of human infections or cases is termed an **epidemic**. Health agencies, such as the World Health Organization, typically monitor incidence data to establish criteria necessary to classify the level of endemicity and to decide when an epidemic is under way. A geographically widespread epidemic on a multicontinental or global scale is called a **pandemic.**

Serological surveys (or **serosurveys**) are a useful epidemiological tool for determining the cumulative infection history of a population with one or more parasites. Serosurveys also may provide information about herd immunity levels in host populations that could affect the efficiency or risk of transmission, and changes in antibody prevalence between sequential serosurveys can provide estimates of reinfection rates. When coupled with morbidity data, serosurveys provide information on the ratio of apparent to inapparent infections. **Random sampling** representatively collects data on the entire population and may provide ecological information retrospectively by analysis of data collected concurrently with each serum sample. This information may be used to understand variation in the risk for infection within population strata such as sex, occupation, and residence history; comparison of exposure data from serosurveys with disease data from surveillance also may help in ascertaining age-related differences in susceptibility to disease. **Stratified sampling** divides the total population into subpopulations based on geographical areas, age, or other criteria. Sampling may be targeted for key risk groups, and stratified sampling that emphasizes high-prevalence times or places may have greater sensitivity for detecting and estimating the incidence of rare or unevenly distributed parasites. Repeated serological testing of the same individuals within a

population can determine the time and place of infection by determining when individuals first become **seropositive** (i.e., serologically positive with circulating antibodies against a specific parasite). This change from seronegative to seropositive is called a **seroconversion**.

Forecasting the risk of human infection usually is accomplished by surveillance of environmental factors, vector abundance, the level of transmission within the primary and/or amplification cycles, and the numbers of human or domestic animal cases. As a general rule, the accuracy of forecasting declines with increased lead time and geographic distance between the surveillance data and the time and place targeted for prediction. Monitoring of viral amplification between vectors and nonhuman hosts in primary cycles is particularly critical for zoonotic vector-borne parasites, because human cases are relatively rare for many pathogens and occur only after transmission has reached a level high enough to spill over from primary cycles. Also, reporting of human disease often occurs after the optimal time window for vector control has passed.

Environmental conditions. Unusually wet or warm weather may indicate favorable conditions for vector activity or population increases in abundance, concurrently increasing the risk of parasite transmission. Variables that are frequently monitored include temperature, rainfall, snowpack (predictive of vernal flooding), and agricultural irrigation. Recently, general circulation models and the real-time monitoring of ocean temperatures have been used to predict broad climate variation. In the Pacific Ocean, for example, warming of sea-surface temperatures and pressure differences in the equatorial Pacific Ocean (termed the "El Niño − southern oscillation") can forecast rainfall patterns in North America and east Africa. El Niño patterns leading to increased rainfall and flooding of mosquito habitats in east Africa have been used to successfully predict outbreaks of Rift Valley fever virus several months in advance.

Vector abundance. Standardized vector population monitoring at fixed locations and time intervals can be used to compare relative changes in temporal and spatial vector abundance that are useful in detecting increased risk of parasite transmission. Collection methods (e.g., traps versus active sampling) and choices of sampling locations are critical for effectively sampling vectors such as mosquitoes for surveillance purposes. Sampling sites should be in or near habitats that maximize their effectiveness in collecting the target vector species. Sampling in a systematic fashion over time can produce historical baselines useful in determining anomalous increases or decreases in abundance. Unusual increases in vector abundance and survival may provide an early warning of incipient enzootic transmission that can lead to epidemics.

Parasite transmission. Systematically monitoring the level of parasite infection in vector or vertebrate populations provides direct evidence that the parasite is present and actively being transmitted. For zoonotic diseases, the level of enzootic transmission usually is directly related to the risk of spillover transmission to humans or domestic animals. Transmission activity may be monitored by the use of vector infection rates, vertebrate host infection rates, sentinel seroconversion rates, and clinical cases.

Vector infection rates. Sampling vectors and testing them for parasites allows for estimation of the level of infection in the vector population in various habitats. When vectors are tested individually, prevalence data are expressed as percentages; for example, 10 females infectious with sporozoites per 50 tested would result in an estimate of 20% infectious prevalence. When combined with abundance estimates, infectious rates also may be expressed as infectious vectors per sampling unit per time interval; 10 bites per human per night × 0.2 infectious prevalence = 2 infectious bites per human per night. These data provide an index of the transmission rate or the **entomological inoculation rate**. When infectious rates are low, vectors are abundant, and sampling is independent of vector age, vectors usually are tested for infection in lots or pools. This is especially useful for parasites such as viruses where it is not possible to distinguish infectious stages. The upper limit on pool size should account for expected infection prevalence (larger pools may be used if expected prevalence is low) and the sensitivity limits of the laboratory testing method used to detect infection. Infection prevalence is usually expressed as a minimum infection rate = parasite-positive pools/total vectors tested. There are recent mathematical approaches for calculating prevalence estimates and confidence intervals from pooled samples that rely on maximum likelihood estimation methods and allow for the possibility that more than one vector may be positive in a single pool.

Vertebrate host infection rates. Introduced zoonoses, such as sylvatic plague in prairie dogs and other North American rodents, frequently produce elevated mortality in nonhuman hosts that may be used to monitor epizootics of these parasites over time and space. Large numbers of dead American crows have been a hallmark of WNV outbreaks since its invasion of North America (Fig. 4.4), and counts of reports by the public have been used for surveillance purposes to indicate recent transmission as well as to forecast human risk using predictive spatial models. In contrast, long-established, endemic zoonoses rarely result in discernible vertebrate host mortality. St. Louis encephalitis virus, an endemic North American virus closely related to WNV, rarely causes mortality in avian host populations. Testing reservoir or amplifying hosts for infection is often necessary to monitor the level of enzootic parasite transmission. Stratified sampling (directly by parasite isolation or indirectly by seroprevalence) usually targets newborn, immunologically naïve hosts or age-related patterns in immunity to understand changes in

FIGURE 4.4 A dead American crow; reports of dead birds and confirmation of the cause of death by testing specimens for virus provide a means of monitoring acute infections in vertebrate reservoir populations.

transmission over time. For example, testing nestling birds for viremias can provide information on the level of enzootic encephalitis virus transmission.

Monitoring the incidence of newly infected individuals in a population over time is necessary to detect increased transmission activity. Because many parasites cause ephemeral parasitemias or are present only for a limited time period, sampling frequently emphasizes the monitoring of seroprevalence. Figs. 4.5 and 4.6 show methods for collecting and taking blood from free-ranging birds to detect infection status by monitoring seroprevalence. IgM antibody, which rises rapidly after infection, is parasite specific and decays relatively quickly, so IgM testing can indicate the level of recent infection, whereas monitoring IgG antibody (IgY in birds) documents the population's past exposures to the parasite. Detection of antibodies in juvenile birds implies recent transmission activity. Sampling, marking, releasing, recapturing, and resampling wild animals are most useful in providing information on the time and place of infection in free-roaming animal populations.

Sentinel seroconversion rates. Sentinels typically are animals that can be monitored over time to quantify the incidence of new parasite infections. Trapping wild animals or birds is labor intensive, and seroprevalence in wild vertebrates may provide little information on the time and place of infection, especially if the host species has a large home range. To circumvent this problem, caged or tethered natural hosts or suitable domestic animals of known infection history are placed in areas where parasite transmission is to be monitored and are repeatedly bled to detect infection. To ensure maximum sensitivity, a suitable sentinel should be fed on frequently by the primary vector species, easy to diagnose when infected, unable to develop a circulating parasitemia that could infect additional vectors, not succumb to infection, and be inexpensive to maintain and easy to bleed or otherwise sample for infection. Chickens, for example, are useful sentinels in

FIGURE 4.5 A mist net used to capture birds to monitor for seroprevalence as an indication of previous viral infection.

FIGURE 4.6 Taking a blood sample from a California quail caught in a mist net.

FIGURE 4.7 A coop used to house sentinel chickens.

mosquito-borne encephalitis virus surveillance programs (Figs. 4.7 and 4.8). Flocks of seronegative chickens are placed at farm houses or other suitable localities, housed in standard coops covered with coarse wire mesh to permit mosquito access. Chickens are then bled weekly or biweekly to determine seroconversions to WEEV, St. Louis encephalitis virus, or WNV. Small blood samples taken on filter paper (Fig. 4.8) can be tested with use of a semi-automated enzyme immunoassay to detect seroconversions. Because the chickens are confined and the date of seroconversion is known within a 1- to 2-week interval, the time and place of infection can be determined, and the number of chickens seroconverting per flock provides an estimate of the relative intensity of transmission.

Clinical cases. Detecting infection among domestic animals may be an important indication that an epizootic is under way and that the risk of human infection is elevated.

FIGURE 4.8 Collection of blood droplets on a strip of filter paper after pricking the comb of a chicken with a lancet; individual birds can be bled systematically over time to detect seroconversion.

Domestic animals such as horses often are more exposed to vectors than are humans and therefore provide a more sensitive indication of parasite transmission, unless they are protected by vaccination. For pathogens that amplify in rural foci, clinical human cases in rural areas in close association with primary transmission cycles may presage increased risk for epidemic transmission in urban settings. For most arboviruses (e.g., WNV), most human infections are inapparent. Symptoms for clinical cases are varied and range from mild fever through severe neurological disease, often with debilitating sequelae and occasionally death.

Many vector-borne diseases affect only a small percentage of the human population, are difficult to predict in space and time, or do not impart long-lasting protective immunity. This limits the usefulness of vaccines and makes vector control and personal protective measures the most effective intervention methods for limiting risk for human disease. Control programs attempt to maintain vector abundance below thresholds for the transmission of parasites to humans or domestic animals. When these programs fail, personal protection with repellents or insecticide-impregnated clothing, bed nets, or curtains is often the only recourse. Vaccination is a viable method of control for specific vector-borne diseases, if the vaccine imparts lasting immunity, as in the case of yellow fever virus. However, many parasites such as the *Plasmodium* sp. that cause malaria elicit a weak immune response that provides only short-term and partial protection. The need for continued revaccination at short intervals severely limits a vaccine's value, especially in developing countries where delivery systems can be difficult and inconsistent. Although breakthroughs in chemotherapy have been useful in case management, it remains the mandate of the medical-veterinary entomologist to devise strategies that combine epidemiological and ecological information to effectively reduce or eliminate the risk of vector-borne diseases.

EMERGING VECTOR-BORNE DISEASES

An expanding and rapidly growing human population, increased and rapid travel, the globalization of commerce, and a variety of anthropogenic factors, including climate warming and urbanization, have produced conditions conducive for the emergence and/or resurgence of infectious diseases, including many transmitted by vectors. By definition, an **emerging disease** has shown a recent increase in incidence, severity, and/or distribution and threatens to continue to increase in the future. The return of the mosquito *Ae. aegypti* into many of the areas where it had been eradicated during campaigns against yellow fever in the 1960s has allowed for the expansion of DENV. Sequential epidemics of different DENV serotypes also have led to increased risk for severe disease, including fatal dengue hemorrhagic fever or shock syndrome.

In recent years, three mosquito-borne viruses have spread rapidly throughout the Americas, highlighting the need for better tools to combat exotic pathogens and predict their emergence. WNV spread from its point introduction in New York throughout temperate North America and then into the neotropics and across most of South America within a 6-year period from 1999 to 2005. Chikungunya and Zika viruses, which are in different viral families but are transmitted by the same urban *Ae. aegypti* mosquitoes, have also spread from long-established enzootic foci in Africa to many other parts of the globe, including much of the Americas since 2013. These newly emerging viruses provide useful case studies, because their emergence has been dependent on a series of historical, genetic, and anthropogenic factors that converged to enable the emergence and spread of these vector-borne diseases.

Many emerging diseases have been enabled by a confluence of events that have altered human demography and vector or pathogen distributions, spurred by anthropogenic environmental change and pathogen evolution, to facilitate emergence to epidemic form in areas outside of historic boundaries.

1. **Human migration.** Human movements have established routes repeatedly used by populations and exploited by vectors and parasites. For example, the movement of Indian and African diaspora facilitated the spread of Chikungunya virus (CHIKV) out of Africa and into India and other areas around the Indian Ocean.
2. **Globalization of commerce.** The globalization of commerce, originally by the sailing ships of the colonial European empires, and recently by the rapid international exchange of goods such as in the used tire trade, established conditions suitable for the inadvertent transport of *Ae. aegypti* and *Ae. albopictus,* respectively. Both species lay drought-resistant eggs in dark areas such as water barrels or tires that collect rainwater, enabling the transport of immature stages and the global establishment of both effective arbovirus vectors.
3. **Diagnostics.** There was initial confusion relating to the recognition of CHIKV and WNV, both of which cause diverse symptoms that can be difficult to diagnose. CHIKV symptoms are similar to those of dengue fever, and most likely many cases in Africa and Asia were initially confused with dengue until serology was used to support clinical diagnosis. For WNV, the original identification cases based on serological specimens as closely related St. Louis encephalitis virus delayed recognition and still complicates serological diagnosis in humans and birds. In addition, the severity of febrile illness for WNV was not recognized until 2002, when an expanded case definition was finally published, greatly expanding the known scope of the ongoing epidemic.
4. **Anthropogenic changes.** The global human population has tripled in size during the past 75 years, increasing the demand for resources, altering the environment, and bringing human populations into more frequent contact. This has changed landscapes, reduced ecological diversity and increased the number and size of population centers. As of 2012, for the first time in history, more people lived in urban than in rural settings. The movement of rural populations to urban centers in developing countries has resulted in poorly planned population centers with little municipal infrastructure and the creation of inadequate housing where water must be stored for domestic use and wastewater and solid wastes are disposed in open drains. These urban slums have created conditions suitable for the explosion of peridomestic mosquitoes such as *Ae. aegypti,* a highly efficient vector that feeds predominantly on humans, as well as the viruses they transmit. In North America, modern urbanization has led to the creation of irrigation systems and municipal drainage systems that provide an ideal habitat for the production of large populations of *Culex* mosquitoes. In addition, reduced avian diversity and increases in peridomestic species including American crows, house sparrows, and house finches—all competent hosts for WNV—has enabled highly efficient urban transmission by mosquitoes in the *Culex pipiens* complex. Ecosystem simplification by both agriculture and urbanization seems to have created situations that make amplification more rapid and transmission more efficient than in complex rural ecosystems where vector biting is distributed over a range of vertebrate species, often of varying host competence. This is especially true for Chikungunya and Zika viruses where transmission is human–*Aedes* mosquito–human in tropical urban centers. Global warming or climate change is another anthropogenic factor that may contribute to the receptivity of northern latitudes for previously tropical viruses. Increased temperatures have created warmer and longer summers at northern latitudes, potentially extending the annual season of risk for vector-borne diseases. This may be especially true for WNV where increased human disease incidence in the great plains of the United States and Canada has coincided with above-normal summer temperatures.
5. **Pathogen evolution.** The invasive strains of both CHIKV and WNV differed from their ancestral genotypes, and these changes seem likely to have contributed to their emergence. For CHIKV, genetic changes may have allowed the shift from forest *Aedes* and *Culex* enzootic vectors to *Aedes albopictus* in the African offshore islands of Comoros. In contrast, mutation of the WNV helicase gene initially led to increased virulence in crows, enabling the infection of moderately susceptible members of the *Culex pipiens* complex at northern

latitudes and then the emergence of a North American WN02 genotype that is more efficiently transmitted by *Culex* mosquitoes, enabling rapid virus amplification at northern latitudes. During the epidemic expansion of both viruses, the genotype of the invading virus has remained relatively conserved, despite the relatively high mutation rate in flaviviruses and selection pressure to adapt to local conditions.

New Tools

Recent progress in molecular genetics and computing have provided medical entomologists new tools to understand the biology, ecology, and epidemiology of vector-borne pathogens.

Molecular biology. The rise of molecular tools has opened many exciting avenues for research on vector-borne diseases from the scale of individual organisms or tissues to populations. Parasite infections trigger a cascade of molecular processes that are governed primarily by the genetics of the parasite and its vector or host. These interactions determine the course of disease and immunity and whether parasites will be transmitted. The range of potential interactions between host, vector, and parasite genotypes and resulting phenotypes will form the basis for many future careers for medical entomologists. Population genetics and other molecular methods also offer a growing array of tools to understand the patterns of mixing within host, vector, and parasite populations. Mixing is the result of movement and mating, and genomics and proteomics are offering new abilities to resolve these patterns at very fine scales that are critical for understanding variation in vector-borne disease transmission.

Modeling. Vectorial capacity, entomological inoculation rate, and related modeling concepts presented earlier in this chapter provide an excellent conceptual framework for understanding the parameters that regulate transmission. However, this is only a start. Growth in computational power and the emergence of large databases in recent decades have allowed for the rise of modeling as an indispensable discipline within medical entomology. Complemented by the new molecular methods described here, statistical and mathematical models are now able to address the complexities of vector-borne disease systems in more mechanistic ways than ever before. Dynamical and stochastic processes can now be studied in great detail at a range of scales, which provides an ever-expanding window into spatial and temporal heterogeneities in vector-borne disease. Prediction remains a challenge for many vector-borne diseases, and models that explicitly account for the sampling process and estimate uncertainty about the future or unobserved places offer ways forward that are sure to yield exciting new maps and forecasts from future medical entomologists.

Medical entomologists play a critical role in preventive medicine programs charged with detecting and investigating the emergence of new vector-borne pathogens and developing new strategies for their control. Urbanization and the invasion of previous wilderness areas will continue to be accompanied by veterinary and public health challenges from vector-borne diseases. Devising methods for containing and eradicating emerging vectors and parasites will provide many opportunities for advances in preventive medicine into the foreseeable future.

REFERENCES AND FURTHER READING

Beaglehole, R., Bonita, R., & Kjellstrom, T. (1993). *Basic epidemiology*. Geneva: World Health Organization.

Bruce-Chwatt, L. J. (1980). *Essential malariology*. London: William Heinemann Medical Books Ltd, 354 pp.

Davis, J. R., & Lederberg, J. (2001). *Emerging infectious diseases from the global to the local perspective. Workshop summary*. Washington, DC: National Academies Press, 134 pp.

Garrett-Jones, C. (1970). Problems of epidemiological entomology as applied to malariology. *Miscellaneous Publications of the Entomological Society of America, 7*, 168–178.

Gregg, M. B. (1988). Epidemiological principles applied to arbovirus diseases. In T. P. Monath (Ed.), *The Arboviruses: Epidemiology and ecology* (Vol. 1, pp. 292–309). Boca Raton, Florida: CRC Press.

Herms, W. B., & James, M. T. (1961). How arthropods cause and carry disease. In W. B. Herms' (Ed.), *Medical entomology* (5th ed., pp. 15–26). New York: Macmillan & Co.

Jawetz, E., Melnick, J. L., & Adleberg, E. A. (1972). Host-parasite relationships. In *Review of medical microbiology* (pp. 128–135). Los Altos, California: Lange Medical Publications.

Last, J. M. (Ed.). (1995). *A dictionary of epidemiology* (3rd ed.). Oxford University Press, 180 pp.

Macdonald, G. (1957). *The epidemiology and control of malaria*. Oxford University Press, 201 pp.

Moore, C. G., McLean, R. G., Mitchell, C. J., Nasci, R. S., Tsai, T. F., Calisher, C. H., et al. (1993). *Guidelines for arbovirus surveillance programs in the United States. US Dept Hlth Human Svcs, Centers for Disease Control and Prevention, Division of Vector Borne Infectious Diseases*. Colorado: Ft. Collins, 83 pp.

Rice, P. L., & Pratt, H. D. (1992). *Epidemiology and control of vector-borne diseases*. U. S. Dept. Hlth, Educ., & Welfare, Publ. Hlth Serv. Pub. No. (HMS0) 72-8245, 52 pp.

Smith, D. L., Perkins, T. A., Reiner, R. C., Jr., Barker, C. M., Niu, T., Chaves, L. F., et al. (2014). Recasting the theory of mosquito-borne pathogen transmission dynamics and control. *Transactions of the Royal Society of Tropical Medicine and Hygiene, 108*, 185–197.

Smith, D. L., Battle, K. E., Hay, S. I., Barker, C. M., Scott, T. W., & McKenzie, F. E. (2012). Ross, MacDonald, and a theory for the dynamics and control of mosquito-transmitted pathogens. *PLoS Pathogens, 8*. e1002588.

This page intentionally left blank

Chapter 5

Forensic Entomology

William L. Krinsky
Division of Entomology, Peabody Museum of Natural History, Yale University, New Haven, CT, United States

Forensic entomology is the study of insect biology as it relates to societal problems that come to the attention of the legal profession and that often must be resolved by legal proceedings. The term "forensic" (from the Greek "forum") refers to the public forum or courts of law. Forensic cases involving insects and other arthropods (e.g., mites, ticks, and spiders) that receive the most publicity are those involving unnatural deaths. In such cases, arthropods can be used to help date the time of death or determine whether a corpse has been moved after death. Forensic entomology, however, encompasses many other aspects of entomology and investigative purposes.

Because insects and their arthropod relatives are found in every environment inhabited by humans, the forensic entomologist may be consulted about problems that concern structural entomology, stored-product entomology, occupational hazards involving arthropods, veterinary and wildlife entomology, as well as those in which arthropods are associated with injuries, death, or criminal activity (medicolegal entomology). The latter aspects of forensic entomology are emphasized in this chapter. An entomologist who specializes in any of these areas may at times be consulted and act in the capacity of a forensic entomologist.

Traditionally, forensic entomology has not been a part of medical or veterinary entomology but rather a small specialty of forensic medicine. However, within the past 60 years, some medical entomologists have become specialists in forensic entomology because carrion insects feeding on corpses seen in many forensic cases are the same, or similar to, species that feed on blood and other tissues of living humans and that are typically studied by medical entomologists.

A forensic entomologist must undergo broad, intensive training in all aspects of insect biology, including anatomy, physiology, morphology, taxonomy, ecology, and developmental cycles. Such a background is essential because virtually any insect may at some time or place provide useful information for the investigation of a problem requiring legal intervention. Therefore, a forensic entomologist should be foremost an entomologist, with a specialization in forensics. Because forensic cases require the accurate identification of the arthropods involved, as well as a clear understanding of their developmental stages and life cycles, a forensic entomologist must have the knowledge to pursue such analyses and the background to understand the work of other entomological specialists. Because flies are usually the most important group of insects involved in medico-criminal forensic cases, an entomologist who provides information in such cases must have particular expertise in the Diptera. Familiarity with the species and habits of insects found in a given geographic area is essential for an analysis of a case in that region.

For many years in the United States, and more recently in Europe, entomological testimony has been admissible in criminal cases. The forensic entomologist must limit his or her analysis and testimony to the entomological evidence and base any conclusions solely on observations of insect biology. Other considerations related to suspects, motives, nonarthropod organisms, and other physical evidence are matters for experts in those areas of study. The rationale for the use of insects in forensic investigations is that these organisms can provide more objective evidence than that provided by witness testimony, the variable appearance of corpses that have been decomposing for longer than 24–48 h, and other forms of evidence lacking a scientific basis.

HISTORY

The earliest crime in which insects are known to have provided evidence occurred between 907 and 960 AD in China. Flies settling on the head of a deceased man indicated where a deadly blow had been made (Greenberg and Kunich, 2002). The first known case in which insects associated a murderer with his victim was described by Sung Tz'u, also in China, in 1247 (McKnight, 1981).

Medical and Veterinary Entomology. https://doi.org/10.1016/B978-0-12-814043-7.00005-4

A Chinese farmer was murdered by being slashed with a sickle. At an inquest, all of the farmers of the village were asked to place their sickles on the ground. Within a short time, flies, apparently attracted to residual blood, landed on only one of the 70 to 80 sickles assembled, incriminating its owner as the murderer.

Most of the history of forensic entomology deals with medico-criminal cases, especially homicides, rather than civil law proceedings such as liability cases in which remuneration is sought for damages to structures, commodities, animals, or humans. The forensic entomologist is usually asked to analyze arthropods associated with a corpse as a means of approximating the time since death (**postmortem interval [PMI]**) or, often more accurately, the postinfestation interval. The first recognition that insect development on a corpse could be useful in determining the interval from the time of death until discovery of the body occurred in Europe in the mid-19th century, and the first book to describe various arthropods infesting a dead body was published by Mégnin (1894).

One of the most famous English murder cases in which insects were used to help date the time of death involved the investigation of Dr. Buck Ruxton in 1935. The dismembered remains of Mrs. Ruxton and her nursemaid were found in a stream bed. The neat disarticulation of the bodies suggested that the murderer had some anatomical knowledge. Blow fly maggots (*Calliphora vicina*; formerly *C. erythrocephala*) found on the remains were third-instar larvae. This indicated that the remains must have been placed in the ravine 12−14 days before discovery, a time consistent with the disappearance of the victims from Dr. Ruxton's house. The fly evidence helped to convict Dr. Ruxton, who was found guilty and hanged.

Recent studies of carcasses and corpses have refined the analysis of the progression of arthropods associated with different stages of decomposition (Nuorteva, 1977; Smith, 1986; Campobasso et al., 2001). In 1990, a step-by-step procedural guide was published as a handbook for medico-criminal forensic entomologists (Catts and Haskell, 1990). Besides murder investigations, medico-criminal entomological proceedings may involve investigations of suicide, abuse or neglect, rape, and illicit drug trafficking (Catts and Goff, 1992; Benecke and Lessig, 2001).

LEGAL CASES INVOLVING LIABILITY

Structural Entomology

Forensic entomologists may be consulted when property evaluated by pest-management personnel and stated to be pest free, either before or after pest control treatment, begins to deteriorate as the result of the activity of wood−boring insects. The forensic entomologist is often asked to evaluate whether inspection of the now-damaged property and/or treatment was carried out properly.

Stored-Product Entomology

Most legal cases involving stored-product infestations rely on a forensic entomologist to provide information about the identification, origin, and destructive habits of the infesting arthropods.

Occupational Hazards Associated With Arthropods

Cases may involve personnel working in warehouses, factories, arthropod-rearing laboratories, and other facilities where insects and other arthropods or their parts, secretions, or excretions may be present. Individuals may become sensitized to these organisms or their associated materials and develop dermatological or respiratory problems. The latter can sometimes be life threatening. In all of these cases in which there is a question of liability, the potentially causative arthropods must be identified, and a forensic entomologist may be called on to explain the relationship between the arthropods and the experienced reaction or discomfort. Specific allergic and toxic problems are often associated with wasps (Vespidae) and bees (Apidae) attracted to food storage and processing facilities; paederine staphylinid beetles attracted to lights on oil rigs and buildings; dermestid, silvanid, and curculionid beetle infestations of stored products; and with various insects, such as bees, wasps, moths, mosquitoes, grasshoppers, locusts, cockroaches, bed bugs, and lady beetles in rearing facilities.

Veterinary and Wildlife Entomology

Legal cases involving insects and domestic animals often result from poor animal husbandry practices leading to uncontrolled populations of nuisance or pestiferous insects, such as house flies and stable flies. A forensic entomologist being consulted about such cases should have a thorough knowledge of arthropods of veterinary importance. One type of criminolegal case involving wildlife entomology is poaching. Arthropods feeding on carcasses of animals that have been shot or poisoned can be analyzed to help determine when and where the animals were killed, providing evidence that may be helpful in identifying a person guilty of illegal hunting or criminal mischief. Knowledge of the arthropods that infest living wild animals as well as those species that feed on carrion is necessary to discern which organisms may have been present when the animal was alive as opposed to those that colonized the body after death. Confusing these organisms can lead to erroneous estimates of the PMI.

LEGAL CASES INVOLVING HOMICIDES, SUSPICIOUS AND ACCIDENTAL DEATHS, AND ABUSE

Medicolegal entomology is the area of forensic entomology that involves the interpretation of events surrounding various kinds of injuries and deaths in which arthropods are associated with a body premortem or postmortem. Specimens and data collected by a forensic entomologist can be used to help determine the time since death, the geographical location at which the death occurred, the season of the year when the death occurred, movement or storage of a body after death, specific sites of trauma on a body, time of dismemberment, duration and intervals of submersion of a body, and presence of illicit drugs or other pharmaceuticals and to link suspects with the scene of a crime or with victims of murder, child neglect, or elder or sexual abuse (Catts and Goff, 1992; Benecke and Lessig, 2001; Campobasso and Introna, 2001). Identification of arthropods and knowledge of their defensive, feeding, and reproductive behaviors can help differentiate between premortem and postmortem wounds and can help to interpret the streaking, tracking, and deposition of blood, tissues, and bodily fluids present at a crime scene. Ant bites and roach feeding sites on corpses have been misinterpreted as premortem chemical burns. Blood regurgitated by flies and blood spots through which flies and roaches have crawled may confuse blood spatter patterns that can provide evidence of how a victim was assaulted or injured during an attack or fall.

Sudden Death With Arthropod Association

In cases in which sudden death has occurred and an arthropod has been found associated with the body or in the vicinity of the body, the possibility must be considered that death may have been caused by hypersensitivity to an arthropod bite or sting or as the result of an anaphylactic response to such an injury. In such cases, the forensic-medical entomologist must identify the arthropod and evaluate its potential to cause such a reaction. A definitive determination of the cause of death will rely on a careful analysis of the available entomological and medical information, with particular attention to evidence of allergic or unusual immune responses recorded in the medical history of the deceased.

Automobile-Accident Death

In some cases, a stinging insect (e.g., wasp or bee) or insect that resembles such an insect (e.g., wasp- or bee-mimicking fly) may be recovered from a vehicle in which a person has died. In these instances, the possibility must be raised that the driver panicked or was distracted by such an insect, resulting in an accident. Determining the responsible party in a fatal or injurious accident will have direct bearing on the legal outcome of such an occurrence.

Arthropods as Signs of Neglect or Abuse or as Agents of Murder

Several families of flies (e.g., Muscidae, Calliphoridae, Sarcophagidae) include carrion-feeding species that have larvae that may also invade living vertebrate tissue, causing myiasis. Myiasis seen in patients in hospitals and nursing facilities may indicate poor nursing care or neglect. Surgical dressings or bandages that are not frequently changed and untreated wounds or bedsores may attract female flies that oviposit or larviposit at these sites. Similarly, myiasis may occur in cases where flies are attracted to infants or small children who have not had their diapers regularly changed. In any of these situations, myiasis may be cited as a sign of neglect or abuse during legal proceedings. An entomologist in consultation with the medical personnel involved must investigate and determine the identity of the flies and analyze the circumstances under which the flies gained access to the infested individuals. Testimony by a forensic entomologist is often crucial in providing the background necessary to incriminate the party guilty of neglect or abuse.

Reports of the purposeful use of venomous arthropods as agents of abuse or murder are rare. There is one report of parents shutting up an infant in a room with wasps in an attempt to murder it. Powders made by grinding up dried toxic insects (e.g., blister beetles) have been used as poisons. As in other cases of abuse or death, the finding of a venomous arthropod or poison derived from an arthropod at the scene should at least suggest the possibility that the arthropod was directly related to the cause of injury or death.

Illicit Drug Transport, Use, and Overdose

Insects and mites of various kinds have been found associated with *Cannabis sativa* (marijuana or hemp). Identification of the arthropods found within shipments or seizures of marijuana and other illegal substances recovered by authorities can sometimes help pinpoint their origin. Insects recovered in such cases have included species of Diptera, Lepidoptera, Coleoptera, and Hymenoptera. Although many of the species found are cosmopolitan stored-product pests, others are known only from specific geographic regions and from particular ecological settings. In these cases, arthropods can provide evidence that helps incriminate drug traffickers. Diverse arthropods are inadvertently shipped around the world. More than 270 species of mites and insects have been recorded from shipments of hemp alone (Batra, 1976).

Human remains are often found in an advanced state of decay in which the tissues have largely decomposed. In these cases, where there may not be any available material for toxicological testing, the carrion-infesting arthropods or their remains (e.g., fly larval skins, dermestid exuviae, and frass) may provide material that can be assayed for various pharmaceuticals or toxins. Gas and liquid chromatography and mass spectrometry have been used successfully to identify various barbiturates as well as cocaine, heroin, and other controlled substances, and heavy metals, such as arsenic and mercury, in insect tissues associated with corpses. The results of qualitative analyses of the insects compared in some cases with analyses of tissues from the corpses have been consistent, but quantitative analyses have not been reliable. Until further studies are done, toxicological information from carrion insects as evidence of drug use, overdoses, or poisoning must be used with caution in legal proceedings (Introna et al., 2001; Lopes de Carvalho, 2010).

Suspicious Deaths

Arthropod evidence may be helpful in discerning whether transport of a corpse has occurred from the site where a suicide, abduction, attack, or murder has taken place. Careful attention to the species of arthropods found associated with a body might indicate that the species or types of arthropods are not consistent with the ecological setting where the body is found or that the species are not known from the geographic area where the body is discovered. Recovery of arthropods and arthropod parts from clothing, debris, and any containers or vehicles in which the body may have been transported can be helpful in discerning the initial location of the body. Arthropod remains found on the outside of a vehicle (e.g., on the windshield, radiator grill, or surface of a vehicle) can prove useful in this regard and may also provide evidence of where a suspect has traveled. In a multiple homicide case in California (USA), the prime suspect had rented a car in Ohio and claimed he had never driven it outside of the state. Insects that were recovered from the car radiator and air filter included the remains of a grasshopper, a paper wasp, and two true bugs, all species found only west of Kansas or only in far Western states. The insect evidence helped to convict the murderer (Kimsey, 2007).

Arthropod evidence can be used to link a suspect to a victim. Finding the same kind of arthropod at the site of a body as one associated with the clothing or vehicle of a potential suspect has helped to incriminate individuals in several murder cases. Arthropods as diverse as grasshoppers and mites have brought murderers to justice. In one case, part of a grasshopper leg was found on the corpse. Subsequently, the rest of the same grasshopper (matched by viewing the alignment of the broken leg with the damaged whole insect under a microscope) was found in the cuff of the pants of a potential suspect, placing him at the scene of the crime. In another case, during a murder investigation in California, chiggers (trombiculid mites) provided evidence to help convict a murderer. Investigators at the scene where the victim was found developed very itchy skin rashes, with spots on the ankles, waist, and buttocks. Similar lesions were seen on a suspect in the case. Further analysis and field study indicated that these lesions were caused by the bites of the chigger *Eutrombicula belkini*, a species found in very limited geographic areas, which included where the murder victim was discovered. The association of the chigger bites on the investigating team and the suspect and the presence of these rare mites at the scene in southern California helped incriminate the suspect. He was found guilty of first-degree murder and sentenced to life imprisonment (Webb et al., 1983).

In some cases, accumulations of maggots or other insects at particular anatomical sites have been used as indicators of trauma at those sites. Finding large numbers of maggots feeding on surfaces of the body other than at the common oviposition sites at body orifices may suggest the presence of wounds. However, insect evidence alone is not sufficient to conclude that such sites attractive to insects were created by trauma to the victim. The insect distribution may be used only to corroborate other evidence, such as pathological or physical evidence of stab wounds, cuts, gunshot wounds, or abrasions.

STAGES OF DECOMPOSITION

A body begins to decay at the moment of death and continues to decompose in a progression that will vary depending on the temperature and conditions to which the body is exposed. Cold or hot temperatures, low or high humidity, submergence in water or other fluids, burial, and access of insects, scavengers, and other animals to a corpse will affect the degree of decomposition at any given time and the rate of decay. Putrefaction starts at about 50°F and is most active between 70° and 100° F.

Mummification and **adipocere formation** are two somewhat unusual forms of decomposition occurring under specific environmental conditions. Mummification occurs when dehydration of the body begins before putrefactive changes in very dry, hot environments. The decay process is prevented or slowed by temperatures between 100° F and 212° F, at which fluids rapidly evaporate and mummification can occur. Mummified bodies have darkened, leathery skin (Fig. 5.5) that is not readily attractive to carrion insects. In cases where partially mummified corpses have been found that are infested with fly larvae, it has sometimes been assumed that vertebrate scavengers have disrupted or dismembered the bodies, allowing fly colonization to occur (Westerfield trial, 2002).

Saponification, the conversion of tissues to a yellowish-white waxy substance called adipocere, may occur if a body has been continuously exposed to moisture, either submerged or on a damp substrate. Bodies decay about twice as quickly in air as in water and about 8 times more quickly in air than when buried in earth. Although the decay process (excluding mummification and saponification) is a continuum, for the purpose of comparing different corpses in different environmental situations and as a means of standardizing postmortem analyses, five **stages of decomposition** have been defined that reflect the condition of the body and the arthropods most often found during those periods in the decay process. These stages are characterized by the progression: discoloration, bloating, liquefaction, skeletonization, and dry remains.

The fresh (first) stage of decay shows little or no signs of decomposition, but external color changes begin to appear. Internal changes are occurring as a result of bacterial and protozoal activity. Bloating (second stage) occurs with the breakdown of the intestinal tract releasing gas-forming, anaerobic bacteria that cause swelling of the corpse and the odors of decaying tissues. Active decay begins when the gas escapes and the body collapses. Active decomposition results from **autolysis** of tissues following the liberation of enzymes from cells and the action of bacteria and fungi growing on the remains. The skin begins to liquefy and has a darkened appearance, and the odor of decay is very strong (liquefaction). Advanced decay (skeletonization) is the period when the body is drying out; some flesh and hair may remain and fermentation occurs in anaerobic pockets of the body. The dry remains stage includes the slow decay of the remaining desiccated tissues, hair, teeth, and bones over the course of months or years. The duration of each stage varies with the environmental conditions to which a corpse is exposed. Body temperature can give an indication of the PMI within about the first 24 h. Some bodies reach a state of putrefaction within 48−72 h, which would, under other conditions, require 10−14 days to attain. Because of this variability, estimates of the PMI based on the degree of decomposition in cases where a body has been found more than 24 h since death are fraught with difficulty. Observations of insects, for which developmental times are known for given temperatures, provide more objective evidence for making such estimates.

INSECT SUCCESSION AND POSTMORTEM INTERVAL

The stages of decomposition also can be characterized by the **succession** of insects that inhabit the body at any given stage. Most insects of forensic importance in staging the degree of decomposition are species of flies, beetles, and moths (Table 5.1). Just as geographic and environmental conditions affect the rate of decomposition, the species and numbers of arthropods infesting a corpse also vary with climate and geography. Greater diversity is seen when a corpse is exposed in warm months or rainy seasons than in colder or dry seasons. In addition, any given species may be present at more than one stage of decomposition, depending on the condition of different parts of the body, and the presence of microhabitats on the body suitable for different developmental stages of a particular arthropod species. The rate of decomposition and the progression of different stages also are somewhat dependent on the changes brought about by the feeding and activity of arthropods at any given stage.

Typically, the first arthropods to arrive at a corpse, often within minutes, are blow flies (Calliphoridae) (Figs. 5.1 and 5.2). Gravid females feed on body secretions, especially about the eyes, nose, mouth, exposed anal and urogenital orifices, or open wounds. It is at these moist sites that they deposit their eggs (Fig. 5.2). An individual female may lay as many as 300 eggs. The eggs hatch within about 24 h under optimal conditions, and the first-instar larvae (maggots) begin to feed. The duration of each successive stage of the fly life cycle varies with the species, ambient temperature, and other environmental conditions. First-instar larvae molt to second instars, which in turn feed and molt to third-instar larvae; the latter feed for the longest of the three stages, typically leaving the corpse about 7−10 days after the eggs were deposited. At that time the larvae seek dry places in which to transform to puparia and subsequently emerge as adult flies (Fig. 5.3). Because blow flies are usually the first insects to arrive at a corpse, they often provide the most useful information about the elapsed time since a corpse was present in an environment in which insects had access. The durations of the developmental cycles of the forensically important blow flies have been determined under different laboratory conditions, allowing one to estimate the time interval between fly arrival and the recovery of a given developmental stage on a corpse.

Various other flies (e.g., house fly and latrine flies) that are attracted to feces or urine that may be associated with a decaying corpse arrive within the first few days. Predaceous insects and other arthropods (e.g., spiders and mites) that feed on blow fly eggs and larvae appear as decomposition continues. These include staphylinid, histerid, and silphid beetles (Fig. 5.4); ants and vespid wasps; and some muscid larvae, such as *Hydrotaea* species. These predaceous species persist, feeding on fly larvae and puparia as well as on the immature stages of each other in some cases. As the corpse dries out and the period of blow fly larval feeding ends, cheese skipper flies (Piophilidae), clerid beetles (*Necrobia rufipes*), sap beetles (Nitidulidae), and various larder (Dermestidae) and hide beetles (Trogidae) may arrive to feed on the dried skin, hair, and connective tissues.

TABLE 5.1 Insect Species Commonly Associated With Different Stages of Decomposition

Stage of Decomposition	Order	Family	Common Genera and Species
Fresh	Diptera	Calliphoridae (blow flies)	*Lucilia sericata,* L. *coeruleiviridis,** *Phormia regina,* *Cochliomyia macellaria,** *Calliphora vicina, C. vomitoria*
Bloated	Diptera	Calliphoridae (as above) plus:	
		Sarcophagidae (flesh flies)	*Sarcophaga haemorrhoidalis, S. bullata**
		Muscidae (house, latrine, and dump flies)	*Musca domestica, Fannia scalaris, Hydrotaea leucostoma*
	Coleoptera	Staphylinidae (rove beetles)	*Creophilus maxillosus, Platydracus* spp.
Active decay	Diptera	Calliphoridae, Sarcophagidae, and Muscidae (as above)	
	Coleoptera	Staphylinidae (as above) plus:	
		Silphidae (Carrion beetles)	*Necrophila americana,** *Nicrophorus* spp., *Oiceoptoma* spp.
Advanced decay	Coleoptera	Staphylinidae and Silphidae (as above) plus:	
		Histeridae (Hister beetles)	*Hister* spp., *Saprinus* spp.
	Diptera	Sepsidae (black scavenger flies)	*Sepsis* spp.
		Sphaeroceridae (small dung flies)	
		Scathophagidae (dung flies)	*Scathophaga* spp.
		Stratiomyidae (Soldier flies)	*Hermetia illucens*
		Phoridae (Scuttle flies)	*Megaselia scalaris*
Dry remains	Diptera	Piophilidae (Skipper flies)	*Piophila casei*
	Coleoptera	Cleridae (Checkered beetles)	*Necrobia rufipes*
		Nitidulidae (Sap beetles)	*Omosita* spp.
		Dermestidae (Larder and Carpet beetles)	*Dermestes* spp., *Anthrenus* spp., *Attagenus* spp.
		Trogidae (Hide beetles)	*Trox* spp.
	Lepidoptera	Pyralidae (Pyralid moths)	*Aglossa* spp.
		Tineidae (Clothes moths)	*Tinea pellionella, Tineola bisselliella, Trichophaga tapetzella*

All families listed are found worldwide, except Antarctica; species marked with an asterisk (*) are found only in the Western Hemisphere; all others have a wide global distribution.

FIGURE 5.1 Green blow fly, *Lucilia sericata* (Calliphoridae), adult. *Photograph by Nathan D. Burkett-Cadena.*

FIGURE 5.2 Green blow fly, *Lucilia sericata* (Calliphoridae), female with freshly deposited eggs on dead animal tissue; blue blow fly, *Calliphora vicina*, feeding at upper left. *Photograph by William L. Krinsky.*

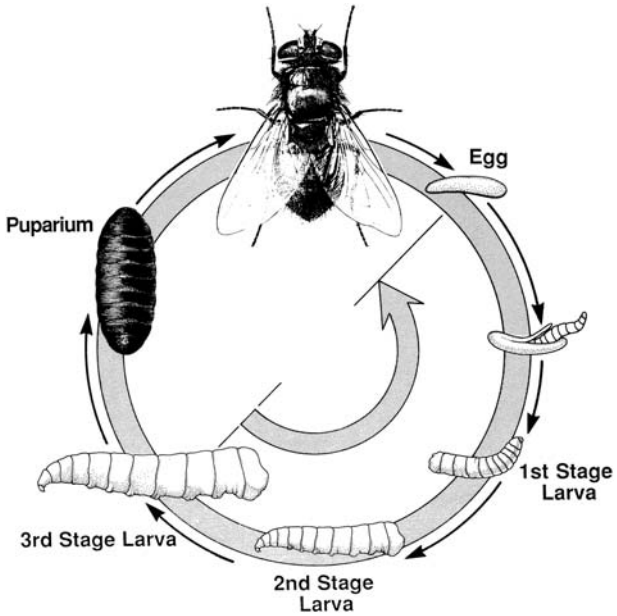


FIGURE 5.3 Blow-fly development. Life cycle showing developmental stages: egg, larva, pupa, and adult. *Original by William L. Krinsky.*

FIGURE 5.4 Carrion beetle, *Necrophila americana* (Silphidae), adult. *Photograph by Nathan D. Burkett-Cadena.*

A few species of moths (Pyralidae and Tineidae) can breed in the completely dried remains.

An entomological analysis of a crime scene is useful only if it can be determined why each of the diverse arthropods recovered was present at the given location and whether its presence was consistent with the environment in which it was found. Arthropods collected should be assigned to one of four species-group designations: necrophagous, predaceous or parasitic, omnivorous, and adventitious. An understanding of the interactions of these species with a corpse and with each other enables the forensic entomologist to make an estimate of the PMI (Campobasso et al., 2001).

Ideally, a forensic entomologist should be the one who collects any arthropod material at the scene of an investigation. This both ensures that the material will be properly preserved and maintained and allows the entomologist to make his or her own observations of the circumstances involved in the case. Often an entomologist must rely on specimens, records, and photographs provided by others. In such cases, the conclusions drawn must be tempered by the nature of the materials provided and the level of experience of the personnel who collected the evidence and recorded the information.

Analysis of the insect fauna on a corpse provides the basis for an estimate of a minimal PMI. It is a minimal estimate because there is no way to precisely determine when flies or other insects find a body or to know the time that elapsed between their arrival and the deposition of eggs (or larvae, in some cases) on the body. Blow flies are the insects most often used for determining an estimate of the minimal PMI. The interval between the arrival of flies on a

body and the discovery of the insects on a body is the **postcolonization interval** (= postinfestation interval). When a noninfested person dies in an environment where insects do not have immediate access to the body, the PMI will be longer than the postcolonization interval. Alternatively, if the victim is infested with flies (or other insects) before death (e.g., myiasis in a conscious, comatose, or paralyzed individual), the postcolonization interval will be longer than the PMI. In the absence of both pathological evidence of myiasis and physical or environmental evidence of insects being excluded from the corpse for some period postmortem, the postcolonization interval is regarded as an estimate of the minimal PMI.

Determination of the precolonization interval is the most difficult because of a lack of entomological evidence. Consequently, disputes in homicide cases often relate to the degree to which the minimal PMI estimate takes into account the factors that may have affected the length of time between death and the discovery of a body by insects. Information that is needed to better assess the precolonization interval includes identification of the sensory mechanisms responsible for attracting the insects to a corpse, identification of the chemical clues that are the attractants, and a better understanding of the insect behavior involved.

Bodies discovered within a few weeks after death as determined by the presence of entomological specimens consistent with early infestation enable the entomologist to make a more accurate estimation of a minimal PMI than in cases in which insect succession is more advanced. In the latter case, where a body is found on which blow fly development has ceased, insects active at this later stage of decomposition can be identified and compared with the succession of insects known to infest corpses. Living insects as well as insect remains (exuviae, shed puparial or pupal cases, frass, or dead insects) that can be identified can help to develop a timeline of insect infestation.

Establishing how long insects have been developing on a body requires climatic data from the site at which a body was discovered, as well as identification of the infesting insects and their developmental stages. The development of blow flies, as for most plants and other insects, is dependent on temperature, with increased temperature (below lethal values) directly related to increased growth. In blow flies, as in plants, assuming other conditions (humidity, light, etc.) are suitable for growth, by summing the number of degrees of temperature over time, it is possible to correlate a total number (**accumulated degree hours [ADH]**, degrees × hours) with a given stage of development. ADH are calculated to determine the minimal PMI. The ADH values needed for development of the egg and development to each larval stage, puparium, and the adult of most of the forensically important blow flies have been determined by rearing the flies at different temperatures in the laboratory. Identification of the species and developmental stages of blow flies found on a corpse enables a comparison of the latest stages found with known information about how much time had to have elapsed at different temperatures for these stages (egg, particular larval instar, or puparium) to have developed. By obtaining hourly temperature readings that most closely represent the temperatures that occurred over a period of days at the site where a body is found, one can add the ADH that occurred from the collection of specimens backward in time (Fig. 5.3, large counterclockwise arrow) to determine how many hours must have elapsed since the first fly eggs were laid on the body.

The PMI has been estimated in numerous murder cases based on the determination of the ages of eggs, larvae, or puparia found associated with a victim. Larvae have been used most often for this determination (Fig. 5.5). In one case, more than 4,000 maggots of *Phormia regina* were recovered from a body wrapped in a piece of carpet and dumped along an interstate highway. By correlating climatic conditions with the ages of the larvae and the timing of emergence of adult flies from the oldest larvae collected, a PMI estimate of about 7 days was made, which was within 1 day of when the victim was last seen alive (National Library of Medicine; entomology case study).

The use of ADH is one method for estimating a minimal PMI. Other approaches which include ecological modeling and multivariate analysis, have been proposed to take into account many of the variables that affect the development of corpse-infesting insects (Faris et al., 2016; van der Ham, 2016). Many factors may interfere with the time of arrival of insects on a body, the nature of the insect stages found on a body, and the assumed linear correlation of temperature and insect development. Factors affecting the microclimate on a corpse, such as insulating materials (clothing, wrappings; position of a body in a building, container, or vehicle) and position of a body in relation to vegetation or

FIGURE 5.5 Partially mummified corpse infested with blow fly maggots. This homicide victim was found and placed in the body bag within 7 days of the murder. *Courtesy of William L. Krinsky.*

overshadowing materials, must be considered. In cases where there is evidence that the victim was feverish, chilled, or even frozen before death or before being placed in a location where insects would have access to the body, estimates of the time required for development of the insects found must be adjusted to accommodate these differences. The developing maggots themselves when aggregated into a maggot mass at one site on a body may markedly raise the temperature at which they are feeding and growing compared with the ambient temperature.

Disruption or dismemberment of a body and predation of necrophagous insects by vertebrate scavengers; changes in the rate of insect development caused by insect ingestion of illicit drugs from tissues of the corpse; and changes in microclimatic conditions at the discovery site (e.g., wind, precipitation) or on the corpse itself related to the number and location of maggots (Slone and Gruner, 2007) must all be considered when an estimate of PMI is made based on insect development.

With recent interest in improving the methods by which all forensic evidence is obtained to ensure that they are reproducible, testable, and verifiable by error analysis, more emphasis is being placed on evaluating the precision, accuracy, and bias of forensic entomology methods (Ieno et al., 2010; Villet et al., 2010). In addition, more consideration is being given to recognition of variation within a species and between populations of forensically important species (Picard and Wells, 2009). Quantitative genetics, population genetics, functional genomics, and other quantitative disciplines that have been applied to ecology and epidemiology need to be used more frequently in forensic entomology studies (Tomberlin et al., 2011).

Traditional morphological methods for identifying forensically important species and for aging specimens are now being refined with chemical, radiographic, and genetic analyses. Cuticular hydrocarbon profiles enable identification of species, as well as age determination of larvae and adults (Moore et al., 2014; Pechal et al., 2014). Detailed examination of puparia with microcomputed tomography can now provide more accurate and graphic information than dissection and histological examination for determining the age of puparia based on developmental changes in the pupa (Martin-Vega et al., 2016). Genomic analyses have been developed for species identification of adult flies and for the identification and aging of larvae (Picard et al., 2012). Refinement of the techniques should allow more rapid identification of species collected at crime scenes and other sites of forensic interest. Additional uses of DNA analysis include specific identification of blood meals from hematophagous insects collected at a crime scene and of semen in the alimentary tract of necrophagous insects that might link a victim or a scene to a specific suspect (Amendt, Zehner et al., 2010).

As the methodology in forensic entomology becomes more sophisticated and the interpretation of entomological events following death becomes more precise, entomology should become even more important in criminal investigations. Forensic entomology will continue to provide a different and valuable approach to the investigative process that is a collaborative effort of law enforcement personnel, forensic pathologists, and other forensic specialists.

REFERENCES AND FURTHER READING

Amendt, J., Campobasso, C. P., Goff, M. L., & Grassberger, M. (Eds.). (2010). *Current concepts in forensic entomology*. Dordrecht: Springer.

Amendt, J., Richards, C. S., Campobasso, C. P., Zehner, R., & Hall, M. J. R. (2011). Forensic entomology: Applications and limitations. *Forensic Science, Medicine and Pathology, 7*, 379–392.

Amendt, J., Zehner, R., Johnson, D. G., & Wells, J. W. (2010). Future trends in forensic entomology. In J. Amendt, C. P. Campobasso, M. L. Goff, & M. Grassberger (Eds.), *Current concepts in forensic entomology* (pp. 353–368). Dordrecht: Springer.

Batra, S. W. T. (1976). Some insects associated with hemp or marijuana (*Cannabis sativa* L.) in northern India. *Journal of the Kansas Entomological Society, 49*, 385–388.

Benecke, M. (2001). A brief history of forensic entomology. *Forensic Science International, 120*, 2–14.

Benecke, M., & Lessig, R. (2001). Child neglect and forensic entomology. *Forensic Science International, 120*, 155–159.

Byrd, J. H., & Castner, J. L. (2010). *Forensic Entomology: Utility of arthropods in legal investigations* (2nd ed.). Boca Raton: CRC Press.

Campobasso, C. P., Di Vella, G., & Introna, F. (2001). Factors affecting decomposition and Diptera colonization. *Forensic Science International, 120*, 18–27.

Campobasso, C. P., & Introna, F. (2001). The forensic entomologist in the context of the forensic pathologist's role. *Forensic Science International, 120*, 132–139.

Catts, E. P., & Goff, M. L. (1992). Forensic entomology in criminal investigations. *Annual Review of Entomology, 37*, 253–272.

Catts, E. P., & Haskell, N. H. (Eds.). (1990). *Entomology & death: A procedural guide*. Clemson: SC: Joyce's Print Shop.

Dekeirsschieter, J., Frederickx, C., Verheggen, F. J., Boxho, P., & Haubruge, E. (2013). Forensic entomology investigations from Doctor Marcel Leclercq (1924–2008): A review of cases from 1969 to 2005. *Journal of Medical Entomology, 50*, 935–954.

Erzinçlioğlu, Y. Z. (2000). *Maggots, murder and men: Memories and reflections of a forensic entomologist*. Colchester, UK: Harley.

Faris, A. M., Wang, H.-H., Tarone, A. M., & Grant, W. E. (2016). Forensic entomology: Evaluating uncertainty associated with postmortem interval (PMI) estimates with ecological models. *Journal of Medical Entomology, 53*, 1117–1130.

Greenberg, B. (1991). Flies as forensic indicators. *Journal of Medical Entomology, 28*, 565–577.

Greenberg, B., & Kunich, J. C. (2002). *Entomology and the Law: Flies as forensic indicators*. Cambridge Univ. Press.

Hart, A. J., & Whitaker, A. P. (2006). Forensic entomology: Insect activity and its role in the decomposition of human cadavers. *Antenna, Bulletin of the Royal Entomological Society, 30*, 159–164.

Ieno, E. N., Amendt, J., Fremdt, H., Saveliev, A. A., & Zuur, A. F. (2010). Analysing forensic entomology data using additive mixed effects moddelling (*sic*). In J. Amendt, C. P. Campobasso, M. L. Goff, & M. Grassberger (Eds.), *Current concepts in forensic entomology* (pp. 139−162). Dordrecht: Springer.

Introna, F., Campobasso, C. P., & Goff, M. L. (2001). Entomotoxicology. *Forensic Science International, 120*, 42−47.

Kimsey, L. (Wednesday 4, July 2007). The case of the Red-Shanked grasshopper. *Los Angeles Times*, A21.

LeClercq, M. (1969). *Entomological parasitology*. Oxford: Pergamon Press.

Lopes de Carvalho, L. M. (2010). Toxicology and forensic entomology. In J. Amendt, C. P. Campobasso, M. L. Goff, & M. Grassberger (Eds.), *Current concepts in forensic entomology* (pp. 163−178). Dordrecht: Springer.

Magni, P. A., Voss, S. C., Testi, R., Borrini, M., & Dadour, I. R. (2015). A biological and procedural review of forensically significant *Dermestes* species (Coleoptera: Dermestidae). *Journal of Medical Entomology, 52*, 755−769.

Martin-Vega, D., Hall, M. J. R., & Simonsen, T. J. (2016). Resolving confusion in the use of concepts and terminology in intrapuparial development studies of cyclorrhaphous Diptera. *Journal of Medical Entomology, 53*, 1249−1251.

McKnight (translation of Sung Tz'u), B. E. (1981). *The Washing away of wrongs: Forensic medicine in Thirteenth century China*. Ann Arbor: Center for Chinese Studies, University of Michigan.

Mégnin, P. (1894). *La Faune des Cadavres. Encyclopédie scientifique des Aide-Mémoire, No. 101B*. Paris: Gautier-Villars *et fils*.

Moore, H. E., Adam, C. D., & Drijfhout, F. P. (2014). Identifying 1st instar larvae for three forensically important blowfly species using "fingerprint" cuticular hydrocarbon analysis. *Forensic Science International, 240*, 48−53.

National Library of Medicine. Forensic views of the body - Entomology case study. http://www.nlm.nih.gov/exhibition/visibleproofs/galleries/cases/insect.html.

Nuorteva, P. (1977). Sarcosaprophagous insects as forensic indicators. In C. G. Tedeschi, W. G. Eckert, & L. G. Tedeschi (Eds.), *Forensic medicine: A study in trauma and environmental hazards* (Vol. 2, pp. 1072−1095). Philadelphia: W. B. Saunders Co.

Paula, M. C., Morishita, G. M., Cavarson, C. H., Goncalves, C. R., Tavares, P. R. A., Mendonca, A., et al. (2016). Action of ants on vertebrate carcasses and blow flies (Calliphoridae). *Journal of Medical Entomology, 53*, 1283−1291.

Pechal, J. L., Moore, H., Drijfhout, F., & Benbow, M. E. (2014). Hydrocarbon profiles throughout adult Calliphoridae aging: A promising tool for forensic entomology. *Forensic Science International, 245*, 65−71.

Picard, C. J., Johnston, J. S., & Tarone, A. M. (2012). Genome sizes of forensically relevant Diptera. *Journal of Medical Entomology, 49*, 192−197.

Picard, C. J., & Wells, J. D. (2009). Survey of genetic diversity of *Phormia regina* (Diptera: Calliphoridae) using amplified fragment length polymorphisms. *Journal of Medical Entomology, 46*, 664−670.

Rivers, D. B., Ciarlo, T., Spelman, M., & Brogan, R. (2010). Changes in development and heat shock protein expression in two species of flies (*Sarcophaga bullata* [Diptera: Sarcophagidae] and *Protophormia terraenovae* [Diptera: Calliphoridae] reared in different sized maggot masses. *Journal of Medical Entomology, 47*, 677−689.

Sachs, J. S. (2001). *Corpse − nature, forensics, and the struggle to pinpoint time of death*. Cambridge, MA: Perseus Publishing.

Slone, D. H., & Gruner, S. V. (2007). Thermoregulation in larval aggregations of carrion-feeding blow flies (Diptera: Calliphoridae). *Journal of Medical Entomology, 44*, 516−523.

Smith, K. G. V. (1986). *A manual of forensic entomology*. Ithaca, NY: British Museum (Natural History) and Cornell Univ. Press.

Souza, C. M., Thyssen, P. J., & Linhares, A. X. (2011). Effect of nandrolone decanoate on the development of three species of *Chrysomya* (Diptera: Calliphoridae), flies of forensic importance in Brazil. *Journal of Medical Entomology, 48*, 111−117.

Tarone, A. M., Picard, C. J., Spiegelman, C., & Foran, D. R. (2011). Population and temperature effects on *Lucilia sericata* (Diptera: Calliphoridae) body size and minimum development time. *Journal of Medical Entomology, 48*, 1062−1068.

Tomberlin, J. K., & Benbow, M. E. (Eds.). (2015). *Forensic entomology: International dimensions and frontiers*. Boca Raton: CRC Press.

Tomberlin, J. K., Mohr, R., Benbow, M. E., Tarone, A. M., & VanLaerhoven, S. (2011). A roadmap for bridging basic and applied research in forensic entomology. *Annual Review of Entomology, 56*, 401−421.

Van der Ham, J. (2016). Permutation tests of hierarchical cluster analyses of carrion communitiens and ter potential use in forensic entomology. *Journal of Medical Entomology, 53*, 1238−1241.

Villet, M. H., Richards, C. S., & Midgley, J. M. (2010). Contemporary precision, bias and accuracy of minimum post-mortem intervals estimated using development of carrion-feeding insects. In J. Amendt, Campobasso, M. L. Goff, & M. Grassberger (Eds.), *Current concepts in forensic entomology* (pp. 109−137). Dordrecht: Springer.

Vincent, C., Kevan, D. K. M. E., Leclercq, M., & Meek, C. L. (1985). A bibliography of forensic entomology. *Journal of Medical Entomology, 22*, 212−219.

Webb, J. P., Jr., Loomis, R. B., Madon, M. B., Bennett, S. G., & Greene, G. E. (1983). The chigger species *Eutrombicula belkini* Gould (Acari: Trombiculidae) as a forensic tool in a homicide investigation in Ventura County, California. *Bulletin of the Society of Vector Ecologists, 8*, 141−146.

Westerfield trial. (2002). (Transcripts) legacy.sandiegouniontribune.com/news/metro/danielle/transcripts.html.

Whitworth, T. (2006). Keys to the genera and species of blow flies (Diptera: Calliphoridae) of America north of Mexico. *Proceedings of the Entomological Society of Washington, 108*, 689−725.

Chapter 6

Cockroaches (Blattaria)

Richard J. Brenner[1,2] and Richard D. Kramer[3]

[1]Director, ATIP Foundation, Arlington, TX, United States; [2]Formerly Assistant Administrator of Technology Transfer, USDA, Agricultural Research Service, Beltsville, MD, United States; [3]Board Certified Entomologist (BCE), CEO Innovative Pest Management, Inc., Brookeville, MD, United States

Cockroaches are among the oldest and most primitive of insects. Earlier studies had concluded that they evolved about 350 million years ago (Mya) during the Silurian Period, diverging together with the mantids from an ancestral stock that also gave rise to termites (Boudreaux, 1979). More recently, researchers have scrutinized the phylogeny of Dictyoptera (800 taxa, 10,000 molecular characters, combined with controlled fossil evidence) and have determined the most recent common ancestors of cockroaches and termites would date back to the Permian, about 275 Mya (Legendre et al., 2015). Cockroaches are recognized as the order Blattaria or Blattodea. Although the majority of species are feral and not directly associated with people, a few species have evolved in proximity to human habitations where they have adapted to indoor environments. Their omnivorous feeding behavior, facilitated by their unspecialized chewing mouthparts, has contributed to a close physical relationship between cockroach populations and humans, with resultant chronic exposure of humans to these pests.

The presence of some species in the home and commercial kitchens (e.g., German and brown-banded cockroaches) often is an indicator of poor sanitation or substandard housekeeping. Although they are primarily nuisance pests, their presence can have important health implications. Cockroaches are generalists that feed on virtually any organic substance grown, manufactured, stored, excreted, or discarded by humans. Consequently, food supplies and preparation surfaces are at risk of contamination by pathogens associated with cockroaches. Because species that infest structures typically have high reproductive rates, humans commonly are exposed to high levels of potentially allergenic proteins associated with cockroaches, which can lead to significant respiratory ailments. Cockroaches also can serve as intermediate hosts of parasites that debilitate domestic animals.

TAXONOMY

There are about 4,400 species of cockroaches worldwide, which are assigned to more than 500 genera. About 70 species occur in the United States, 24 of which have been introduced from other parts of the world. According to Atkinson et al. (1991), 17 of these species are pests of varying degrees. There are five cockroach families, three of which include most of the pest species: **Blattidae**, **Blattellidae**, and **Blaberidae**. Species in the family **Cryptocercidae** are unusual in that they have gut symbionts similar to those found in termites, and they live in family groups in decaying logs. Members of the family **Polyphagidae** include those dwelling in arid regions where they are capable of moving rapidly through sand. Species in these two families are rarely pests. The family Blattidae includes relatively large cockroaches that are the most common peridomestic pests throughout much of the world. Blattellid cockroaches range in length from less than 25 mm (e.g., *Supella* and *Blattella*) to 35–40 mm (e.g., *Periplaneta* and *Parcoblatta* spp.). *Parcoblatta* species are feral, occasionally invading homes but seldom reproducing indoors. Blaberid cockroaches range greatly in size and include some of the more unusual species such as the Cuban cockroach, which is green as an adult, and the Surinam cockroach, which is parthenogenetic in North America. Nearly all of the blaberids that occur in the United States are restricted to subtropical regions and have minor medical or veterinary significance. Taxonomic keys for adults are provided by McKittrick (1964), Cornwell (1968), Roth (1985), and Helfer (1987). A pictorial key for identifying the egg cases of common cockroaches is provided by Scott and Borom (1964).

MORPHOLOGY

Cockroaches have retained their basic ancestral form. The Blattodea includes cockroaches and termites. Cockroaches are distinguished from termites and other insect orders by

Medical and Veterinary Entomology. https://doi.org/10.1016/B978-0-12-814043-7.00006-6

morphological characters associated with wing size and venation, biting-chewing mouthparts, and prominent cerci. They differ from other orthopteroid insects by having hind femora that are not enlarged, cerci typically with eight or more segments, a body that is dorsoventrally flattened and generally ovoid, and a head that is largely concealed from above by a relatively large pronotum.

An indicator of cockroach infestations is their egg cases, or **oothecae** (sing. ootheca), which can be useful in differentiating species infesting buildings. Most cockroach oothecae persist in the environment after the nymphs have emerged, providing a history of infestation.

These purse-shaped capsules typically contain 5 to 40 embryos (Fig. 6.1), and their coloration ranges from light brown to chestnut brown depending on the degree of sclerotization. A keel that runs the anterior length of the ootheca permits transport of water and air to the developing embryos. Each embryo is contained in a separate compartment that may or may not be obvious externally. In some species (e.g., German and brown-banded cockroaches) lateral, anterior-to-posterior indentations denote the individual developing embryos. Others have only weak lateral indentations (e.g., brown and smokybrown cockroaches), and still others have no lateral indentations but differ in their symmetry (e.g., Oriental, American, and Australian cockroaches).

The mouthparts of cockroach nymphs and adults (Fig. 2.2A) are characterized by strongly toothed mandibles for biting and chewing. Maxillary and labial palps are well developed with five and three segments, respectively. Antennae are long and whiplike, originate directly below the middle of the compound eyes, and consist of numerous small segments. The arrangement of three ocelli near the antennal sockets is variable: they are well developed in winged species (macropterous) but rudimentary or lacking in species with reduced wings (brachypterous) or those lacking wings altogether (apterous).

Adults generally have two pairs of wings that are folded fanwise at rest. The front wings, called **tegmina** (sing. tegmen), are typically hardened and translucent with well-defined veins. The hindwings are membranous and larger. In some species, such as the wood cockroaches (e.g., *Parcoblatta* sp.), females are brachypterous and incapable of flight, whereas males are macropterous. Other species, such as the Florida woods cockroach (*Eurycotis floridana*), have only vestigal wing buds as adult males and females. In cockroaches, all three pairs of legs are well developed, with large coxae and slender, long segments that aid in the rapid running characteristic of these insects. Each femur has two longitudinal keels that typically are armed with spines. The tibiae are often heavily spined and are used for defense against predators. Each tarsus consists of five segments with a pair of claws and may bear a padlike arolium

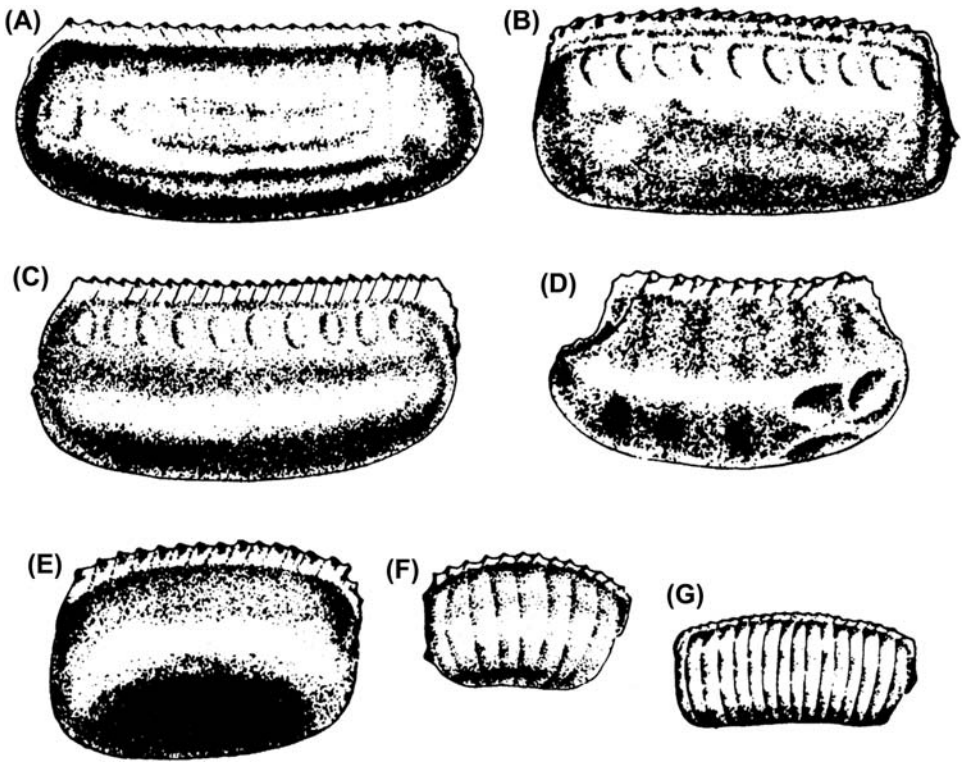


FIGURE 6.1 Cockroach oothecae (egg cases). (A) Australian cockroach (*Periplaneta australasiae*); (B) brown cockroach (*P. brunnea*); (C) smokybrown cockroach (*P. fuliginosa*); (D) Oriental cockroach (*Blatta orientalis*); (E), American cockroach (*P. americana*); (F) brown-banded cockroach (*Supella longipalpa*); (G) German cockroach (*Blattella germanica*). *Courtesy of U.S. Public Health Service.*

FIGURE 6.2 Developmental stages of cockroaches, represented by *Periplaneta brunnea*. Left to right: first, second, third, and fourth nymphal instars; adult female, adult male. *Courtesy of Daniel R. Suiter.*

that aids in walking on smooth surfaces. Ventral pads, or pulvilli, are present on tarsomeres 1–4. A pair of caudal cerci have small ventral hairs that are sensitive to vibrations caused by low-frequency sound and air movement; their stimulation initiates an escape response.

The posterior end of the abdomen of some nymphs and all males bears a pair of **styli** (sing. stylus) between the cerci, arising from the sternum of the ninth abdominal segment. In winged species, the styli may be apparent only when viewed ventrally. The structure of the styli serves to distinguish males from females. Generally the males also can be recognized by their more slender bodies, with laterally tapered and dorsally flattened external genitalia (terminalia). The terminalia of the more robust females are notably broader than in males and bear a conspicuous subgenital plate that is rounded or keel-like when viewed ventrally. Associated with this plate is a relatively large genital chamber (=genital pouch) in which the ootheca develops. For a more detailed description of cockroach genitalia, see McKittrick (1964) or Cornwell (1968). Nymphal stages are similar in appearance to adults but lack wings, have incompletely developed genitalia, and may vary markedly in color from the adult.

LIFE HISTORY

Cockroaches are paurometabolous insects. The immatures generally are similar in appearance to the adults except for their undeveloped sexual organs and lack of fully developed wings (Fig. 6.2). Reproduction in cockroaches is typically sexual, although parthenogenesis is reported in a few species. Comparative life-history data for some of the more common cockroach pests are provided in Table 6.1.

In cockroaches, embryogenesis and oviposition occur in one of three ways. Most species are oviparous, including all *Periplaneta* species and the Oriental and brown-banded cockroaches. Eggs of oviparous species are protected inside a thick-walled, impermeable ootheca that is deposited soon after it is formed. Embryonic development occurs external to the female. The German cockroach is oviparous, but the female carries the ootheca protruding from the genital chamber until just hours before hatching occurs. The ootheca is softer than in *Periplaneta* sp., allowing uptake of water and nutrients from the genital pouch. Females of a few cockroaches, such as *Blaberus* sp. and the Surinam cockroach, produce an ootheca that is extruded, rotated, and then retracted into the genital pouch. The eggs are incubated internally until hatching. *Diploptera punctata*, a pest species in Hawaii, is the only cockroach in which the eggs hatch while still in the genital pouch. Embryogenesis takes 1–8 weeks, depending on the species.

The number of nymphal instars varies from 5 to 13, depending on the species, nutritional sources, and

TABLE 6.1 Life Histories of Selected Common Species of Cockroaches, Showing the High Degree of Variability Within Species Due to Environmental Temperatures and Nutritional Availability

Cockroach	Number of Eggs/ Ootheca	Number of Nymphal Instars	Developmental Time (days)	Embryonic Development
German	30–40	5–7	103	Internal/extruded
Asian	38–44	5–7	52–80	Internal/extruded
Brown-banded	14–18	6–8	90–276	External
American	12–16	10–13	168–700	External
Smokybrown	20	9–12	160–716	External
Australian	24	10–12	238–405	External
Oriental	16	7–10	206–800	External
Surinam	26	8–10	127–184	Internal

microclimate. Development of pestiferous species through the nymphal stadia requires 6–7 weeks for German cockroaches to longer than 1 year for *Periplaneta* species and other larger cockroaches. Typically, the nymphs exhibit strong aggregation tendencies, governed largely by aggregation pheromones. These pheromones act as locomotory inhibitors; when cockroaches perceive the pheromone, they become relatively stationary. Studies of various species have shown that development to the adult stage is quicker when nymphs are reared in groups rather than in isolation. However, aggregation does have a biological cost; those reared in groups typically are smaller and cannibalism may occur. Longevity of cockroaches varies from several weeks to longer than 1 year.

BEHAVIOR AND ECOLOGY

Mating in cockroaches generally is preceded by courtship behavior facilitated by sex pheromones. In some species a blend of volatile compounds is produced by virgin females to attract and orient males (e.g., *Periplaneta* sp. and the brown-banded cockroach). In the German cockroach, the sex pheromone is a blend of nonvolatile and volatile cuticular components that elicits courtship by males following palpation of the female's integument by the male's antennae. Once courtship is initiated in the male, he turns away from the female and raises his wings to expose dorsal **tergal glands**; the female feeds on pheromones from these glands as the male grasps her genitalia with his pair of caudal claspers. Most species copulate in an end-to-end position. During the hour or so that follows, a spermatophore is formed and passed from the male into the genital chamber of the female. Only about 20% of females mate again after the first gonotrophic cycle.

Cockroaches can be categorized ecologically as domestic, peridomestic, or feral. **Domestic** species live almost exclusively indoors and are largely dependent on humans for resources (food, water, and harborage) for survival. They rarely are able to maintain themselves outdoors. Although this group contains the smallest number of species, it presents the greatest concern to human health. Domestic species include the German and brown-banded cockroaches. **Peridomestic** species are those that survive in or around human habitation. Although they do not require humans for their survival, they are adept at exploiting the amenities of civilization. This group is represented by American, Australian, brown, and smokybrown cockroaches (all *Periplaneta* sp.), the oriental cockroach, and the Florida woods cockroach. **Feral** species are those in which survival is independent of humans. This group includes more than 95% of all species in the world. Only a few occur indoors as occasional and inadvertent invaders that typically do not survive in a domestic environment. They are of little or no medical importance, although recent articles suggest that using these species as food sources (Asia) does pose risk to immunologically compromised individuals, as well as to those who are immunocompetent (Zhang et al., 2011).

Cockroach behavior and survival are strongly influenced by their need for food, water, and safe harborage from potential predators and detrimental microclimates. They are omnivorous and will consume virtually any organic matter, including fresh and processed foods, stored products, and even book bindings and pastes on stamps and wallpaper when more typical foodstuffs are not available. Cockroaches have the same general problems with water balance as do other terrestrial arthropods. Their relatively small size results in a high surface area-to-volume ratio and a high risk of losing water through respiration, oral and anal routes, or the cuticle. Temperature, air flow, relative humidity, and availability of liquid water greatly affect water regulation.

As a result of these physiological considerations, physical constraints of the environment usually determine habitat preferences of cockroaches in and around structures. Oriental, Turkestan, and American cockroaches, for example, require high moisture and occur in damp terrestrial environments such as septic tanks and municipal sewer systems. Brown, smokybrown, and Florida woods cockroaches occur in a wider range of habitats associated with trees, wood and leaf piles, wall voids, and foundation blocks of buildings. Brown-banded cockroaches are more tolerant of drier conditions and commonly occur in kitchens, pantries, and bedrooms. German cockroaches occupy harborages near food and water. Consequently, they are found primarily in kitchens and pantries and secondarily in bathrooms when their populations are high. In mixed populations of German and brown-banded cockroaches, the German cockroach tends to outcompete the brown-banded cockroach within 9 months.

Cockroaches are adept crawlers and are capable of rapid movement even across windows and ceilings. Flight ability varies with species. Some are incapable of flight except for crude, downward gliding used as an escape behavior. Others are weak fliers, occasionally seen flying indoors when disturbed. Still others are relatively strong fliers that are particularly active at sunset when they may be attracted indoors by lights and brightly lit surfaces. Attraction to light is especially common in the Asian, Surinam, and Cuban cockroaches and in many of the wood cockroaches (*Parcoblatta* sp.).

Pestiferous cockroaches that occur indoors are typically nocturnal and tend to avoid lighted areas. This enables them to increase their numbers and become established in structures before human occupants even become aware of their presence.

COMMON COCKROACH SPECIES

The following cockroach species are commonly encountered in and around structures in the United States and are the ones most frequently brought to the attention of medical entomologists.

Oriental Cockroach *(Blatta orientalis)*

This peridomestic cockroach (Fig. 6.3) is believed to have originated in northern Africa and from there spread to Europe and western Asia, South America, and North America. It is a relatively lethargic species that prefers cooler temperatures than the German cockroach and is primarily a concern in temperate regions of the world. They are now considered major U.S. household pests in parts of the Northwest, Midwest, and southern states (McCanless, 2000, 2014, 2017). Adults are black and 25−33 mm long. Males are short-winged but do not fly, and females are brachypterous. Their tarsi lack aroliar pads, precluding this cockroach from climbing on smooth vertical surfaces. Oothecae are 8−10 mm long, each typically containing 16 eggs. Also commonly known as **waterbug** and **shad roach**, this species is usually associated with damp or wet conditions, such as those found in decaying wood, heavy ground cover (e.g., ivy), water-meter boxes, floor drains, sump pumps, and the lower levels of structures. It infests garbage chutes of apartment complexes, sometimes reaching upper floors. Development is slow compared with that of most other species, typically requiring about 1 year or longer depending on temperature conditions. Adults may live for many months. Mobility is fairly restricted, making control easier than for most other species. This species is rarely seen during the daytime.

FIGURE 6.3 Oriental cockroach (*Blatta orientalis*), male. *Courtesy of University of Florida/IFAS.*

Turkestan Cockroach *(Blatta lateralis)*

This peridomestic cockroach (Fig. 6.4) originated in North Africa, the Middle East, and Asia. It was inadvertently introduced to the United States likely through transport of military goods and equipment from the Middle East by U.S. military personnel in the late 1970s in Lathrope, CA, El Paso, TX (1980), Scottsdale and Tucson, AZ (1984), and Fort McPherson, GA (2005). Brisk internet sales as food for reptiles also makes further spread of this invasive species more probable (Kim and Rust, 2013). Compared with the Oriental cockroach, a shorter developmental period (ca. 50% less), greater longevity, and an unusually large number of oothecae (twice that of the Oriental cockroach) produced are viewed as the major reasons this species is displacing the Oriental cockroach and becoming the predominant peridomestic species in these regions. Adults are 14−25 mm long. Females have very short, nonfunctional wings and are black, with light bands on the anterior margin of the thorax. Males are fully winged and light brown. Oothecae are 9−12 mm long, each typically containing 18 eggs. The biology of this species is similar to that of the Oriental cockroach, although their developmental time (3−4 months from egg to adult) is much shorter. The adults can live for almost 1 year. This cockroach is typically found infesting warehouses, steam tunnels, and sewers.

FIGURE 6.4 Turkestan cockroach (*Blatta lateralis*), female. *Photograph by Nathan D. Burkett-Cadena.*

American Cockroach (*Periplaneta americana*)

The American cockroach (Fig. 6.5) is a large species with adults being 34−53 mm in length. It is reddish brown, with substantial variation in light and dark patterns on the pronotum. Adults are winged and capable of flight. Nymphs typically complete development in 1.5−2 years while undergoing 10−13 molts. Adults live an average of seven months, but longevity may exceed two years. Females drop or glue their oothecae (8 mm long) to substrates within a few hours or days of formation. Each ootheca has

FIGURE 6.5 American cockroach (*Periplaneta americana*), female. *Courtesy of University of Florida/IFAS.*

FIGURE 6.6 Australian cockroach (*Periplaneta australasiae*), female. *Courtesy of University of Florida/IFAS.*

12−16 embryos. A female generally produces 9−10 egg cases during her life.

The American cockroach is perhaps the most cosmopolitan peridomestic pest species. Together with other closely related *Periplaneta* sp., *P. americana* is believed to have spread from tropical Africa to North America and the Caribbean on ships engaged in slave trading. Today this species infests most of the lower latitudes of both hemispheres and extends significantly into the more temperate regions of the world.

The habitats of this species are quite variable. American cockroaches commonly are found in commercial buildings, landfills, municipal sewage systems, storm drainage systems, septic tanks, crawl spaces beneath buildings, attics, treeholes, canopies of palm trees, voids in walls, ships, caves, and mines (i.e., in places characterized by darkness, high humidity, and low air flow). Studies conducted in Arizona (USA) indicated movement by a number of individuals several hundred meters through sewer systems and into neighboring homes. This species often can be seen at night on roofs and in air stacks or vents of sewage systems through which they enter homes and commercial buildings. Entrance also is gained to homes through laundry vent pipes and unscreened or unfiltered attic ventilation systems. This cockroach is known to move from crawl spaces of hospitals via pipe chases into operating theaters, patients' rooms, storage facilities, and food-preparation areas. Consequently, the potential of this cockroach for disseminating pathogenic microorganisms can be a significant concern for health-care personnel.

Australian Cockroach (*Periplaneta australasiae*)

Adult body coloration is similar to that of the American cockroach but with paler lateral markings on the anterior-lateral edges of the tegmina (Fig. 6.6). The pronotum is ringed with similar coloration. Adults are slightly smaller than American cockroaches, measuring 32−35 mm in length. Developmental time is about 1 year, and females typically live for an additional 4−6 months. A female can produce 20−30 oothecae during her lifetime; the ootheca is about 11 mm long and contains about 24 embryos. Embryonic development requires about 40 days. Nymphs are strikingly mottled, distinguishing them from nymphs of other *Periplaneta* sp.

This peridomestic species requires somewhat warmer temperatures than the American cockroach and does not occur in temperate areas other than in greenhouses and other pseudo-tropical environs. In the United States, outdoor populations are well established in Florida and along the coastal areas of Louisiana, Mississippi, Alabama, and Georgia. It commonly is found in environments similar to those inhabited by the smokybrown cockroach. In situations where both species occur (e.g., treeholes, attics), the Australian cockroach tends to displace the smokybrown. It can be a serious pest in greenhouses and other tropical environments in more temperate latitudes, such as atriums and interior plantscapes, where it can cause feeding damage to plants, notably, seedlings. Once in structures they occupy habitats similar to those of American cockroaches.

Brown Cockroach (*Periplaneta brunnea*)

The brown cockroach (Fig. 6.7) is smaller than the American cockroach (33−38 mm), and its pronotal markings are more muted. The most apparent diagnostic character for separating these two species is the shape of the last segment of the cercus; in the brown cockroach, the length is about equal to the width, whereas in the American cockroach, the length is about three times the width. The ootheca of the brown cockroach usually is larger (7−13 mm) and contains more embryos (24). The brown cockroach affixes its oothecae to substrates using salivary secretions. They give the ootheca a grayish hue not typical of other *Periplaneta*

FIGURE 6.7 Brown cockroach (*Periplaneta brunnea*), female. *Courtesy of University of Florida/IFAS.*

sp. that attach their oothecae with salivary secretions. This species is more subtropical than the American cockroach, occurring throughout the southeastern United States, where it infests homes and outbuildings. It is less frequently associated with sewage than is the American cockroach. Because of its similar appearance to the American cockroach, it is often misidentified and may be more widely distributed than is commonly recognized. In Florida, *P. brunnea* is commonly found in canopies of palm trees and attics. It also readily infests natural cavities, buildings, and other structures, like the American cockroach.

Smokybrown Cockroach (*Periplaneta fuliginosa*)

The smokybrown cockroach (Fig. 6.8) has become a major peridomestic pest throughout the southern United States, including southern California, and extends as far north as the mid-western states. It can be differentiated from the American cockroach by its slightly smaller size (25–33 mm) and uniform dark coloration. Although developmental times are quite variable, individuals mature in 1.5–2 years. Adults may live for about seven months. Females produce several ootheca that are 10–11 mm in length with 20 embryos, at 11-day intervals.

Primary foci for this peridomestic species in the southeastern United States are treeholes, canopies of palm trees, loose mulches such as pine straw or pine bark, and firewood piles. Within structures, *P. fuliginosa* seeks the ecological equivalent of treeholes, areas characterized as dark, warm, protective, and moist, with little air flow and near food resources. These include the soffit (eves) of underventilated attics, behind wall panels, the interstices of block walls, false ceilings, pantries, and storage areas. From these harborages, individuals forage for food and water, generally returning to the same refugia. Mark–release–recapture studies using baited live traps have shown that the median distance traveled between successive recaptures is less than 1 m, but that some adults may forage at distances of more than 30 m.

Florida Woods Cockroach (*Eurycotis floridana*)

This cockroach is restricted to a relatively small area of the United States along the Gulf of Mexico from eastern Louisiana to southeastern Georgia. It is mentioned here only because of its defensive capabilities. It is a large, dark-reddish-brown to black cockroach (Fig. 6.9), 30–40 mm long. Although small wing pads are evident, adults are apterous and are relatively slow moving. Oothecae are 13–16 mm long and contain about 22 embryos. *Eurycotis floridana* occurs in firewood piles, mulches, treeholes, attics, wall voids, and outbuildings. Last-instar nymphs and adults, if alarmed, can spray a noxious mix of aliphatic compounds that are both odoriferous and caustic. If sprayed into the eyes or onto soft tissues, a temporary burning sensation is experienced. Domestic dogs and cats quickly

FIGURE 6.8 Smokybrown cockroach (*Periplaneta fuliginosa*), female. *Courtesy of University of Florida/IFAS.*

FIGURE 6.9 Florida woods cockroach (*Eurycotis floridana*), male. *Courtesy of University of Florida/IFAS.*

learn to avoid this species. Among its common names are the **Florida cockroach**, the **Florida woods roach**, the **Florida stinkroach,** and **palmettobug**. The latter term also is commonly used for other *Periplaneta* sp.

Brown-Banded Cockroach (*Supella longipalpa*)

Like the German cockroach, this domestic species (Fig. 6.10) probably originated in tropical Africa, where it occurs both indoors and outdoors. In North America and Europe, it is confined almost exclusively to indoor environments of heated structures. In warm climates, infestations occur particularly in apartments without air conditioning and in commercial establishments with relatively high ambient temperatures, such as pet stores and animal-care facilities, office buildings, and schools. Adults are similar in size to the German cockroach (13−14.5 mm long) but lack pronotal stripes. Adults have two dark bands of horizontal stripes on the wings, whereas nymphs have two prominent bands running across the mesonotum and first abdominal segment. The brown-banded cockroach derives its name from these bands. Populations tend to occur in the nonfood areas of homes such as bedrooms, living rooms, and closets, as well as in electronics equipment, such as Central Processing Units (CPUs) and telephones, and storage cabinets in commercial office buildings. Male brown-banded cockroaches occasionally fly and are attracted to lights. Members of this species seek harborage higher within rooms than does the German cockroach. Developmental time from egg to adult averages 5−6 months. The ootheca is small, only 5 mm long, with an average of 18 embryos and an incubation time of 35−80 days. Females affix their oothecae to furniture, in closets, on or behind picture frames, and in bedding. Transporting *S. longipalpa* with furniture to new locales is common. Although this species occurs with other cockroaches in homes, the German cockroach often outcompetes it within a few months.

German Cockroach (*Blattella germanica*)

This cockroach also is known as the **steamfly** in Great Britain. It is believed to have originated in northern or eastern Africa, or Asia, and has spread from there via commerce. The German cockroach is considered to be the most important domestic pest species throughout the developed world. Adults are about 16 mm long with two dark, longitudinal bands on the pronotum (Fig. 6.11). It requires warm (optimally 30−33°C), moist conditions near adequate food resources. It primarily inhabits kitchens and pantries, with secondary foci in bathrooms, bedrooms, and other living spaces in heavily infested structures. Although this species is nocturnal like most other cockroaches, some individuals may be seen moving about on walls and in cupboards during the daylight hours where infestations are heavy. Their wing musculature is vestigial, making them unable to fly except for short, gliding, downward movements. *Blattella germanica* does not readily move between buildings; however, it does occur in garbage collection containers and outbuildings near heavily infested structures.

The German cockroach has a high reproductive potential. Females produce an ootheca (6−9 mm) containing about 30−40 embryos within 7−10 days after molting to the adult, or about 2−3 days after mating. The female carries the egg case until a few hours before hatching of the nymphs, preventing access of any oothecal parasitoids or predators. Oothecae are produced at intervals of 20−25 days, with a female producing four to eight oothecae during her lifetime. Nymphs complete their development in 7−12 weeks.

This species is the main cockroach pest in most households and apartment complexes. Control is difficult,

FIGURE 6.10 Brown-banded cockroach (*Supella longipalpa*), female. *Courtesy of University of Florida/IFAS.*

FIGURE 6.11 German cockroach (*Blattella germanica*), female. *Courtesy of University of Florida/IFAS.*

in part because of their movement between apartments through plumbing chases in shared or adjacent walls. Researchers studying more than 1,000 apartments in Florida concluded that the median number of cockroaches per apartment was greater than 13,000. This high biotic potential makes this species a major nuisance, as well as a pest with implications for human health.

Asian Cockroach (*Blattella asahinai*)

The Asian cockroach is closely related to the German cockroach, and they are difficult to distinguish morphologically. In fact, Asian and German cockroaches are capable of hybridizing and producing fertile offspring, which further complicates their identifications. Techniques have been developed to differentiate these two species and their hybrids based on cuticular hydrocarbons in the waxy layer of the integument.

Despite their morphological similarity, *B. asahinai* differs from *B. germanica* in several aspects of its behavior and ecology. It is both a feral and a peridomestic species. Nymphs of the Asian cockroach commonly occur, sometimes in large numbers, in leaf litter and in areas of rich ground cover or well-maintained lawns. Unlike the German cockroach, the adults fly readily and are most active beginning at sunset when they fly to light-colored walls or brightly lit areas. This behavior can make invasion a nightly occurrence in homes near heavily infested areas. Flight does not occur when temperatures at sunset are below 21°C.

Like those of the German cockroach, Asian cockroach females carry their oothecae until shortly before they are ready to hatch. The ootheca is similar in size and contains a similar number of embryos as the German cockroach (30−40). Nymphs are smaller than their *B. germanica* counterparts and are somewhat paler in appearance. Development from egg to adult requires about 65 days, with females producing up to six oothecae during their life span. Adults are slightly smaller than those of *B. germanica* (average of 13 mm).

The Asian cockroach was first described in 1981 from specimens collected in sugar cane fields on the Japanese island of Okinawa. When it was first discovered in the United States in 1986, the Asian cockroach was found only locally in three counties in Florida from Tampa to Lakeland; populations already had become established with densities as high as 250,000 per hectare. By 1993 this species had spread to at least 30 Florida counties and had infested citrus groves throughout the central part of the state. It is now a common occurrence throughout northern Florida, and it has been recorded in southeastern Georgia. It feeds on succulent early growth of citrus nursery stock, tassels of sweet corn, strawberries, cabbage, tomatoes, and other agricultural products, although there has been no evidence of significant economic damage.

Infestations of apartments by *B. ashinai* have become common in much of Florida. This cockroach also has become an increasing problem in warehouses, department stores, hotels, fast-food establishments, automobile dealerships, and other businesses with hours of operation that extend beyond dusk.

Surinam Cockroach (*Pycnoscelus surinamensis*)

This species is believed to have originated in the Indo-Malayan region. It commonly occurs outdoors in the southeastern United States from North Carolina to Texas. The adults are fairly stout, 18−25 mm in length, with shiny brown wings and a black body (Fig. 6.12). Nymphs characteristically have shiny black anterior abdominal segments, whereas the posterior segments are dull black and roughened. In North America this species is unusual in that it is parthenogenetic, producing only female offspring; elsewhere both males and females are found. The ootheca is 12−15 mm long, is poorly sclerotized, and contains about 26 embryos. The ootheca is retained inside the genital chamber from which the nymphs emerge in about 35 days. Females produce an average of three oothecae and live about 10 months in the laboratory. This cockroach commonly burrows into compost piles and the thatch of lawns. Transfer of fresh mulch into the home for potting plants can result in household infestations. Adult females fly and are attracted to light. They are most likely to be noticed by homeowners at night when they fly into brightly lit television screens. This species commonly is transported in commercial mulch and plant material to more temperate areas of the United States where it has been known to infest greenhouses, indoor plantings in shopping malls, atriums in office buildings, and zoos.

FIGURE 6.12 Surinam cockroach (*Pycnoscelus surinamensis*). *Courtesy of University of Florida/IFAS.*

PUBLIC HEALTH IMPORTANCE

Cockroaches infesting human dwellings and workplaces represent a more intimate and chronic association than do most other pests of medical-veterinary importance. High populations of any cockroach species may adversely affect human health in several ways. These include contamination of food with their excrement, mechanical dissemination of pathogens, induced allergies, psychological stress, and bites. Although documentation of bites is limited, there are reports of cockroaches feeding on fingernails, eyelashes, skin calluses of hands and feet, and food residues about the faces of sleeping humans, causing blisters and small wounds (Roth and Willis, 1957, 1960). There are other accounts of bites around the mouths of infants in heavily infested homes and even in hospitals. American and Australian cockroaches are the more often implicated species. Bites by the Oriental cockroach have resulted in inflammation of the skin, degeneration of epithelial cells, and subsequent necrosis of the involved tissues.

While many individuals develop a tolerance for cockroach infestations, others may experience psychological stress. The level of stress tends to be proportional to the size of the cockroaches and the magnitude of the infestation. An aversion to cockroaches may be so strong that some people become irrational in their behavior, imagining a severe infestation even when there is none. This illusion of abundant cockroaches has caused some families to move out of their homes. High cockroach populations also produce a characteristic odor that can be unpleasant or even nauseating to some people. Food stuffs may become contaminated with the excrement of cockroaches, which, on subsequent ingestion, may cause vomiting and diarrhea.

The presence of cockroaches in homes does not necessarily imply poor housekeeping. Peridomestic species such as the American and the Oriental cockroach commonly infest municipal sewage systems or septic tanks and may move into homes through sewage lines. Any of the *Periplaneta* sp. may develop high outdoor populations, inducing individuals to seek less crowded environments. At such times, they often enter homes through attic vents, through breaches in construction joints, or through crawl spaces. This tends to occur in early fall. While they are active at night, the smokybrown cockroach, Asian cockroach, and feral wood roaches (*Parcoblatta* species) often find their way into even the best-kept homes. Adults frequently alight on doors illuminated by entrance lights or on window screens of lighted rooms. Entrance is gained once the door is opened or by squeezing past window-screen frames.

Poor housekeeping and unsanitary conditions contribute significantly to cockroach infestations. The German cockroach and, to a lesser degree, the brown-banded cockroach are the principal banes of apartment dwellers. Their survival is enhanced by crowded living quarters, associated clutter, and the accumulated organic debris associated with food preparation. Construction practices used to build apartment complexes (e.g., common wiring ducts, sewage lines, and refuse areas) can contribute to the spread of cockroaches in multi-unit dwellings.

PATHOGENIC AGENTS

The significance of cockroaches in public health remains controversial despite the logical assumption that they play a role in transmitting pathogenic agents. Given that cockroaches are so closely associated with humans and poor sanitation, the potential for acquiring and mechanically transmitting disease agents is very real. They are capable of transmitting microorganisms (Fig. 6.13) and other disease agents indirectly by contaminating foods or food-preparation surfaces. Garcia et al. (2012) suggest that the control of peridomestic cockroach species in food-related environments is essential in preventing food-borne illnesses.

Table 6.2 lists pathogenic organisms that have been isolated from cockroaches in domestic or peridomestic environments. At least 32 species of bacteria in 16 genera are represented. These include such pathogens as *Bacillus subtilis*, a causative agent of conjunctivitis; *Escherichia coli* and nine strains of *Salmonella*, causative agents of diarrhea, gastroenteritis, and food poisoning; *Salmonella typhi*, the causative agent of typhoid; and four *Proteus* spp. that commonly infect wounds. These isolations primarily have involved American, German, and Oriental cockroaches. Cockroaches also have been found harboring the eggs of seven species of helminths, at least 17 fungal species, three protozoan species, and two strains of poliomyelitic virus (Brenner et al., 1987; Koehler et al., 1990; Brenner, 1995). Researchers in Costa Rica have shown that

FIGURE 6.13 Bacteria adhering to tarsus of German cockroach (*Blattella germanica*). *From Gazivoda and Fish, 1985; permission of New York Entomological Society.*

TABLE 6.2 Bacteria Pathogenic to Humans That Have Been Isolated From Field-Collected Cockroaches

Pathogen	Associated Disease	Cockroach Species
Acinetobacter sp.	Nosocomial infection	*Blattella germanica, Periplaneta americana*
Aeromonas sp.	Wound and other infections; diarrhea	*B. germanica, Diploptera punctata*
Alcaligenes faecalis	Gastroenteritis, secondary infections, urinary tract infections	*Blatta orientalis, P. americana*
Bacillus subtilis	Conjunctivitis, food poisoning	*Blaberus craniifer, B. orientalis, B. germanica, P. americana*
Bacillus cereus	Food poisoning	*B. craniifer*
Campylobacter jejuni	Enteritis	*B. orientalis, P. americana*
Citrobacter sp.	Urinary tract infections, infant meningitis	*B. germanica, D. punctata, P. americana*
Clostridium novii	Gas gangrene	*B. orientalis*
Clostridium perfringens	Food poisoning, gas gangrene	*B. orientalis* and other species
Enterobacter sp.	Bacteremia	*B. germanica, D. punctata, P. americana*
Enterococcus sp.	Urinary tract and wound infections	*B. germanica, P. americana*
Escherichia coli	Diarrhea, wound infection	*B. orientalis, B. germanica, D. punctata, P. americana*
Hafnia alvei	Diarrhea	*B. germanica, P. americana*
Klebsiella sp.	Pneumonia, urinary-tract infections	*B. germanica, D. punctata, P. americana*
Leptospira ssp.	Leptospirosis	*Periplaneta* spp.
Mycobacterium leprae	Leprosy	*B. germanica, P. americana, P. australasiae*
Nocardia sp.	Actinomycetoma	*P. americana*
Morganella morganii	Wound infection	*B. germanica, P. americana*
Oligella urethralis		*P. americana*
Pantoea sp.	Wound infection	*B. germanica*
Proteus rettgeri	Wound infection	*P. americana*
Proteus vulgaris	Wound infection	*B. craniifer, B. orientalis, D. punctata, P. americana*
Proteus mirabilis	Gastroenteritis, wound infection	*P. americana*
Pseudomonas sp.	Respiratory infections, gastroenteritis	*D. punctata, Blaberus craniifer, B. orientalis, B. germanica, P. americana*
Salmonella sp.	Food poisoning, gastroenteritis	*D. punctata*
Salmonella bredeny	Food poisoning, gastroenteritis	*P. americana*
Salmonella newport	Food poisoning, gastroenteritis	*P. americana*
Salmonella oranienburg	Food poisoning, gastroenteritis	*P. americana*
Salmonella panama	Food poisoning, gastroenteritis	*P. americana*
Salmonella paratyphi-B	Food poisoning, gastroenteritis	*P. americana*
Salmonella pyogenes	Pneumonia	*B. orientalis*
Salmonella typhi	Typhoid	*B. orientalis*
Salmonella typhimurium	Food poisoning, gastroenteritis	*B. germanica, Nauphoeta cinerea*
Salmonella bovis-morbificans	Food poisoning, gastroenteritis	*P. americana*
Salmonella bareilly	Food poisoning, gastroenteritis	*P. americana*
Sphingobacterium sp.	Sepsis	*B. germanica, P. americana*

Continued

TABLE 6.2 Bacteria Pathogenic to Humans That Have Been Isolated From Field-Collected Cockroaches—cont'd

Pathogen	Associated Disease	Cockroach Species
Serretia sp.	Food poisoning	*B. orientalis, B. germanica, D. punctata, P. americana*
Shigella dysenteriae	Dysentery	*B. germanica, Blatta lateralis*
Sphingobacterium mizutae		*P. americana*
Staphylococcus aureus	Wound infection, skin infection, infection of internal organs	*B. craniifer, B. orientalis, B. germanica, D. punctata, P. americana*
Staphylococcus epidermidis	Wound infection	*B. germanica, P. americana*
Streptococcus faecalis and other spp.	Pneumonia	*B. orientalis, B. germanica, P. americana*
Vibrio spp.	Not applicable	*B. orientalis*
Yersinia pestis	Plague	*B. orientalis*

Australian, American, and Madeira cockroaches become infected with the protozoan *Toxoplasma gondii* after eating feces of infected cats. This suggests the possibility of cockroach involvement in the maintenance and dissemination of this parasite, which causes toxoplasmosis in humans, cats, and other animals.

Although many pathogens have been recovered from natural populations of cockroaches, this does not necessarily mean that cockroaches serve as their vectors. Isolation of pathogens from cockroaches simply may be indicative of the natural microbial fauna and flora in our domestic environment. Under certain circumstances, however, cockroaches have the potential for serving as secondary vectors of agents that normally are transmitted by other means. Anecdotal accounts associating diseases in humans with the occurrence of cockroaches and microbes lend some credence to the hypothesis that these pests can serve as vectors. Burgess (1982) reported the isolation from German cockroaches of a serotype of *S. dysenteriae* that was responsible for an outbreak of **dysentery** in Northern Ireland. Mackerras and Mackerras (1948) isolated *Salmonella bovis-morbificans* and *Salmonella typhimurium* from cockroaches captured in a hospital ward where **gastroenteritis**, attributed to the former organism, was common. In subsequent experimental studies, *Salmonella* organisms remained viable in the feces of cockroaches for as long as 40 days postinfection (Mackerras and Mackerras, 1949). Some of the most compelling circumstantial evidence suggesting that cockroaches may be vectors was noted in a correlation between cases of infectious **hepatitis** and cockroach control at a housing project during 1956−1962 in southern California (Tarshis, 1962). The study area involved more than 580 apartments and 2,800 persons; 95% of the apartments had German cockroaches and a lesser infestation of brown-banded and Oriental cockroaches. After pest control measures were initiated, the incidence of endemic infectious hepatitis decreased for 1 year. When treatments were discontinued during the following year because the insecticide was offensive to apartment dwellers, the cockroach population increased, accompanied by a corresponding increase in the incidence of hepatitis. Effective control measures were applied the following two years, and cockroach populations and cases of infectious hepatitis dropped dramatically while hepatitis rates remained high in nearby housing projects where no pest control measures were conducted.

INTERMEDIATE HOSTS

Cockroaches can serve as intermediate hosts for animal parasites (Table 6.3). Roth and Willis (1960) published an extensive list of biotic associations between cockroaches and parasitic organisms that potentially infest humans. The eggs of seven species of helminths have been found naturally associated with cockroaches: hookworms (*Ancylostoma duodenale* and *Necator americanus)*, giant human roundworm (*Ascaris lumbricoides)*, other *Ascaris* spp., pinworm (*Enterobius vermicularis*), tapeworms (*Hymenolepis* sp.), and the whipworm *Trichuris trichuria*. Development of these helminths in cockroaches has not been observed. These relationships probably represent incidental associations with the omnivorous feeding behavior of cockroaches. However, cockroaches may serve as potential reservoirs and possible vectors through mechanical transfer in areas where a high incidence of these pathogens in humans is accompanied by substantial cockroach infestations. Human infestations by spirurid nematodes associated with cockroaches are known only for the **cattle gullet worm** (*Gongylonema pulchrum*) in the United States, Europe, Asia, and Africa and for the **stomach worm**

TABLE 6.3 Cockroaches as Intermediate Hosts of Parasites of Veterinary Importance

Phylum and Parasite	Scientific Name	Definitive Hosts	Cockroach Intermediate Host
Acanthocephala (thorny-headed worms)			
	Moniliformis moniliformis	Rat, mice, dog, cat (primates)	*Blatta orientalis, Blattella germanica*
	Moniliformis dubius	Rat	*B. germanica, Periplaneta americana, Periplaneta brunneus*
	Prosthenorchis elegans Prosthenorchis spirula	Captive primates	*B. germanica, Leucophaea maderae,* others
Pentastomida (tongue worms)			
	Raillietiella hemidactyli	Reptiles	*P. americana*
Nematoda (round worms)			
Gastric metazoan parasites	*Abbreviata antarctica*	Reptiles	*Nauphoeta cinerea*
Esophageal and gastrointestinal worm	*Abbreviata caucasica*	Primates (humans)	*B. germanica*
Stomach worm	*Cyrnea colini*	Prairie chicken, turkey, bobwhite, quail	*B. germanica, P. americana*
Esophagus worm	*Gongylonema neoplasticum*	Rodents, rabbit	*B. orientalis, P. americana*
Gullet worm	*Gongylonema pulchrum*	Cattle (humans)	*B. germanica*
Gullet worm	*Gongylonema* sp.	Marmosets and Tamarins	*P. americana*
Stomach worm	*Mastophorus muris*	Rodents, cat	*Leucophaea maderae, P. americana*
Eye worm	*Oxyspirura mansoni*	Chicken, turkey	*Pycnoscelus surinamensis*
Eye worm	*Oxyspirura parvorum*	Chicken, turkey	*P. surinamensis*
Esophageal worm	*Physaloptera rara*	Dog, cat, raccoon, coyote, wolf, fox	*B. germanica*
Esophageal worm	*Physaloptera praeputialis*	Dog, cat, coyote, fox	*B. germanica*
Round worms	*Protospirura bonnei Protospirura muricola*	Monkeys	*B. germanica, Supella longipalpa*
Stomach worm	*Spirura rytipleurites*	Cat, rat	*B. orientalis*
Stomach worm	*Tetrameres americana*	Chicken, bobwhite, ruffed grouse	*B. germanica*
Stomach worm	*Tetrameres fissipina*	Ducks, geese, waterfowl, chicken, turkey, pigeon, quail	Various species

Rare definitive hosts are listed in parentheses.

Abbreviata caucasia in Africa, Israel, Colombia, and Chile. Human cases involving these parasites are rare and cause no pathology.

There are a number of reports of bronchopulmonary infection caused by *Lophomonas blattarum*, a multiflagellated protozoan that parasitizes the gut of cockroaches and termites (see Saldana et al., 2017). Once the cockroach voids the protozoan in the feces, the parasite forms a cyst that is quite persistent in the environment. Nearly 140 cases have been reported, mostly from China, where there are also urban and suburban cultural practices of consuming cockroaches. The majority of cases involve immunocompromised persons, although cases in immunocompetent persons are also documented.

COCKROACH ALLERGIES

The importance of **cockroach allergies** and their implications on childhood asthma has been well documented since first reports were published in the 1960s. Allergic reactions result after initial sensitization to antigens after inhalation, ingestion, dermal abrasion, or injection.

Allergens produced by cockroaches now are recognized as one of the more significant indoor allergens of modernized societies. Among asthmatics, about half are allergic to cockroaches. This rate is exceeded only by allergies to house-dust mites. Sensitivity to cockroaches also affects about 10% of nonallergic individuals, suggesting a subclinical level of allergy.

Symptoms exhibited by persons allergic to cockroaches are similar to those described by Wirtz (1980), who reported on occupational allergies in entomologists. They include sneezing and a runny nose, skin reactions, and eye irritation in about two-thirds of the cases. In the more severe cases, individuals may experience difficulty breathing or, even more alarming, anaphylactic shock after exposure to cockroaches. Such allergic reactions can be life threatening (Brenner et al., 1991).

Since the 1990s, research has focused on determining the specific components of cockroaches that cause allergy. Laboratory technicians exhibit strong allergies to cast skins and excrement of German cockroaches, whereas most patients seen at allergy clinics react primarily to cast skins and whole-body extracts of German cockroaches. Once individuals are hypersensitized, they may experience severe respiratory distress simply by entering a room where cockroaches are held.

Several proteins that can cause human allergies have been identified in the German cockroach. Different exposure histories are likely to result in allergies to different proteins. Cast exoskeletons, excrement, and partially consumed food of cockroaches, in addition, to living cockroaches, all produce allergenic proteins. Some are extremely persistent and can survive boiling water, ultraviolet light, and harsh pH changes, remaining allergenically potent for decades. Traditionally, whole-body extracts have been used to screen for allergens in skin tests and in bronchial challenges for diagnosing cockroach allergies (Fig. 6.13). However, use of more specific antigens that become aerosolized in cockroach-infested homes may be more appropriate, as this is likely to be the sensitizing material. Studies with laboratory colonies have shown that a population of several thousand German cockroaches produced several micrograms of aerosolized proteins in 48 h. Consequently, the presence of cockroaches may have profound respiratory implications for asthmatic occupants of infested structures. For a general discussion on aerosolized arthropod allergens, see Solomon and Mathews (1988), and for a recent comprehensive review of cockroach allergen literature, see Pomés et al. (2017).

Development of an allergy to one insect (or other arthropod) species can result in broad cross-reactivity to other arthropods, including shrimp, lobster, crab and crawfish, sowbugs (isopods), and house-dust mites. Chronic indoor exposure to cockroach allergens, therefore, may have significant and widespread effects on human health. For a comprehensive review of literature on cockroach allergens with a focus on improving diagnosis and treatment, see Pomés and Arruda (2014). Implications for human health may extend beyond inhalant allergens. One recent article elucidates the association between allergic diseases, allergic-sensitization, and attention-deficit/hyperactivity disorder in children. Cockroach and other arthropod allergens (mite and crab) were significant correlates (Yang et al., 2018).

Remarkably, the literature also supports a beneficial aspect of cockroaches to human health. *Kangfuxin* is a popular traditional Chinese medicine based on ethanol extracts from the American cockroach, *Periplaneta americana*. Four compounds isolated from these extracts have been shown to have pharmacological values in healing of burns, wounds, and ulcers (Zhu, et al., 2018). Earlier studies (Zhang et al., 2013) had also indicated there is value in using a *P. americana* extract as a protective intestinal mucosal barrier in patients with sepsis.

VETERINARY IMPORTANCE

Cockroaches serve as intermediate hosts for a number of parasitic worms of animals (Table 6.3). Most of these relationships are of no economic importance. The majority of the parasites are nematodes in the order Spirurida, all members of which use arthropods as intermediate hosts. Species infesting dogs and cats, among other hosts, attach to the mucosa of the gastrointestinal tract where erosion of tissue may occur at the points of attachment. Although serious damage seldom occurs, anemia and slow growth may result. Several cockroach-associated nematodes occur in Europe and North America. The **esophageal worms** *Physaloptera rara* and *P. praeputialis* are the most widespread species in the United States. They develop in the German cockroach, field crickets, and several species of beetles.

Poultry also are parasitized by nematodes that undergo development in cockroaches. The Surinam cockroach is the intermediate host for the **poultry eye worms** *Oxyspirura mansoni* and *O. parvorum*. Both occur in many parts of the world. In the United States, their distribution is limited to Florida and Louisiana. The German cockroach has been incriminated as the intermediate host for chicken and turkey parasites, including the **stomach worms** *Tetrameres americana, T. fissispina,* and *Cyrnea colini*; the latter also develops in the American cockroach. *Cyrnea colini* apparently causes no significant damage to poultry, but *Oxyspirura* sp. can cause pathology ranging from mild conjunctivitis to severe ophthalmia with seriously impaired vision. *Tetrameres fissispina* can cause severe damage to the proventriculus of infested birds.

Several nematode parasites of primates, rats, and cattle use cockroaches as intermediate hosts (Table 6.3).

These include *Gongylonema neoplasticum* and *Mastophorus muris* in rodents. Both genera occur widely in the United States, where they cause no known pathological problems. The **gullet worm** of cattle, *Gongylonema pulchrum*, has been shown experimentally to undergo development in the German cockroach. A *Gongylonema* sp. has been isolated from tongue scrapings of primates in zoos, where cockroaches and dung beetles have been found to be the intermediate hosts.

Exotic zoo animals also can become infested with parasitic nematodes for which cockroaches serve as possible intermediate hosts. *Protospirura bonnei* and *P. muricola*, for example, have been found in cockroaches collected in cages of monkeys. In a case of "wasting disease" in a colony of common marmosets, more than 50% of German and brown-banded cockroaches captured in the animal room in which they were housed contained the coiled larvae of *Trichospirura leptostoma* in muscle cells (Beglinger et al., 1988).

Acanthocephalans (thorny-headed worms) commonly infest primates in zoos and research facilities. *Prosthenorchis elegans* and *P. spirula* occur naturally in South and Central America. Their natural intermediate hosts are unknown. In captivity, primates become infected after eating any of several cockroach species in which the intermediate stages of the parasite have completed development. Heavily infested primates frequently die within a few days. The proboscis of acanthocephalan adults commonly penetrates the intestines of the primate host, causing secondary infections, perforation of the gut wall, and peritonitis.

One **pentastomid** (tongue worm), *Raillietiella hemidactyli*, develops in cockroaches and reptilian hosts. In Singapore, infested geckos are a common occurrence in houses where heavy infestations of *R. hemidactyli* larvae have been found in American cockroaches. Remnants of cockroaches are found commonly in the guts of these lizards.

For additional information on the veterinary importance of cockroaches, see Chitwood and Chitwood (1950), Roth and Willis (1957), Levine (1968), and Noble and Noble (1976).

PREVENTION AND CONTROL

Integrated pest management strategies are commonly used to manage cockroach infestations. Integrated pest management is a multifaceted approach that attempts to eliminate the habitat and conditions that sustain cockroach populations, using mechanical, biological, physical, and/or chemical means.

Sanitation

Cleanup to reduce cockroaches should focus mainly on the food residue in and around coffee machines, sinks, stoves, microwave ovens, refrigerators, trash cans, and furniture where food residues accumulate. Removal of clutter, such as corrugated cardboard, is especially important because clutter provides excellent harborage for cockroaches.

Harborage Elimination

Permanent reduction of cockroach populations can be achieved by eliminating harborage through caulking and sealing. If not performed properly, this can make a bad problem worse by creating additional inaccessible harborage. Building designs and construction techniques can significantly influence cockroach survival. By manipulating microclimates in areas of structures frequented by cockroaches, homes and other buildings can be rendered less hospitable to pest species, while at the same time greatly reducing aerosolized allergens. Nontoxic repellents can be used to deter cockroaches from entering specific areas.

Physical Control

This includes a variety of mechanical techniques such as vacuuming, sticky traps, and pitfall traps, which are used to reduce cockroach numbers by removing them from the environment. Heat, cold, anoxia, and steam also can be used to kill cockroaches.

Biological Control

This has drawn increased attention in recent years. Among the natural agents that have been investigated are parasitic wasps, nematodes, and sporulating fungi. Females of the eulophid wasp *Aprostocetus hagenowii* and the encyrtid wasp *Comperia merceti* deposit their eggs in the oothecae of certain peridomestic cockroaches. Major shortcomings in using these wasps are difficulties involved in their mass production and the fact that they do not completely eliminate cockroach infestations. However, *A. hagenowii* has been shown to reduce populations of the peridomestic *Periplaneta* sp. after inundative or augmentative releases of this wasp. *Comperia merceti* parasitizes oothecae of the brown-banded cockroach and is the only known parasitoid of a domestic species. The use of **parasitic nematodes** (e.g., *Steinernema carpocapsae*) and several **fungal pathogens** that have been isolated from cockroaches have not yet proved to be effective as practical management tools. Another drawback to their use is the allergenic nature of several components of nematodes and many sporulating fungi that can become airborne and, on inhalation, cause asthmatic responses in humans.

Traditionally, cockroaches were controlled by using residual **pesticides**, such as organophopshates and carbamates (most are no longer registered for use) applied to

harborage sites and areas frequented by foraging individuals (Ebling, 1975; Rust et al., 1995). Other widely used products for these purposes include pyrethroids and botanicals such as pyrethrins, as well as several new classes of insecticides that are metabolic inhibitors or disruptors (e.g., chlornicotinyls, pyrroles, macrocyclic lactones, amidinohydrazone, and phenylpyrazoles). These active ingredients are formulated into a variety of products, such as wettable powders, emulsifiable concentrates, aerosols, dusts, microencapsulates, and baits. Other materials with different modes of action can be used. For instance, boric acid is delivered as a fine powder or a dilute solution, which when ingested damages the gut epithelium of cockroaches and kills them by interfering with nutrient absorption. Inorganic silica dust is absorptive, reducing cuticular lipids and causing desiccation. The use of baits containing several of the active ingredients mentioned here currently are used extensively to control cockroaches. These baits are used indoors in the form of child-resistant bait stations and/or gels that are applied in cracks and crevices, making them inaccessible to children and pets. Scatter baits are commonly used outdoors to treat mulches and other landscaping materials that harbor cockroaches.

Insect Growth Regulators

Insect growth regulators can be used to prevent cockroaches from reaching maturity. Two commonly used regulators are juvenile hormone analogs and chitin synthesis inhibitors. **Juvenile hormone analogs** regulate morphological maturation and reproductive processes. They are highly specific to arthropods, have very low mammalian toxicity, and are effective at exceptionally low rates of application. Such compounds include hydroprene and pyriproxifen. **Chitin synthesis inhibitors** prevent normal formation of chitin during molting. These compounds cause many of the affected nymphs to die during the molting process. Males that survive to the adult stage often have reduced life expectancies, whereas females tend to abort their oothecae.

REFERENCES AND FURTHER READING

Atkinson, T. H. P., Koehler, G., & Patterson, R. S. (1991). Catalog and atlas of the cockroaches (Dictyoptera) of North America north of Mexico. *Miscellaneous Publications of the Entomological Society of America, 78*, 1–86.

Beglinger, R., Illgen, B., Pfister, R., & Heider, K. (1988). The parasite *Trichospirura leptostoma* associated with wasting disease in a colony of common marmosets, *Callithrix jacchus*. *Folia Primatologica, 51*, 45–51.

Bell, W. J., Roth, L. M., & Nalepa, C. A. (2007). *Cockroaches: Ecology, behavior, and natural history*. Baltimore: The Johns Hopkins University Press, 272 pp.

Boudreaux, H. B. (1979). *Arthropod phylogeny with special reference to insects*. New York: John Wiley & Sons, 320 pp.

Brenner, R. J. (1988). Focality and mobility of some peridomestic cockroaches in Florida. *Annals of the Entomological Society of America, 81*, 581–592.

Brenner, R. J. (1991). Asian Cockroaches: Implications to the food industry and complexities of management strategies. In J. R. Gorham (Ed.), *U.S. Food & Drug Administration Technical Bulletin No. 4Ecology and management of food-industry pests* (pp. 121–130).

Brenner, R. J. (1995). Economics and medical importance of German cockroaches. In M. K. Rust, J. M. Owens, & D. A. Reierson (Eds.), *Understanding and controlling the German cockroach*. Oxford University Press, 430 pp.

Brenner, R. J., Barnes, K. C., Helm, R. M., & Williams, L. W. (1991). Modernized society and allergies to arthropods: Risks and challenges to entomologists. *American Entomologist, 37*, 143–155.

Brenner, R. J., Koehler, P. G., & Patterson, R. S. (1987). Implications of cockroach infestations to human health. *Infections in Medicine, 4*, 349–355, 358,359, 393.

Brenner, R. J., Patterson, R. S., & Koehler, P. G. (1988). Ecology, behavior, and distribution of *Blattella asahinai* (Orthoptera: Blattellidae) in central Florida. *Annals of the Entomological Society of America, 81*, 432–436.

Burgess, N. R. (1982). Biological features of cockroaches and their sanitary importance. In D. Bajomi, & G. Erdos (Eds.), *The modern defensive approach of cockroach control* (pp. 45–50). International Symposia, Bologna.

Chitwood, B. G., & Chitwood, M. B. (1950). *An introduction to nematology. Section I. Anatomy*. Baltimore: B.G. Chitwood, 213 pp.

Cornwell, P. B. (1968). *The cockroach* (Vol. 1). London: Hutchinson & Co. Ltd., 392 pp.

Ebling, W. (1975). *Urban entomology*. Berkeley: University of California., Div. Agr. Sci.

Elgderi, R. M., Ghenghesh, K. S., & Berbash, N. (2006). Carriage by the German cockroach (*Blattella germanica*) of multiple-antibiotic-resistant bacteria that are potentially pathogenic to humans, in hospitals and households in Tripoli, Libya. *Annals of Tropical Medicine and Parasitology, 100*, 55–62.

Garcia, F., Notario, M. J., Cabanas, J. M., Jordano, R., & Medina, L. M. (2012). Incidence of bacteria of public health interest carried by cockroaches in different food-related environments. *Journal of Medical Entomology, 49*, 1481–1484.

Gazivoda, P., & Fish, D. (1985). Scanning electron microscope demonstration of bacteria on tarsi of *Blattella germanica*. *Journal of the New York Entomological Society, 93*, 1064–1067.

Gonzalez-Astudillo, V., Bustamante-Rengifo, J. A., Bonilla, A., Lehmicke, A. J. J., Castillo, A., & Astudillo-Hernandez, M. (2016). Synantropic cockroaches (Blattidae: *Periplaneta* spp.) harbor pathogenic *Leptospira* in Columbia. *Journal of Medical Entomology, 53*, 177–182.

Helfer, J. R. (1987). *How to know the grasshoppers, cockroaches and their allies*. Dubuque: W. C. Brown Co, 353 pp.

Koehler, P. G., Patterson, R. S., & Brenner, R. J. (1990). Cockroaches (Chapter 3). In K. Story (Ed.), *Mallis handbook of pest control* (7th ed.). Cleveland: Franzak and Foster Co.

Kim, T., & Rust, M. K. (2013). Life history and biology of the invasive Turkestan cockroach (Dictyoptera: Blattidae). *Journal of Economic Entomology, 106*, 2428–2432.

King, C., Jones, H. I., & Chin, Y. T. (2013). Arthropod intermediate hosts of *Abbreviata Antarctica* (Nematoda: Physalopteridae) in Australia. *The Journal of Parasitology, 99*, 708–711.

Legendre, F., Nel, A., Svenson, G. J., Robillard, T., Pellens, R., & Grandcolas, P. (2015). Phylogeny of Dictyoptera: Dating the origin of cockroaches, praying mantises and termites with molecular data and controlled fossil evidence. *PLoS One, 10*(7), e0130127.

Lemos, A. A., Lemos, J. A., Prado, M. A., Pimenta, F. C., Gir, E., Silva, H. M., et al. (2006). Cockroaches as carriers of fungi of medical importance. *Mycoses, 49*, 23–25.

Levine, N. D. (1968). *Nematode parasites of domestic animals and of man*. Minneapolis: Burgess Pub.Co., 600 pp.

Mackerras, M. J., & Mackerras, I. M. (1948). *Salmonella* infections in Australian cockroaches. *Australian Journal of Science, 10*, 115.

Mackerras, I. M., & Mackerras, M. J. (1949). An epidemic of infantile gastroenteritis in Queensland caused by *Salmonella bovis-morbicans* (Basenau). *Journal of Hygiene, 47*, 166–181.

Massicot, J. G., & Cohen, S. G. (1986). Epidemiologic and socioeconomic aspects of allergic diseases. *The Journal of Allergy and Clinical Immunology, 78*, 954–958.

McKittrick, F. A. (1964). Evolutionary studies of cockroaches. In *Cornell University Agricultural Experiment Station Memoir No. 389*, 192 pp.

McCanless, K. (October 2000). *Oriental cockroach*. University of Florida. http://entomology.ifas.ufl.edu/creatures/urban/roaches/oriental_cockroach.htm#ref.

Mpuchane, S., Allotey, J., Matsheka, I., Simpanya, M., Coetzee, S., Jordaan, A., et al. (2006). Carriage of micro-organisms by domestic cockroaches and implications on food safety. *International Journal of Tropical Insect Science, 26*, 166–175.

Noble, E. R., & Noble, G. A. (1976). *Parasitology* (4th ed.). Philadelphia: Lea & Febiger, 566 pp.

Pai, H.-H., Chen, W.-C., & Peng, C.-F. (2004). Cockroaches as potential vectors of nosocomial infections. *Infection Control and Hospital Epidemiology, 25*, 979–984.

Pomés, A., & Arruda, L. K. (March 1, 2014). Investigating cockroach allergens: Aiming to improve diagnosis and treatment of cockroach allergic patients. *Methods, 66*(1), 75–85.

Pomés, A., Mueller, G. A., Randall, T. A., Chapman, M. D., & Arruda, I. K. (April 18, 2017). New insights into cockroach allergens. In R. K. Bush & J. A. Woodfold (Section Eds.), *Allergens* (Vol. 17. pp. 25).

Peterson, R. K. D., & Shurdut, B. A. (1999). Human health risk from cockroaches and cockroach management: A risk analysis approach. *American Entomologist, 45*, 142–148.

Roth, L. M. (1985). A taxonomic revision of the genus *Blattella* Caudell (Dictyoptera, Blattaria: Blattellidae). *Scandinavian Entomology*, (Suppl. 22), 221 pp.

Roth, L. M., & Willis, E. R. (1957). The medical and veterinary importance of cockroaches. *Smithsonian Miscellaneous Collections, 134*, 1–147.

Roth, L. M., & Willis, E. R. (1960). The biotic associations of cockroaches. *Smithsonian Miscellaneous Collections, 141*, 1–470.

Rust, M. K., Owens, J. M., & Reierson, D. A. (Eds.). (1995). *Understanding and controlling the German cockroach*. Oxford University Press, 430 pp.

Soldana, N. G., Mendoza, F. J., Larrauri, F. R., Trujillo, D. M., Montoya, E. V., Del LaGarza, E. A., et al. (2017). Bronchopulmonary infection by *Lephomonas blattarum* in a pediatric patient after hematopoietic progenitor cell transplantation: First report in Mexico. *Journal of Thoracic Disease, 9*(10), E899–E902.

Scott, H. G., & Borom, M. R. (1964). *Cockroaches: Key to egg cases of common domestic species*. Atlanta, Georgia: U. S. Dept. of Health, Education and Welfare, Public Health Service, Communicable Disease Center.

Smith, E. H., & Whitman, R. C. (2000). *NPCA field guide to structural pests*. Fairfax, VA: National Pest Management Association.

Solomon, W. R., & Mathews, K. P. (1988). Aerobiology and inhalant allergens. In E. Middleton, Jr., C. E. Reed, E. F. Ellis, N. F. Adkinson, Jr., & J. W. Yunginger (Eds.), *Allergy principles and practice* (3rd ed.). Washington, D.C: C. V. Mosby Company.

Tarshis, I. B. (1962). The cockroach—a new suspect in the spread of infectious hepatitis. *The American Journal of Tropical Medicine and Hygiene, 11*, 705–711.

Wirtz, R. A. (1980). Occupational allergies to arthropods—documentation and prevention. *Bulletin of the Entomological Society of America, 26*, 356–360.

Yang, C.-F., Yang, C.-C., & Wang, I.-J. (2018). Association between allergic diseases, allergic sensitization and attention-deficit/hyperactivity disorder in children: A large-scale, population-based study. *Journal of the Chinese Medical Association, 81*, 277–283.

Zhang, H., Wei, L., Xhang, Z., Liu, S., Shao, G., Xhang, J., et al. (2013). Protective effect of *Periplaneta americana* extract on intestinal mucosal barrier function in patients with sepsis. *Journal of Traditional Chinese Medicine, 33*, 70–73.

Zhang, X., Xu, L., Wang, L. L., et al. (2011). Bronchopulmonary infection with *Lophomonas blattarum*: A case report and literature review. *Journal of International Medical Research, 39*, 944–949.

Zhu, J. J., Yao, S., Guo, X., Yue, B. S., Ma, X. Y., & Li, J. (January 20, 2018). Bioactivity-guided screening of wound-healing active constituents from American cockroach (*Periplaneta americana*). *Molecules, 23*(1). https://doi.org/10.3390/molecules23010101. pii: E101.

This page intentionally left blank

Chapter 7

Lice (Phthiraptera)

Lance A. Durden
Department of Biology, Georgia Southern University, Statesboro, GA, United States

Lice can be a menace to humans, pets, and livestock, not only through their blood-feeding or chewing habits but also because of their ability to transmit pathogens. The human body louse has been indirectly responsible for influencing human history through its ability to transmit the causative agents of epidemic typhus, trench fever, and louse-borne relapsing fever. However, most of the approximately 5,000 known species of lice are ectoparasites of wild birds or mammals and have little or no known medical or veterinary importance.

The Phthiraptera are divided into two main morphologically distinct groups: sucking lice and chewing lice. All sucking lice are obligate, hematophagous ectoparasites of placental mammals, whereas the more diverse chewing lice include species that are obligate associates of birds, marsupials, or placental mammals. Although certain chewing lice imbibe blood, most species ingest host feathers, fur, skin, or skin products. Because of the different feeding strategies of the two groups, the blood-feeding sucking lice are far more important than the chewing lice in transmitting pathogens to their hosts.

TAXONOMY

The Phthiraptera are divided into four suborders (Fig. 7.1, Tables 7.1, 7.2): the **Anoplura** (sucking lice) and the **Amblycera, Ischnocera,** and **Rhynchophthirina** (collectively known as chewing lice or biting lice). Previous classifications treated the Anoplura and Mallophaga as separate orders with the Amblycera, Ischnocera, and Rhynchophthirina all included in the Mallophaga. However, phylogenetic analyses have shown that the chewing lice do not represent a monophyletic group and that some are more closely related to members of the Anoplura than to other chewing lice. Further, sucking and chewing lice originated from a common nonparasitic ancestral group within, the Psocoptera (book lice and bark lice). Some recent molecular phylogenetic analyses embed the parasitic lice within the nonparasitic bark lice and book lice, and some authors now recognize the order **Psocodea,** which includes the parasitic lice as well as the book and bark lice. The parasitic lice diverged from the book and bark lice 100−150 million years ago, and the sucking lice diverged from the chewing lice approximately 77 million years ago and proliferated approximately 65 million years ago to parasitize multiple orders and families of mammals (Light et al., 2010). The fossil record for Phthiraptera is sparse with, at present, only one well-documented specimen, an amblyceran menoponid chewing louse from Germany dated at 44.3 ± 0.4 million years old (Dalgleish et al., 2006).

About 550 species of sucking lice have been described (Durden and Musser, 1994a). The sucking lice are currently assigned to 50 genera and 15 families. Price et al. (2003) recognize 4,464 valid species and subspecies of chewing lice; most of these taxa are associated with birds, but 553 of them (12.4%) parasitize mammals. The chewing lice are divided into three suborders (Table 7.1), 11 families, and 205 genera. According to Price et al. (2003), within the chewing lice, the Amblycera includes seven families, about 76 genera, and about 1,341 species; the Ischnocera includes three families, about 130 genera, and about 3,120 species, and the Rhynchophthirina includes one family, one genus, and three species.

Major taxonomic syntheses for the sucking lice include a series of eight volumes by Ferris (1919−1935) that remains the most comprehensive treatment of this group on a worldwide basis. Ferris (1951) updated much of his earlier work in a shorter overview of the group. Kim et al. (1986) compiled a manual and identification guide for the sucking lice of North America. Durden and Musser (1994a) provide a taxonomic checklist for the sucking lice of the world with host records and the known geographical distributions for each species. Beaucournu (1968) provides an identification guide to the sucking lice of rodents, insectivores, and lagomorphs of Western Europe, and Pajot (2000) provides an identification guide to sucking lice of the Afrotropical region.

Medical and Veterinary Entomology. https://doi.org/10.1016/B978-0-12-814043-7.00007-8

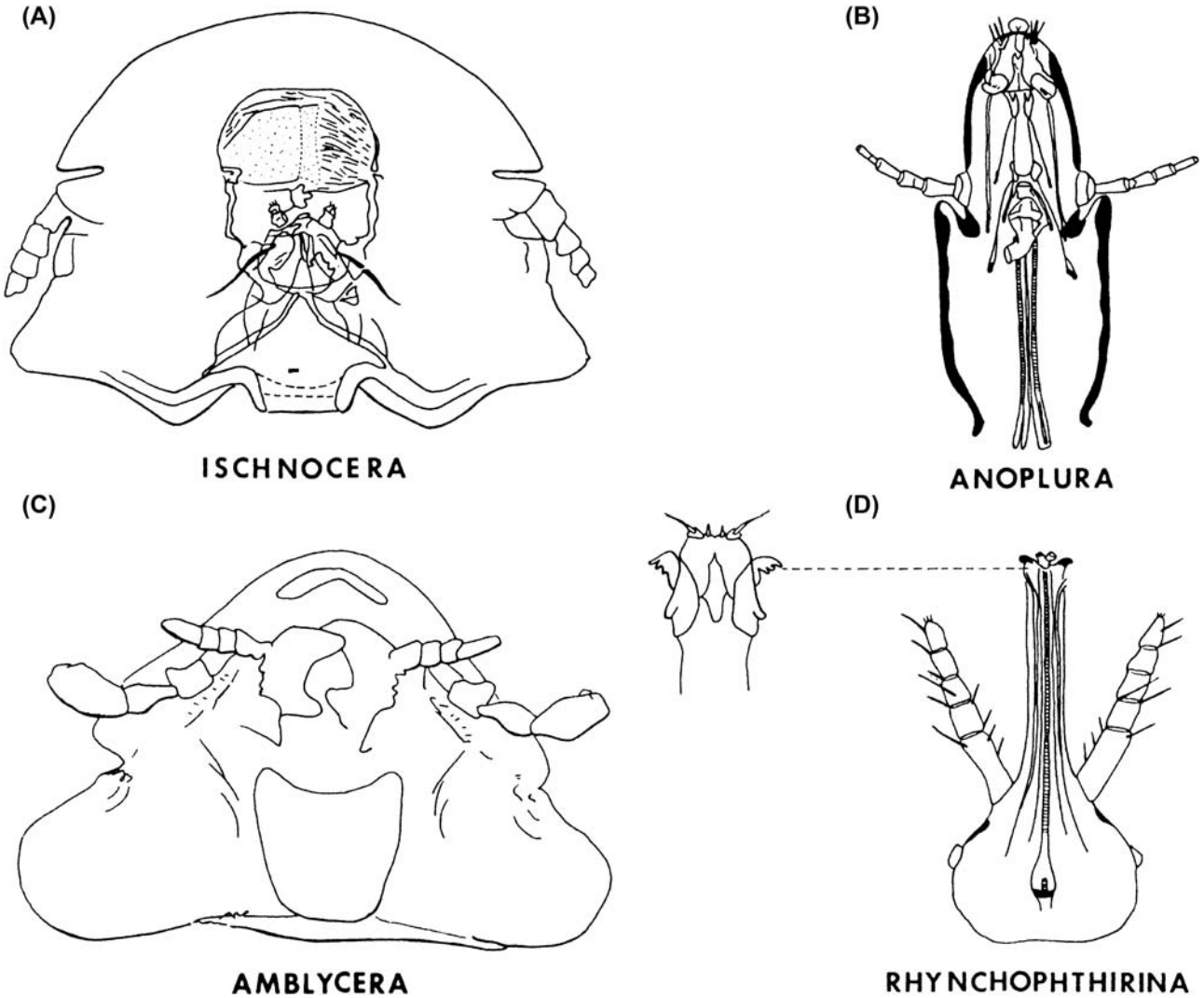


FIGURE 7.1 Head and mouthparts of representatives of each of the four principal groups (suborders) of lice. (A) Ishnocera. (B) Anoplura. (C) Amblycera. (D) Rhynchophthirina. *(A) from Clay, 1938; (B) and (D) from Ferris, 1931; (C) from Bedford, 1932.*

Fewer authoritative identification guides are available for chewing lice. These include a synopsis of the lice associated with laboratory animals (Kim et al., 1973), guides to the lice of domestic animals (Tuff, 1977; Price and Graham, 1997), and an identification guide to the lice of sub-Saharan Africa (Ledger, 1980). These publications provide information on both sucking lice and chewing lice. Checklists of the chewing lice of the world (Price et al., 2003) and of North America (Emerson, 1972) are useful taxonomic references for this group.

Because of the relatively high degree of host specificity exhibited by both chewing and sucking lice, several host–parasite checklists have been prepared. These include a detailed list of both sucking and chewing lice associated with mammals (Hopkins, 1949), a host–parasite list for North American chewing lice (Emerson, 1972), a world host–parasite list for the chewing lice (Price et al., 2003) and a host–parasite checklist for the Anoplura of the world (Durden and Musser, 1994b).

Sucking lice of medical importance are assigned to two families, the Pediculidae and Pthiridae, whereas sucking lice of veterinary importance are assigned to five families, the Haematopinidae, Hoplopleuridae, Linognathidae, Pedicinidae, and Polyplacidae (Table 7.2). Only one species of chewing louse, the dog biting louse, in the family Trichodectidae, has public health importance. Chewing lice of veterinary significance are typically placed in five families: the Boopiidae, Gyropidae, Menoponidae, Philopteridae, and Trichodectidae (Table 7.1).

MORPHOLOGY

Lice are small (0.35–10 mm in the adult stage), wingless, dorsoventrally flattened insects. The elongate abdomen

TABLE 7.1 Classification and Hosts of Chewing Lice of Medical and Veterinary Importance

Lice	Hosts
Suborder Amblycera	
Family Boopiidae	
Heterodoxus spiniger	Dog, other carnivores
Family Gyropidae	
Slender guineapig louse, *Gliricola porcelli*	Guinea pig
Oval guineapig louse, *Gyropus ovalis*	Guinea pig
Family Menoponidae	
Chicken body louse, *Menacanthus stramineus*	Domestic fowl
Shaft louse, *Menopon gallinae*	Domestic fowl
Goose body louse, *Trinoton anserinum*	Geese
Large duck louse, *Trinoton querquedulae*	Ducks
Suborder Ischnocera	
Family Philopteridae	
Slender goose louse, *Anaticola anseris*	Geese
Slender duck louse, *Anaticola crassicornis*	Ducks
Large turkey louse, *Chelopistes meleagridis*	Turkey
Chicken head louse, *Cuclotogaster heterographus*	Domestic fowl
Fluff louse, *Goniocotes gallinae*	Domestic fowl
Brown chicken louse, *Goniodes dissimilis*	Chicken
Large chicken louse, *Goniodes gigas*	Domestic fowl
Wing louse, *Lipeurus caponis*	Domestic fowl
Slender turkey louse, *Oxylipeurus polytrapezius*	Turkey
Family Trichodectidae	
Cattle biting louse, *Bovicola bovis*	Cattle
Goat biting louse, *Bovicola caprae*	Goat
Angora goat biting louse, *Bovicola crassipes*	Goat
Horse biting louse, *Bovicola equi*	Horse
Bovicola limbata	Goat
Donkey biting louse, *Bovicola ocellata*	Donkey
Sheep biting louse, *Bovicola ovis*	Sheep
Cat biting louse, *Felicola subrostrata*	Cats
Dog biting louse, *Trichodectes canis*	Dog, other canids
Suborder Rhynchophthirina	
Family Haematomyzidae	
Elephant louse, *Haematomyzus elephantis*	Elephants

Continued

possesses sclerotized dorsal, ventral, and/or lateral plates in many lice (Fig. 7.2); these provide some rigidity to the abdomen when it is distended by a bloodmeal or other food source. In adult lice the abdomen is 11-segmented and terminates in genitalia and associated sclerotized plates. In females, the genitalia are accompanied by two pairs of finger-like gonopods, which serve to guide, manipulate, and glue eggs onto host hair or feathers. The abdomen is adorned with numerous setae in most lice. Immature lice closely resemble adults (Fig. 7.4) but are smaller, have fewer setae, and lack genitalia. After each nymphal molt, the abdomen is adorned with progressively more setae, and the overall size of the louse increases (Fig. 7.4).

The male genitalia of lice are relatively large, sometimes occupying almost half the length of the abdomen and are conspicuous in cleared, slide-mounted specimens (Figs. 7.15A, 7.16). The terminal, extrusable, sclerotized pseudopenis (=aedeagus) is supported anteriorly by a basal apodeme. Laterally, it is bordered by a pair of chitinized parameres. Two or four testes are connected to the vas deferens, which coalesce posteriorly to form the vesicula seminalis. In the female, the vagina leads to a large uterus to which several ovarioles supporting eggs in various stages of development are connected by the oviducts. Two or more large accessory glands that secrete materials to attach or coat the eggs and a single spermatheca, in which sperm is stored after mating, are situated posteriorly in the abdomen. Except for the human body louse, all lice cement their eggs, called **nits**, onto the hair or feathers of their host. Eggs are usually subcylindrical with rounded ends and a terminal cap, the operculum (Fig. 7.3). On the top of the operculum is a patch of holes or areas with thin cuticle, called aeropyles, through which the developing embryo respires. Most of the egg is heavily chitinized, which helps to protect the embryo from mechanical damage and desiccation (and from insecticides in many cases). A suture of thin cuticle encircles the base of the operculum. At the time of hatching, the first-instar nymph emerges from the egg by cracking this suture and pushing off the operculum.

TABLE 7.2 Classification and Hosts of Sucking Lice (Anoplura) of Medical and Veterinary Importance

Lice	Hosts
Family Echinophthiriidae	
Echinophthirius horridus	Harbor seals
Family Haematopinidae	
Horse sucking louse, *Haematopinus asini*	Horse, donkey
Shortnosed cattle louse, *Haematopinus eurysternus*	Cattle
Cattle tail louse, *Haematopinus quadripertusus*	Cattle
Hog louse, *Haematopinus suis*	Swine
Buffalo louse, *Haematopinus tuberculatus*	Asiatic buffalo, cattle
Family Hoplopleuridae	
Hoplopleura captiosa	House mouse
Tropical rat louse, *Hoplopleura pacifica*	Domestic rats
Family Linognathidae	
African blue louse, *Linognathus africanus*	Goat, sheep, deer
Sheep face louse, *Linognathus ovillus*	Sheep
Sheep foot louse, *Linognathus pedalis*	Sheep
Dog sucking louse, *Linognathus setosus*	Dog, other canids
Goat sucking louse, *Linognathus stenopsis*	Goat
Longnosed cattle louse, *Linognathus vituli*	Cattle
Little blue cattle louse, *Solenopotes capillatus*	Cattle
Family Pedicinidae	
Pedicinus spp.	Old World Primates
Family Pediculidae	
Head louse, *Pediculus humanus capitis*	Human
Body louse, *Pediculus humanus humanus*	Human
Family Polyplacidae	
Rabbit louse, *Haemodipsus ventricosus*	Domestic rabbit
Mouse louse, *Polyplax serrata*	House mouse
Spined rat louse, *Polyplax spinulosa*	Domestic rats
Family Pthiridae	
Crab louse, *Pthirus pubis*	Human

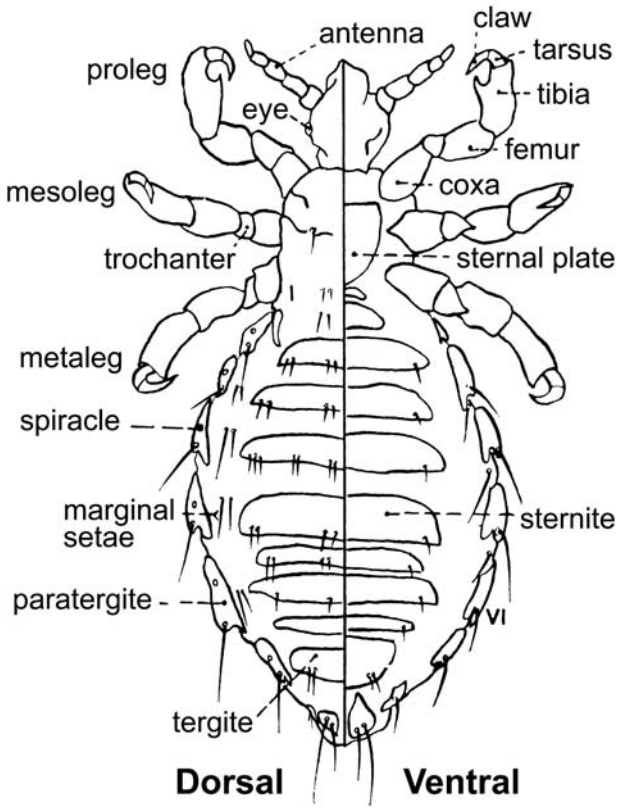


FIGURE 7.2 A generalized sucking louse (Anoplura), showing dorsal (left) and ventral (right) morphology. *From Ignoffo (1959).*

In amblyceran and ischnoceran chewing lice, the head is broader than the thorax (Figs. 7.11A, 7.12, 7.15A,B, 7.17A–F). Amblyceran chewing lice have four-segmented antennae and have retained distinct maxillary palps characteristic of their psocopteran ancestors (Fig. 7.1C). However, ischnoceran chewing lice have three to five antennal segments and lack maxillary palps (Fig. 7.1A). In the Amblycera, the antennae are concealed in lateral grooves (Fig. 7.1C), whereas in the Ischnocera and Rhynchophthirina, the antennae are free from the head (Figs. 7.1A,D, 7.5).

There is a gradation in the specialization of the mouthparts and of the internal skeleton of the head, or tentorium, from the psocopteran ancestor of the parasitic lice through the Amblycera, Ischnocera, Rhynchophthirina, and Anoplura. Although all chewing lice possess chewing mouthparts, the mechanics of these mouthparts differ for different groups. For example, members of the Rhynchophthirina possess tiny mandibles that are situated at the tip of an elongated rostrum (Figs. 7.1D, 7.5). Also, through extreme modifications, members of the chewing louse genus *Trochilocoetes* (parasites of humming birds) have evolved mouthparts that can function as sucking organs.

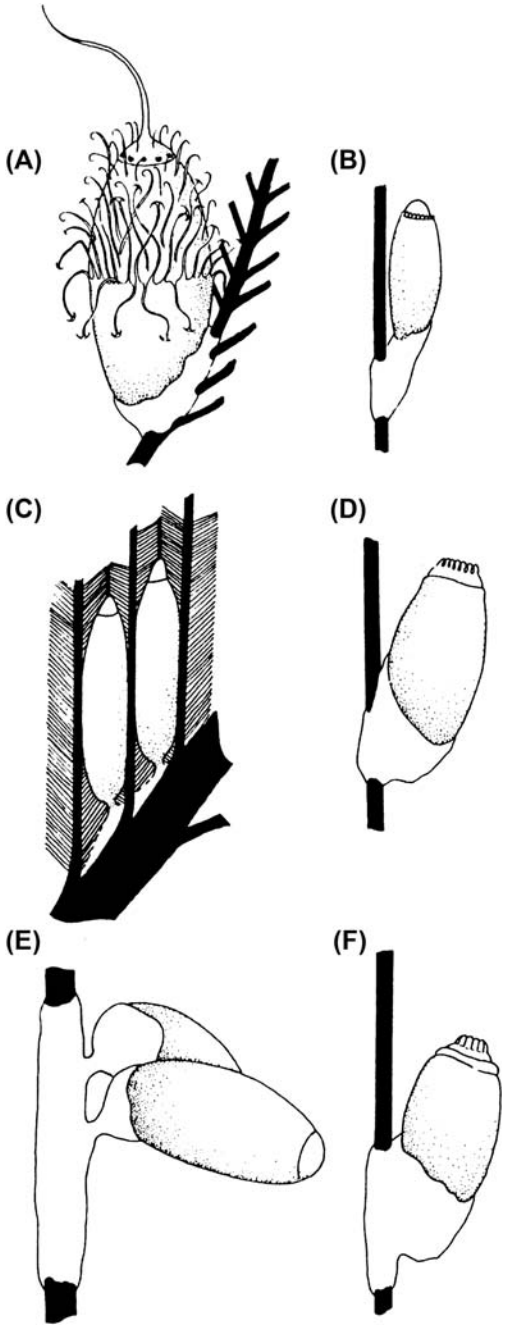


FIGURE 7.3 Eggs (nits) of representative lice. (A) *Menacanthus stramineus* (Amblycera). (B) *Gyropus ovalis* (Amblycera). (C) *Columbicola columbae* (Ischnocera). (D) *Bovicola bovis* (Ischnocera). (E) *Haematomyzus elephantis* (Rhynchophthirina). (F) *Pediculus humanus capitis* (Anoplura). *Modified from Marshall (1981).*

The thorax in chewing lice usually appears dorsally as two, occasionally three, segments. Chewing lice possess one or two simple claws on each leg; species that parasitize birds, typically have two claws.

In sucking lice, the head is slender and narrower than the thorax (Figs. 7.2 and 7.6). Anoplura have three- to five-segmented antennae and lack maxillary palps. Noncompound eyes, which represent groups of ocelli, are reduced or absent in most sucking lice but are well developed in members of the medically important genera *Pediculus* (Figs. 7.6, 7.7 and 7.9) and *Pthirus* (Fig. 7.10). **Ocular points**, or eyeless projections posterior to the antennae, are characteristic of sucking lice in the genus *Haematopinus* (Figs. 7.11D, 7.13 and 7.14).

As indicated by their name, anopluran mouthparts function as sucking devices during blood feeding (Fig. 2.2F). At rest, the mouthparts are withdrawn into the head and are protected by the snoutlike haustellum, representing the highly modified labrum. The haustellum is armed with tiny recurved teeth that hook into the host skin during feeding. The stylets, consisting of a serrated labium, the hypopharynx, and two maxillae, then puncture a small blood vessel (Fig. 2.2F). The hypopharynx is a hollow tube through which saliva (containing anticoagulants and enzymes) is secreted. The maxillae oppose each other and are curved to form a food canal through which host blood is imbibed (Fig. 2.2F).

In sucking lice, all three thoracic segments are fused and appear as one segment. In most species, the legs, especially the hindlegs and midlegs, terminate in highly specialized claws for grasping the host pelage. These **tibiotarsal claws** consist of a curved tarsal element that opposes a tibial spur (Fig. 2.4C,D) to enclose a space that typically corresponds to the diameter of the host hair.

The internal anatomy of lice is best known for the human body louse. As in most hematophagous insects, strong cibarial and esophageal muscles produce a sucking action during blood feeding. The esophagus leads to a spacious midgut composed primarily of the ventriculus. The posterior region of the midgut is narrow and forms a connection between the ventriculus and the hindgut. Ventrally, **mycetomes** (also called bacteriomes or stomach discs by some authors) containing symbiotic microorganisms connect to the ventriculus. These obligate symbiotes synthesize B vitamins that are lacking in the bloodmeal.

LIFE HISTORY

Lice are hemimetabolous insects. Following the egg stage, there are three nymphal instars, the last of which molts to an adult (Fig. 7.4). Although there is wide variation between species, the egg stage typically lasts for 4–15 days and each nymphal instar for 3–8 days, and adults live for up to 35 days. Under optimal conditions, many species of lice can complete 10–12 generations per year, but this is rarely achieved in nature. Host grooming, immune

FIGURE 7.4 Active life stages of the body louse (*Pediculus humanus humanus*). From left to right: N1, first-instar nymph; N2, second-instar nymph; N3, third-instar nymph; adult male; and adult female. Scale bar = 1 mm. Note: Gut has ruptured in the first-instar nymph and the bloodmeal has disseminated throughout its body. *Photograph by James Gathany, Public Health Image Library, Centers for Disease Control and Prevention, Atlanta, Georgia, USA.*

responses, molting or feather loss, hibernation, hormonal changes, as well as predators (especially insectivorous birds on large ungulates), parasites and parasitoids, and unfavorable weather conditions can reduce the number of louse generations.

Fecundities for fertilized female lice vary from 0.2 to 10 eggs per day. Males are unknown in some parthenogenetic species such as the *Damalinia* sp. louse that causes hair-loss syndrome in North American deer and typically constitute less than 5% of adults in the cattle biting louse (*Bovicola bovis*) and less than 1% in the horse biting louse (*Bovicola equi*).

FIGURE 7.5 Elephant louse (*Haematomyzus elephantis*), female; stacked image of cleared specimen. *Photograph by Lorenza Beati and Lance A. Durden.*

BEHAVIOR AND ECOLOGY

Blood from the host is essential for the successful development and survival of all sucking lice. Anoplura are vessel feeders, or **solenophages**, that imbibe blood through a hollow dorsal stylet derived from the hypopharynx (Fig. 2.2F). Contractions of powerful cibarial and pharyngeal muscles create a sucking reaction for imbibing blood.

Chewing lice feed by the biting or scraping action of the mandibles. Bird-infesting chewing lice typically use their mandibles to sever small pieces of feather, which drop onto the labrum and then are forced into the mouth. Chewing lice that infest mammals use their mandibles in a similar manner to feed on host fur. Many chewing lice that infest birds and mammals can also feed on other integumental products such as skin debris and secretions. Some species of chewing lice are obligate, or more frequently facultative, hematophages. Species of chewing lice that imbibe blood typically scrape the host integument until it bleeds. The rhynchophthirinan *Haematomyzus elephantis* (Fig. 7.5), which parasitizes both African and Asian elephants, feeds in this manner.

Symbionts are thought to be present in all lice that imbibe blood. Symbionts in the mycetomes (also called bacteriomes or stomach discs) synthesize vitamins essential to growth and reproduction, and lice deprived of them die after a few days; female lice lacking symbionts also become sterile. Experimentally administering antibiotics to bloodmeals of human head and body lice kills symbiotic bacteria in the mycetomes and eventually results in death of the lice. In female human body lice, some symbionts migrate to the ovary, where they are transferred transovarially to the next generation of lice.

Many lice exhibit host specificity, some to such a degree that they parasitize only one species of host. The hog louse (Fig. 7.14), slender guineapig louse, large turkey louse, and several additional species listed in Tables 7.1 and 7.2 all are typical parasites of a single host species. Host specificity is less stringent in some lice. For example, some lice of veterinary importance parasitize two or more closely related hosts. Examples include the three species that parasitize domestic dogs: *Trichodectes canis* (Fig. 7.15A), *Heterodoxus spiniger* (Fig. 7.15B), and *Linognathus setosus* (Fig. 7.15C). These lice also parasitize

foxes, wolves, coyotes, and occasionally other carnivores. Similarly, the horse sucking louse (*Haematopinus asini*) (Fig. 7.13) parasitizes horses, donkeys, asses, mules, and, sometimes, zebras, whereas the African blue louse, *Linognathus africanus*, parasitizes both sheep and goats. At least six species of chewing lice are commonly found on domestic fowl, most of them parasitizing chickens, but some also feeding on turkeys, guinea fowl, pea fowl, or pheasants (Fig. 7.17, Table 7.1). Lice found on atypical hosts are termed **stragglers**.

Some sucking lice, such as the three forms that parasitize humans, the sheep foot louse, and sheep face louse, not only are host specific but also infest specific body areas from which they can spread in severe infestations. Many chewing lice, particularly species that parasitize birds, also exhibit both host specificity and site specificity; examples include several species that are found on domestic fowl and species confined to turkeys, geese, and ducks (Table 7.1). Lice inhabiting different body regions on the same host typically have evolved morphological adaptations in response to specific attributes of the host site. These include characteristics such as morphological differences of the pelage, thickness of the skin, availability of blood vessels, and grooming or preening activities of the host. Site specificity in chewing lice is most prevalent in the more sedentary, specialized ischnocerans than in the mostly mobile, morphologically unspecialized amblycerans. For example, on many bird hosts, round-bodied ischnocerans with large heads and mandibles are predominately found on the head and neck. Elongate forms with narrow heads and small mandibles tend to inhabit the wing feathers, whereas morphologically intermediate forms occur on the back and other parts of the body.

Some chewing lice inhabit highly specialized host sites. These include members of the amblyceran genus *Piagetiella*, which are found inside the oral pouches of pelicans, and members of several amblyceran genera, including *Actornithophilus* and *Colpocephalum*, which live inside feather quills. Several bird species are parasitized by five or more different species of site-specific chewing lice, and up to 15 species (belonging to three families and 12 genera) may be found on the great tinamou (*Tinamus major*), a bird native to the Neotropical region.

With the exception of the three kinds of lice that parasitize humans, site specificity is less well documented for sucking lice. However, domestic cattle may be parasitized by as many as five sucking louse species, each predominating on particular parts of the body. Similarly, some Old World squirrels and rats can support up to six species of sucking lice, often on different parts of the body.

Because of the importance of maintaining a permanent or close association with the host, lice have evolved specialized host-attachment mechanisms to resist grooming activities of the host. The robust tibiotarsal claws of sucking lice (Fig. 2.4C,D) are very important in securing them to their hosts. Various arrangements of hooks and spines, especially on the head of lice that parasitize arboreal or flying hosts, such as squirrels and birds, also aid in host attachment. Mandibles are important attachment appendages in ischnoceran and rhynchophthirinan chewing lice. In some species of *Bovicola*, a notch in the first antennal segment encircles a host hair to facilitate attachment. A few lice even possess ctenidia ("combs") that are convergently similar in morphology to those characteristic of many fleas. They occur most notably among lice that parasitize coarse-furred, arboreal, or flying hosts. Additionally, chewing lice that parasitize arboreal or flying hosts often have larger, more robust claws than do their counterparts that parasitize terrestrial hosts.

Because of their reliance on host availability, lice are subjected to special problems with respect to their long-term survival. All sucking lice are obligate blood-feeders; even a few hours away from the host can prove fatal to some species. Some chewing lice also are hematophages and, similarly, cannot survive prolonged periods off the host. However, many chewing lice, particularly those that subsist on feathers, fur, or other skin products, can survive for several days away from the host. For example, the cattle biting louse can survive for up to 11 days (this species will feed on host skin scrapings) and *Menacanthus* spp. of poultry for up to 3 days off the host. Off-host survival is generally greater at low temperatures and high humidities. At 26°C and 65% relative humidity (RH), 4% of human body lice die within 24 h, 20% die within 40 h, and 84% die within 48 h. At 75% RH, a small proportion of the sheep foot louse survive for 17 days at 2°C, whereas most die within 7 days at 22°C. Recently fed lice generally survive longer than do unfed lice away from the host. Although most lice are morphologically adapted for host attachment and are disadvantaged when dislodged, the generalist nature of some amblyceran chewing lice better equips them for locating another host by crawling across the substrate. Amblycerans are more likely than other lice to be encountered away from the host, accounting for observations of these lice crawling on bird eggs or in unoccupied nests and roosts.

Host grooming is an important cause of louse mortality. Laboratory mice infested by the mouse louse, *Polyplax serrata*, for example, usually limit their louse populations to 10 or fewer individuals per mouse by regular grooming. Prevention of self or mutual grooming by impaired preening action of the teeth or limbs of mice can result in heavy infestations of more than 100 lice. Similarly, impaired preening due to beak injuries in birds can result in tremendous increases in louse populations (Clayton et al. 2015). Host biting, scratching, rubbing, and licking can also reduce louse populations on several domestic animals.

Whereas most species of lice on small and medium-sized mammals exhibit only minor seasonal differences in population levels, some lice associated with larger animals show clear seasonal trends. Some of these population changes have been attributed to host molting, fur density and length, hormone levels in the bloodmeal, or climatological factors such as intense summer heat, sunlight, or desiccation. On domestic ungulates in temperate regions, louse populations typically peak during the winter or early spring and decline during the summer. An exception to this trend is the cattle tail louse, *Haematopinus quadripertusis,* in which populations peak during the summer.

Another important aspect of louse behavior is the mode of transfer between hosts. Direct host contact appears to be the primary mechanism for louse exchange. Transfer of lice from an infested mother to her offspring during suckling (in mammals) or during nest sharing (in birds and mammals) is an important mode of transfer. Several species of lice that parasitize livestock transfer during suckling, including the sheep face louse and the sheep biting louse, both of which move from infested ewes to their lambs at this time. Lice can also transfer during other forms of physical contact between hosts such as mating or fighting. Transfer of lice between hosts also can occur between hosts that are not in contact. The sheep foot louse, for example, can survive for several days off the host and reach a new host by crawling across pasture land. Nests of birds and mammals can act as foci for louse transfer but these are infrequent sites of transfer.

Dispersal of some lice occurs via **phoresy** in which the lice temporarily attach to other arthropods and are carried from one host to another. During phoresy, most lice attach to larger, more mobile blood-feeding arthropods, usually a fly such as a hippoboscid or muscoid. Phoresy is particularly common among ischnoceran chewing lice. Once thought to represent an anecdotal association between two groups of organisms, phoresy of certain species of chewing lice on hippoboscid flies is now considered to be an important mechanism for dispersal and host location by these lice. Phoresy is relatively rare among sucking lice. This is probably because attachment to the fly is achieved by the less efficient mechanism of grasping with the tibio-tarsal claws.

Mating in lice occurs on the host. It is initiated by the male pushing his body beneath that of the female and curling the tip of his abdomen upward. In the human body louse, the male and female assume a vertical orientation along a hair shaft with the female supporting the weight of the male as he grasps her with his anterior claws. Other lice appear to exhibit similar orientation behavior during mating. Notable exceptions include the crab louse of humans in which both sexes continue to clasp with their tibio-tarsal claws a host hair, rather than each other, during mating and the hog louse in which the male strokes the head of the female during copulation. Some male ischnoceran chewing lice possess modified hooklike antennal segments with which they grasp the female during copulation.

Oviposition behavior by female lice involves crawling to the base of a host hair or feather and cementing one egg at a time close to the skin surface. Two pairs of finger-like gonopods direct the egg into a precise location and orientation as a cement substance is secreted around the egg and hair or feather base. Optimal temperature requirements for developing louse embryos inside eggs are very narrow, usually within a fraction of a degree, such as may occur on a precise area on the host body. For this reason, female lice typically oviposit preferentially on an area of the host that meets these requirements.

LICE OF MEDICAL IMPORTANCE

Three taxa of sucking lice parasitize humans throughout the world: the **body louse, head louse**, and **crab louse** (= pubic louse). All are specific ectoparasites of humans; rarely, dogs or other companion animals may have temporary, self-limiting infestations.

Human head and body lice are closely related and can interbreed to produce fertile offspring in the laboratory. For this reason, they are often treated as separate subspecies of *Pediculus humanus*, as they are in this chapter. Nevertheless, they rarely interbreed in nature, which has prompted some epidemiologists to treat them as separate species: *Pediculus humanus* (body louse) and *P. capitis* (head louse). Recent publications based on gene sequences have provided conflicting evidence for the recognition of human head and body lice as either separate species, subspecies or strains of a single species. One intriguing hypothesis suggests that body lice "emerge" from head louse populations under conditions of poor human hygiene (Li et al., 2010). In partial support of this idea, head louse populations can be assigned to three different phylotypes—A, B, and C, whereas body lice are all assigned to phylotype A.

Recent genetic analyses of human lice have produced indirect evidence for important events during human history (Reed et al., 2004). For example, analysis of two separate head louse genetic lineages suggests that *Homo sapiens* and *Homo erectus* physically interacted at some time during prehistory and that both louse lineages infested *H. sapiens* when *H. erectus* became extinct. Similarly, genetic analysis of crab lice show that humans acquired their pubic lice from gorillas about 3.3 million years ago; gorilla and human crab lice have since evolved into distinct species. Further, because human body lice infest clothing, the origin of body lice on humans might correspond with the time when humans first starting wearing clothes; one study along these lines suggests that this occurred $72{,}000 \pm 42{,}000$ years ago.

Human Body Louse (*Pediculus humanus humanus*)

Infestations with the human body louse are sometimes referred to as *pediculosis corporis*. The human body louse, or **cootie**, was once an almost ubiquitous companion of humans. Today it is less common, especially in developed nations. Body lice persist as a significant problem in less developed nations in parts of Africa, Asia, and Central and South America and on populations of some homeless people worldwide. This is significant because *P. h. humanus* (Figs. 7.4, 7.6 and 7.7) is the only louse of humans that is known to naturally transmit pathogens. The large-scale reduction in body louse infestations worldwide has led to a concomitant decrease in the prevalence of human louse-borne diseases. However, situations that result in human overcrowding and unsanitary conditions (e.g., wars, famines, natural disasters, homelessness) can lead to a resurgence of body louse infestations, often accompanied by one or more louse-borne diseases.

Adult human body lice are 2.3−3.6 mm long. Under optimal conditions their populations can multiply dramatically if unchecked (e.g., if clothes of infested individuals are not changed and washed in hot water at regular intervals). In unusually severe infestations, populations of more than 30,000 body lice on one person have been recorded. Body lice typically infest articles of clothing and crawl onto the body only to feed. Females lay an average of four or five eggs per day, and these typically hatch after 8 days. Unique among lice, females oviposit not on hair but on clothing (Fig. 7.8), especially along seams and creases. Each nymphal instar lasts for 3−5 days, and adults can live for up to 30 days.

Biting by body lice often causes irritation, with each bite site typically developing into a small red papule with a tiny central clot. The bites usually itch for several days but occasionally for a week or longer. Persons exposed to numerous bites over long periods often become desensitized and show little or no reaction to subsequent bites. Persons with chronic body louse infestations may develop a generalized skin thickening and discoloration called **Vagabond's disease** or **Hobo's disease**, names depicting a lifestyle that can promote infestation by body lice. Several additional symptoms may accompany chronic

FIGURE 7.7 Human body louse (*Pediculus humanus humanus*) feeding on a human. *Photograph by James Gathany, Public Health Image Library, Centers for Disease Control and Prevention, Atlanta, Georgia, USA.*

FIGURE 7.6 Human body louse (*Pediculus humanus humanus*), female; stacked image of cleared specimen. *Photograph by Lorenza Beati and Lance A. Durden.*

FIGURE 7.8 Eggs of the human body louse (*Pediculus humanus humanus*) attached to clothing. *Photograph by Elton J. Hansens.*

infestations; these include lymphadenitis (swollen lymph nodes), edema, increased body temperature often accompanied by fever, a diffuse rash, headache, joint pain, and muscle stiffness.

Some people develop allergies to body lice. Occasionally, patients experience a generalized dermatitis in response to just one bite or a small number of bites. A form of asthmatic bronchitis has similarly, been recorded in response to louse infestation allergies. Secondary infections such as impetigo or (rarely) septicemia (blood poisoning) can also result from body louse infestations.

Body lice tend to leave persons with elevated body temperatures and may crawl across the substrate to infest a nearby person. This has epidemiological significance because high body temperatures of lousy persons often result from fever caused by infection with louse-borne pathogens.

Human Head Louse (*Pediculus humanus capitis*)

Infestations with head lice can be referred to as *pediculosis capitis*. The human head louse (Fig. 7.9) is virtually indistinguishable from the human body louse on the basis of morphological characters and its life cycle. Generally, adult head lice are slightly smaller (2.1−3.3 mm in length) than body lice and tend to feed more frequently.

As indicated by their name, human head lice typically infest the scalp and head region. Females attach their eggs to the base of individual hairs. As the hair grows, the eggs become more distant from the scalp. An indication of how long a patient has been infested can be gleaned by measuring the farthest distance of eggs from the scalp and comparing this with the growth rate of hair—about 0.5 inch (1.27 cm) per month.

Today, head lice are far more frequently encountered than are body lice, especially in developed countries. Transmission occurs by person-to-person contact and via shared objects such as combs, brushes, headphones, and caps. School-aged children are at high risk because they are more likely to share these items. About 8% of all children 3−12 years old in the United States are infested with head lice, but in some school districts in the United States, Britain, and France, for example, infestation prevalence approaches 50%. It has been estimated that 6−12 million people, principally children, are infested with head lice annually in the United States. Some ethnic groups, such as persons of recent African origin, have coarser head hairs and are less prone to head louse infestations. The reason for this is that the tibiotarsal claws of these lice cannot efficiently grip the thicker hairs.

FIGURE 7.9 Human head louse (*Pediculus humanus capitis*) (nymph). *Photograph by Nathan D. Burkett-Cadena.*

Although head lice are not typically important in transmitting pathogens, they can mechanically transmit the bacteria *Staphylococcus aureus* and *Streptococcus pyogenes*, both of which can cause skin/tissue infections. Under optimal laboratory conditions, head lice can transmit the causative agent of epidemic typhus, *Rickettsia prowazekii*, but only human body lice are implicated as vectors of this agent in nature. Although *Bartonella quintana,* the causative agent of trench fever, has been detected in head lice from various parts of the world, there is no current evidence of transmission of this pathogen by this louse in nature. The same is true for the emerging pathogenic bacterium *Acinetobacter baumannii*, which has been detected in head lice. Heavy infestations of head lice can cause severe irritation. As is the case with human body lice, the resultant scratching often leads to secondary infections such as impetigo, pyoderma, or (rarely) septicemia. Severe head louse infestations occasionally result in the formation of scabby crusts beneath which the lice tend to aggregate. Enlarged lymph nodes in the neck region may accompany large infestations.

Human Crab Louse (*Pthirus pubis*)

Infestations with crab lice can be referred to as *pediculosis inguinalis* or *pthiriasis*. The crab louse, or pubic louse (Fig. 7.10), is a medium-sized (1.1−1.8 mm long), squat louse, with robust tibiotarsal claws used for grasping thick hairs, especially those in the pubic region. It also may infest coarse hairs on other parts of the body, such as the eyebrows, eyelashes, chest hairs, beards, moustaches, and armpits. This louse typically transfers between human partners during sexual intercourse and other intimate contact; in France, crab lice are sometimes described as "papillons d'amour" (butterflies of love). Transfer via infested bed linen, sofas, or toilet seats can also occur. This is uncommon, however, because crab lice can survive for only a few hours off the host.

Female crab lice lay an average of three eggs per day. Eggs hatch after 7−8 days; the three nymphal instars combined last for 13−17 days. Under optimal conditions,

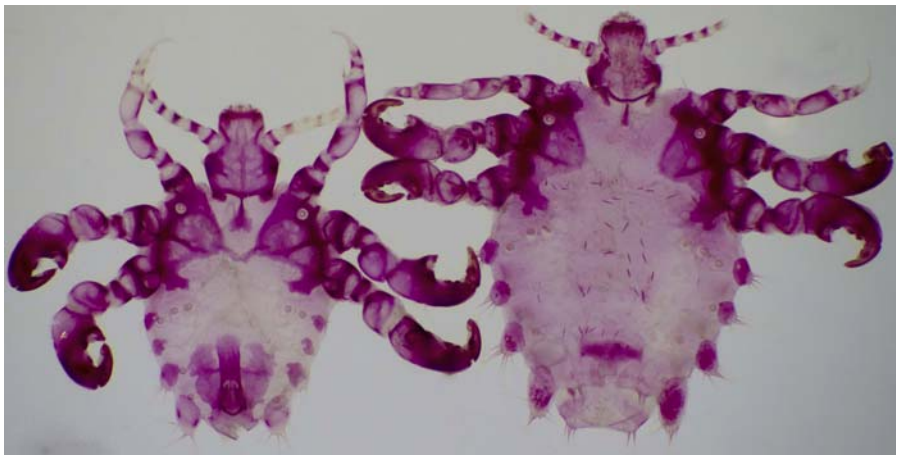


FIGURE 7.10 Crab louse (pubic louse) (*Pthirus pubis*), male (left) and female; stacked image of cleared specimens stained with acid fuschin. *Photograph by Lorenza Beati and Lance A. Durden.*

the generation time is 20–25 days. The intense itching caused by these lice is often accompanied by purplish lesions at bite sites and by small blood spots from squashed lice or louse feces on underwear. Crab lice are widely distributed throughout the world. They are not known to transmit any pathogens.

The generic name *Pthirus* is the taxonomically correct spelling as placed on the "Official List of Generic Names in Zoology" (1958, Opinion 104) with both *Phthirus* and *Phthirius* officially treated as invalid emendations of the original spelling *Pthirus* (see Kim et al., 1986). The correct family name is Pthiridae (not Phthiridae), following the valid generic spelling.

LICE OF VETERINARY IMPORTANCE

A variety of lice infests domestic livestock, poultry, pets, and laboratory animals (Tables 7.1, 7.2). Small rodents usually support few, if any, lice, whereas larger hosts such as livestock animals, including poultry, may be parasitized by extremely large numbers of lice. For example, fewer than 10 mouse lice (*Polyplax serrata*) on a house mouse is a typical burden, but 0.5–1.0 million sheep biting lice (*Bovicola ovis*) may be present on one heavily infested sheep. Although many species of wildlife have their own species of lice, seldom are they a problem. Relatively few lice species of veterinary concern are vectors of pathogens.

Lice of Cattle

Cattle lice cause economic problems worldwide. Both dairy and beef breeds are affected. Domestic cattle are typically parasitized by one species each of *Haematopinus*, *Linognathus*, *Solenopotes*, and *Bovicola*. Domestic Asiatic buffalo are typically parasitized by *Haematopinus tuberculatus* (Table 7.2), which has also successfully transferred to cattle in tropical climates.

The cosmopolitan cattle biting louse (*Bovicola bovis*) (Fig. 7.11A) is the only species of chewing louse that infests cattle. This species is primarily parthenogenetic, but males are occasionally seen. The adult female is about 1.7 mm in length. Females lay an average of 0.7 egg per day, which hatch 7–10 days later. Nymphal instars last 5–7 days each, and adult longevity can be as long as 10 weeks. The preferred host site for this louse is the top line of the back, especially the withers area from which it spreads to the rump and poll area (Watson et al., 1997). In heavy infestations, these lice spread to other body regions. In the most severe infestations, lice may be found beneath heavily encrusted scurf.

The longnosed cattle louse (*Linognathus vituli*) (Fig. 7.11B) is also a worldwide pest. Adult females and males are about 2.4 and 1.8 mm in length, respectively. Females deposit one egg per day, and the life cycle is completed in approximately 21 days. This louse occurs in greater numbers on calves than on mature cattle. The species is widely distributed over the body of the host, but preferred infestation sites are the shoulder, back, neck, and dewlap.

The little blue cattle louse (*Solenopotes capillatus*) (Fig. 7.11C) is also worldwide in distribution. It is a common species on cattle but, because of its small size (adult female 1.5 mm; adult male 1.1 mm) it is commonly mistaken for nymphs of the longnosed cattle louse, which are also blueish in color. Females lay one or two eggs per day. Eggs hatch after about 12 days, and the time from egg to egg is about 28 days. Infestation by *S. capillatus* is usually noticed when dark blue patches, representing aggregations of this louse, appear on the face of the host (i.e., muzzle, cheeks, and around the eyes). Occasionally, the longnosed cattle louse may also be seen within these

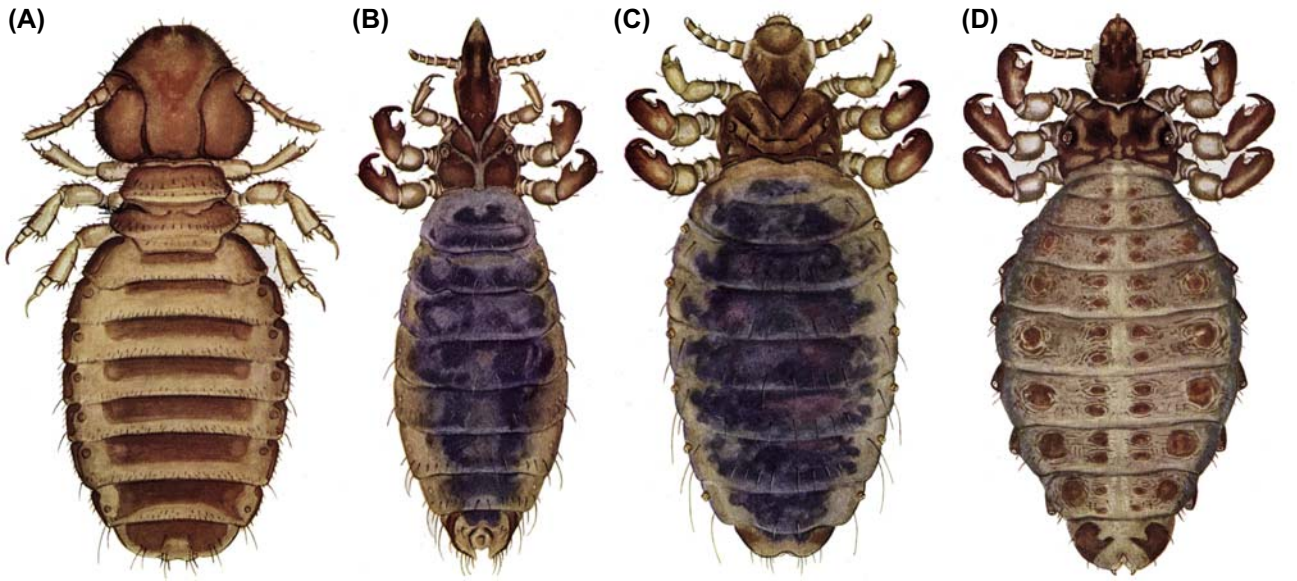


FIGURE 7.11 Lice (all females) of cattle. (A) Cattle biting louse (*Bovicola bovis*). (B) Longnosed cattle louse (*Linognathus vituli*). (C) Little blue cattle louse (*Solenopotes capillatus*). (D) Shortnosed cattle louse (*Haematopinus eurysternus*). *From Matthysse, 1946; original illustrations by Ellen Edmonson.*

clusters. As spring approaches, heavier infestations of the little blue cattle louse may extend to the neck and dewlap.

The cosmopolitan shortnosed cattle louse (*Haematopinus eurysternus*) (Fig. 7.11D) is the largest louse found on cattle in northern states of the U.S. Adult females and males measure 2.9 and 2.3 mm in length, respectively. The female lays an average of 1.4 eggs per day for about 2 weeks, nymphs reach adulthood in about 14 days, and average adult longevity is 10 days for males and 15 days for females. The life cycle from egg to egg normally requires 28 days. Preferred infestation sites are the top of the neck, the dewlap, and the brisket. In severe infestations, the entire region from the base of the horns to the base of the tail can be infested. In warmer weather, this species can also be found abundantly inside and on the tips of the ears. Although heavy infestations are occasionally encountered, the shortnosed cattle louse is the least common species on cattle in Wyoming, Nebraska, and, probably, the neighboring Rocky Mountain and Great Plains states.

The cattle tail louse (*Hematopinus quadripertusus*) is a tropical sucking louse that was inadvertently introduced into Florida in 1945. It has since spread to warmer regions of the United States, including Florida, the Gulf Coast states, and southern California. The cattle tail louse is larger than the closely related short nosed cattle louse. Adult females are 4.0 mm and adult males are 3.2 mm, respectively. Unlike other cattle lice in North America, *H. quadripertusus* is most abundant during the summer. Adult females of this louse, which are normally found on the distal area of the tail, oviposit on the tail hairs. Hatching may be delayed 40+ days in January and February due to cool weather. Consequently the tail brush becomes matted with eggs that hatch when temperatures begin to rise in the spring. In severe infestations, hair may be shed. Eggs hatch after 9 days under optimal conditions, and the entire life cycle can be as short as 25 days. Nymphs migrate over the host body surface. but adults are typically confined to the tail. Although the cattle tail louse is spread via direct contact or contaminated facilities and equipment, phoresy appears to be common. Third-instar nymphs may migrate to the backs and shoulders of the host, where they attach to flies and are carried to a new host. In a sample of 5,000 horn flies collected in Florida, 100 were carrying *H. quadripertusus* nymphs (Kaufman et al., 2005).

Except for *H. quadripertusus*, cattle lice increase in numbers during the winter and early spring in temperate regions. Chronically infested cattle, often referred to as carriers, may have heavy burdens of lice, even in the summer. Cattle producers customarily cull these animals from a herd as they are considered a source of lice for the other animals. Thorough examination of cattle with no clinical signs of lice infestation in the summer, however, has shown that these animals also may harbor all four species of cattle lice (Hanlin, 1994).

Lice of Other Livestock Animals

Horses, donkeys, hogs, goats, and sheep are all parasitized by one or more species of louse (Tables 7.1, 7.2). Except for hogs, all of these animals are parasitized by at least one species of sucking lice and one species of chewing lice. The horse biting louse (*Bovicola equi*) (Fig. 7.12) is the most important louse of equids worldwide. Adult females and males average approximately 1.9 and 1.3 mm, respectively.

FIGURE 7.12 Horse biting louse (*Bovicola equi*), female; stacked image of cleared specimen. *Photograph by Lorenza Beati and Lance A. Durden.*

FIGURE 7.13 Horse sucking louse (*Haematopinus asini*), female; stacked image of cleared specimen. *Photograph by Lorenza Beati and Lance A. Durden.*

Females of this louse oviposit on fine hairs near the skin, usually singly, avoiding the coarse hairs of the mane and tail. This louse typically infests the side of the neck, the flanks, and tail base but can spread to most of the body with the exception of the mane, tail, ears, and lower legs. Long-haired horse breeds are more prone to infestation by *B. equi*. *Haematopinus asini*, the horse sucking louse (Fig. 7.13), is worldwide in distribution but more common in areas with cooler climates. It commonly parasitizes horses, donkeys, and mules. Adult females and males are 3.0 and 2.3 mm in length, respectively. Generally, this louse is found in areas of coarse hair avoided by the horse biting louse: the forelock, mane, base of the tail, and above the hooves.

Infestations by both species of horse lice are heavier during the winter months. As with other species of lice, advanced infestations can spread to additional regions of the body. While these lice normally transfer via direct contact, the use of contaminated grooming equipment and blankets can contribute to their spread.

Domestic swine are parasitized by one louse species, the hog louse (*Haematopinus suis*) (Fig. 7.14). This is a large species of sucking louse in which adult females measure

FIGURE 7.14 Hog louse (*Haematopinus suis*), female; stacked image of cleared specimen. *Photograph by Lorenza Beati and Lance A. Durden.*

5−6 mm in length and males measure over 4.1 mm. Geographically, the hog louse is found wherever hogs are raised, but it is more common in cooler climates. Hog lice normally frequent skin folds of the neck, the ears (often deep within the canal), the tender skin behind the ears, inside the legs, and the inner flanks of swine. Hog lice tend to favor upper regions of their predilection sites in summer and lower regions in winter. The heaviest infestations of hog lice occur in winter, usually December to March in North America, Europe, and northern Asia. Eggs are deposited singly on hairs (except when infestations become very heavy) along the lower parts of the body, in skin folds on the neck, and on and in the ears. Under optimal conditions, females deposit three to six eggs per day. The egg and nymphal stages last approximately 2 weeks each. Adults are thought to live about 1 month, and there can be 6−12 generations per year.

Domestic sheep and goats are parasitized by several species of sucking lice and chewing lice (Tables 7.1, 7.2). Worldwide, the sheep biting louse, *Bovicola ovis*, is the principal louse parasitizing domestic sheep. It is also one of the most studied. In Australia, a major sheep producing country, Roberts (1952) noted that the sheep biting louse occurred throughout the country in sheep-raising areas but was less frequent in, and sometimes absent from, the drier inland districts. This is probably true in the United States as well, where its distribution has not been documented. In 40 years, it has not been seen in Wyoming, one of the major U.S. sheep-producing states, and veterinary entomologists in neighboring states of Montana and Nebraska report that the species is uncommon, if not absent, in those states as well.

Females of the sheep biting louse are about 1.8 mm long, and males are around 1.0 mm. Females attach eggs primarily to wool fibers close to the skin. The incubation period is about 10 days, and the three nymphal stages together last about 3 weeks. The female lays eggs at the relatively slow rate of one egg every 2−4 days and can live for up to 30 days. Despite the low reproductive rate, infestations may reach hundreds of thousands or even a million on a single sheep. *Bovicola ovis* mainly feeds on epidermal scales, scurf, and dermal secretions. In the winter, when louse populations are high, most *B. ovis* are found on the back and mid-sides of the sheep. Lighter summer populations are along the ribs, lower flank, and abdomen.

The African blue louse, *Linognathus africanus*, is a parasite of both sheep and goats. Originally described from sheep in Africa, as its name implies, the species now appears to be distributed worldwide. In the United States, it has been reported from the southern and southwestern United States. Recently, it appears to have established in sheep-producing areas of several Western states, where it has become a major pest of sheep. The species has also been reported from mule deer, Columbian black-tailed deer, and white-tailed deer. Females are 2.2 mm long and males are 1.7 mm. In the winter, when infestations are heaviest, *L. africanus* is found most abundantly on the loin, back, rib, and shoulder areas of sheep. Populations may reach several thousand per sheep. On goats, the distribution is different, with lice occurring on the upper neck, base of the ears, poll, and ventral surface of the jaw.

Lice of Cats and Dogs

Domestic cats are parasitized by one species of chewing louse, whereas dogs are parasitized by two species of chewing lice and one species of sucking louse. All four species appear to be distributed worldwide, but none of them are common associates of healthy cats or dogs in North America or Europe.

The cat biting louse (*Felicola subrostrata*) parasitizes both domestic and feral cats. It may occur almost anywhere on the body.

Both the dog biting louse (*Trichodectes canis*) (Fig. 7.15A) and the dog sucking louse (*Linognathus setosus*) (Fig. 7.15C) parasitize dogs and closely related wild canids. For example, *T. canis* also parasitizes coyotes, foxes, and wolves. A second species of chewing louse of dogs is *Heterodoxus spiniger* (Fig. 7.15B), which evolved in Australasia from marsupial-infesting lice and apparently switched to dingo hosts. It now parasitizes various canids and other carnivores throughout the world. *Trichodectes canis* usually infests the head, neck, and tail region of dogs, where it attaches to the bases of individual hairs. *Linognathus setosus* occurs primarily on the head and neck and may be especially common beneath collars. *Heterodoxus spiniger* can typically be found anywhere on its host.

Lice of Laboratory Animals

The principal species of lice that parasitize laboratory mammals have been discussed by Kim et al. (1973). These lice also parasitize feral populations of their respective hosts.

The house mouse (*Mus musculus*) is often parasitized by the mouse louse (*Polyplax serrata*). Populations of this louse are typically low, with 10 or fewer lice per infested mouse, unless self or mutual host grooming is compromised. Eggs of this louse typically hatch 7 days after oviposition. Together, the three nymphal instars last only 6 days under optimal conditions, which can result in a generation time as short as 13 days.

Domestic rats are often parasitized by the spined rat louse (*Polyplax spinulosa*) (Fig. 7.16) and the tropical rat louse (*Hoplopleura pacifica*). Common hosts include the black rat (*Rattus rattus*) and the Norway rat

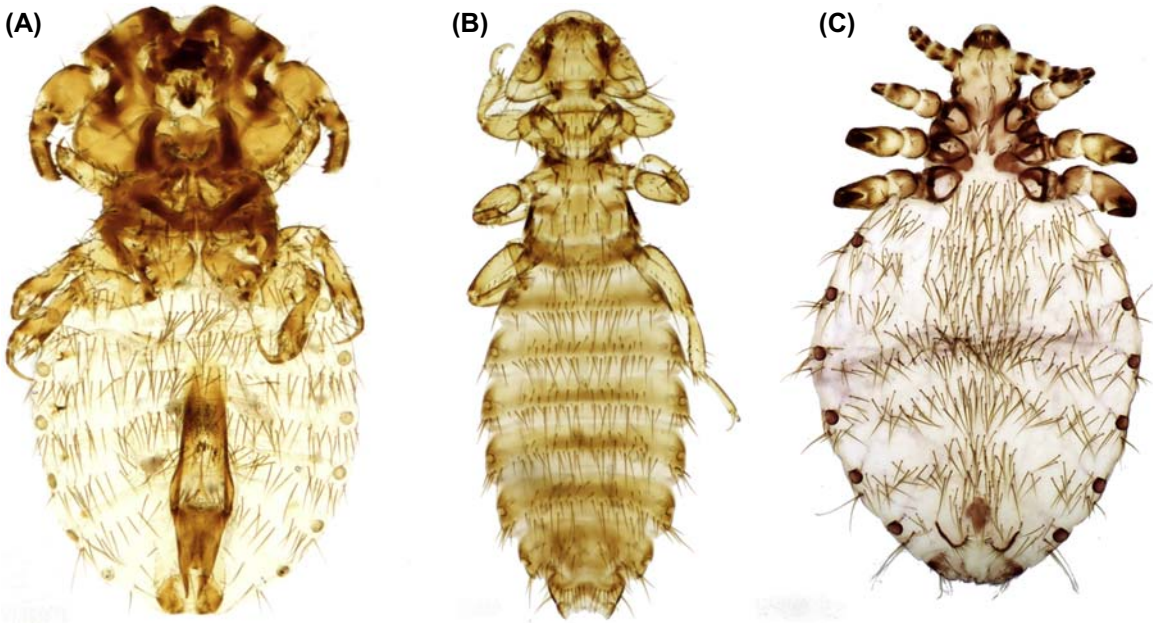


FIGURE 7.15 Lice of domestic dogs. (A) Dog biting louse (*Trichodectes canis*), male. (B) *Heterodoxus spiniger*, female. (C) Dog sucking louse (*Linognathus setosus*), female. Stacked images of cleared specimens. *Photographs by Lorenza Beati and Lance A. Durden.*

FIGURE 7.16 Spined rat louse (*Polyplax spinulosa*), male. Stacked image of cleared specimen. *Photograph by Lorenza Beati and Lance A. Durden.*

(*R. norvegicus*). The spined rat louse parasitizes these hosts throughout the world, whereas the tropical rat louse is confined to tropical, subtropical, or warm temperate regions, including the southern United States.

Laboratory rabbits are parasitized by the rabbit louse (*Haemodipsus ventricosus*). This louse originated in Europe but has accompanied its host wherever it has been introduced throughout the world.

Lice of Poultry and Other Birds

At least nine species of chewing lice infest poultry (Table 7.1, Fig. 7.17) in various parts of the world. Individual birds can be parasitized by multiple species, each of which often occupies a preferred host site.

The chicken body louse (*Menacanthus stramineus*) (Figs. 7.17B, 7.22) is the most common and destructive louse of domestic chickens. It is thought to have originally been a pest of wild turkeys that transferred to domestic poultry and is now common on both chickens and turkeys. The chicken body louse has a worldwide distribution and often reaches pest proportions. Unlike other chicken lice, it is found on the host's skin rather than on the feathers. It may be detected by parting the feathers, especially in the vent area of the bird. This louse is most abundant on the sparsely feathered vent, breast, and thigh regions. However, in heavily infested poultry, it may be found on any part of the body. Adults measure 3−3.5 mm in length. Females lay one or two eggs per day, cementing them in clusters at the bases of feathers, especially around the vent (Fig. 7.22). Eggs typically hatch after 4−5 days. Each nymphal instar lasts about 3 days, and the generation time typically is 13−14 days.

Several other chewing lice are pests of poultry more or less throughout the world (Table 7.1). Adults of the shaft louse (*Menopon gallinae*) (Fig. 7.17F) measure about 2 mm in length and may be seen in a line along the shaft of

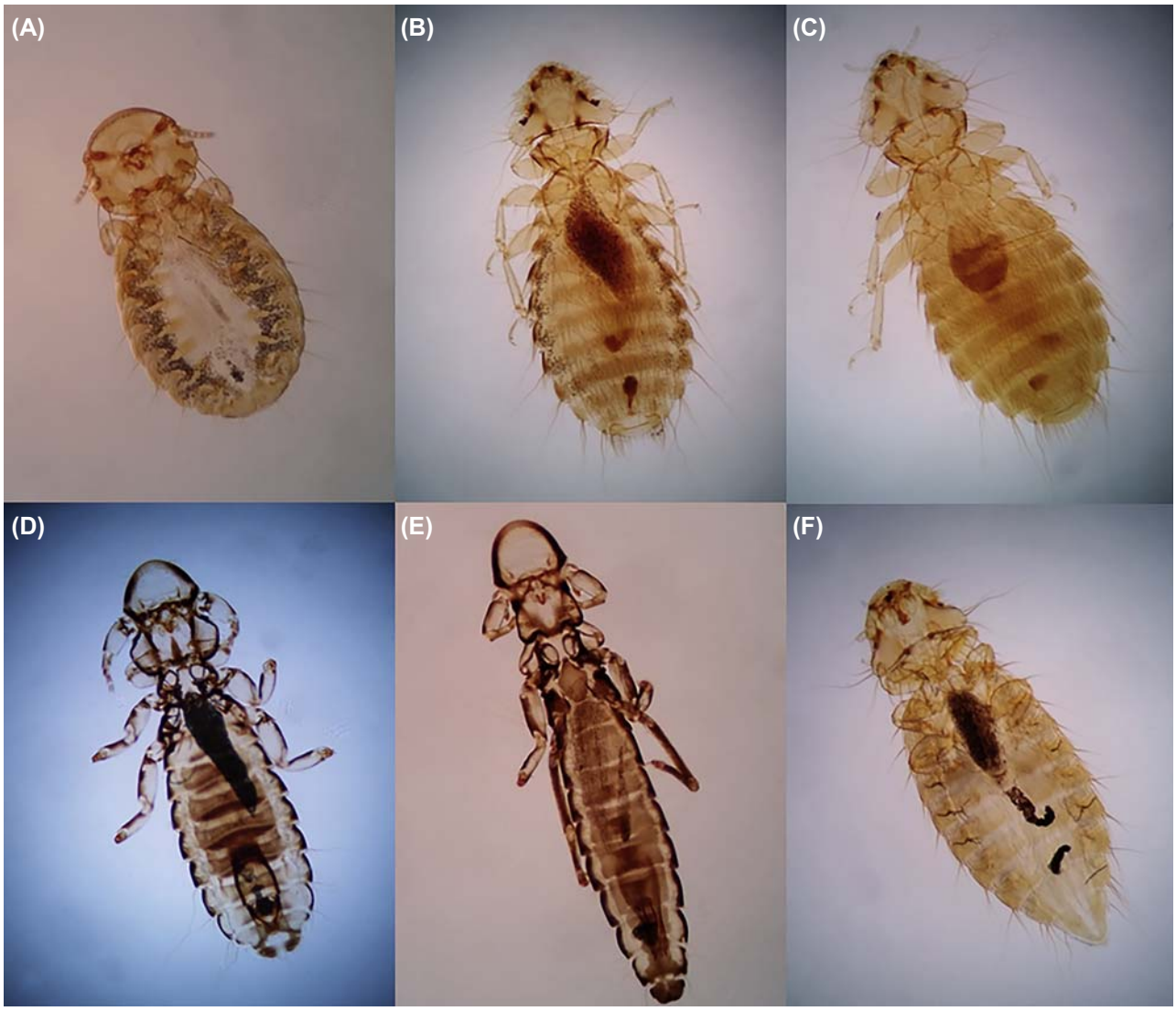


FIGURE 7.17 Common lice of poultry. (A) Fluff louse (*Goniocotes gallinae*). (B) Chicken body louse (*Menacanthus stramineus*). (C) *Menacanthus cornutus*. (D) Chicken head louse (*Cuclotogaster heterographus*). (E) Chicken wing louse (*Lipeurus caponis*). (F) Shaft louse (*Menopon gallinae*). *Photographs by Amy C. Murillo.*

a feather. Although these lice do not normally rest on the skin, they quickly disperse to the skin if disturbed. Females deposit eggs singly at the base of the shaft on thigh and breast feathers. Eggs of the wing louse (*Lipeurus caponis*) (Fig. 7.17E) hatch 4–7 days after the female has cemented them to the base of a feather. Nymphal stages of this species each last 5–18 days, generation time typically is 18–27 days, and females can live up to 36 days. Females of the chicken head louse (*Cuclutogaster heterographus*) (Fig. 7.17D) attach their eggs to the bases of downy feathers. Eggs hatch after 5–7 days, each nymphal instar lasts 6–14 days, and average generation time is 35 days. The fluff louse (*Goniocotes gallinae*) (Fig. 7.17A) is a small louse that often infests the entire body of chickens, especially in the fluffy areas at the feather bases. *Menacanthus cornutus* (Fig. 7.17C) is a fairly large poultry louse and can occur in large numbers, especially on backyard chicken flocks (Murillo and Mullens, 2016).

Poultry lice typically transfer to new birds via direct host contact. However, because most species can survive for several hours or days off the host, they also can infest new hosts during transportation in inadequately disinfected cages or vehicles.

PUBLIC HEALTH IMPORTANCE

Three important pathogens are transmitted to humans by body lice: these are agents of epidemic typhus, trench fever, and louse-borne relapsing fever. Today, the prevalence and importance of all three of these louse-borne diseases are

low compared with times when human body lice were an integral part of human lives and before the widespread use of antibiotics starting in the 1940s. However, trench fever has reemerged as an opportunistic disease of immunocompromised individuals, including persons who are positive for human immunodeficiency virus (HIV). Although these three pathogens are only known to be transmitted by body lice in nature, they have also been sporadically detected in head lice. Further, *Acinetobacter baumannii,* an opportunistic emerging bacterium that can infect humans via other transmission routes, has been found in both body and head lice.

Epidemic Typhus

Epidemic typhus is caused by infection with the rickettsial bacterium *Rickettsia prowazekii.* The entire genome sequence of *R. prowazekii* was reported by Andersson et al. (1998). The disease is also known as louse-borne typhus, louse-borne fever, jail fever, and exanthematic typhus. Some earlier reports of this disease simply referred to it as "typhus." Epidemic typhus persists in several parts of the world, most notably in Burundi, Democratic Republic of Congo, Ethiopia, Nigeria, Rwanda, areas of northeastern and central Africa, parts of Russia, Central and South America, and northern China. Epidemic typhus is largely a disease of cool climates, including higher elevations in the tropics. It thrives in conditions of widespread body louse infestations, overcrowding, and poor sanitary conditions. This disease apparently was absent from the New World until the 1500s, when the colonizing Spanish inadvertently introduced it. One resulting epidemic in 1576−1577 killed 2 million indigenous peoples in the Mexican highlands alone.

The principal vector of *R. prowazekii* is the human body louse. Lice become infected when they feed on a person with circulating *R. prowazekii* in their blood. Infective rickettsiae invade cells that line the louse gut, and multiply there, eventually causing the cells to rupture. Liberated rickettsiae either reinvade gut cells or are voided in louse feces. Other louse tissues typically do not become infected. Because salivary glands and ovaries are not invaded, anterior-station and transovarial transmission do not occur. Infection of susceptible humans occurs via louse feces (posterior-station) when infectious rickettsiae are scratched into the skin in response to louse bites. *Rickettsia prowazekii* can remain infectious in dried louse feces for 60 days. Infection via inhalation of dried louse feces or by crushed lice is an infrequent transmission route.

Transmission of *R. prowazekii* by body lice was first demonstrated by Charles Nicolle when he was working at the Institut Pasteur in Tunis in 1909. During these studies, Nicolle accidentally became infected with epidemic typhus, from which he eventually recovered. He was awarded the Nobel prize in 1928 for his groundbreaking work on typhus. Several other typhus workers also were infected with *R. prowazekii* during laboratory experiments. The American researcher Howard T. Ricketts, working in Mexico, and Czech scientist Stanislaus von Prowazek, working in Europe, both died from their infections and were recognized posthumously when the etiologic agent was named.

Infection with *R. prowazekii* is ultimately fatal to body lice as progressively more and more infected gut cells are ruptured. Infective rickettsiae are first excreted in louse feces 3−5 days after the infectious bloodmeal. Lice usually succumb to infection 7−14 days after the infectious bloodmeal, although some may survive for 20 days.

The disease caused by infection with *R. prowazekii* and transmitted by body lice is called **classic epidemic typhus** because it was the first form of the disease to be recognized. Disease onset occurs 10−14 days after infection by a body louse in classic epidemic typhus. Abrupt onset of fever accompanied by malaise, muscle and head aches, cough, and general weakness usually occur at this time. A blotchy, often reddish-blue rash spreads from the abdomen to the chest and then often across most of the body (Fig. 7.18), typically within 4−7 days after the initial symptoms. The rash rarely spreads to the face, palms, and soles and then only in severe cases. Headache, rash, prostration, and delirium intensify as the infection progresses. Coma and very low blood pressure often signal fatal cases. A case-fatality rate of 10%−20% is characteristic of most untreated outbreaks, although figures approaching 50% have been recorded.

Diagnosis of epidemic typhus involves positive serology, usually by microimmunofluorescence. Primers to *R. prowazekii* can also amplify DNA from infected persons or lice using polymerase chain reaction techniques. Antibiotic treatment, especially with doxycycline or tetracycline, usually results in rapid and complete recovery. Vaccines are available but are not considered to be sufficiently effective for widespread use.

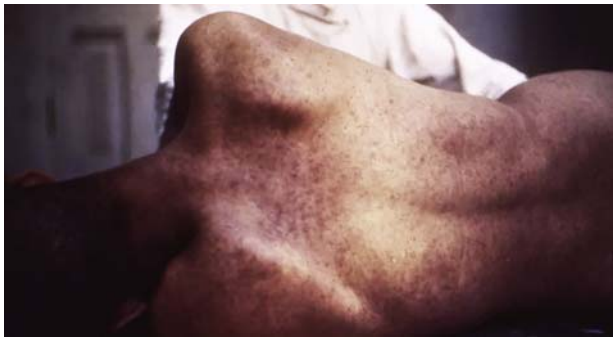


FIGURE 7.18 Patient infected with epidemic (louse-borne) typhus showing characteristic maculopapular cutaneous rash. *Courtesy, Public Health Image Library, Centers for Disease Control and Prevention, Atlanta, Georgia, USA.*

Persons who recover from epidemic typhus typically harbor *R. prowazekii* in lymph nodes or other tissues for months or years. This enables the pathogen to later reinvade other body tissues to cause disease. This form of the disease is called **recrudescent typhus** or **Brill–Zinsser disease**. The latter name recognizes two pioneers in the study of epidemic typhus, **Nathan Brill,** who first recognized and described recrudescent typhus in 1910, and **Hans Zinsser,** who demonstrated in 1934 that it is a form of epidemic typhus. Zinsser's (1935) book *Rats, Lice and History* is a pioneering account of the study of epidemic typhus in general.

Recrudescent epidemic typhus was widespread during the nineteenth and early twentieth centuries in some of the larger cities along the east coast of the United States (e.g., Boston, New York, and Philadelphia). At that time, immigrants from regions that were rampant with epidemic typhus, such as eastern Europe or Ireland, presented with Brill–Zinsser disease after being infected initially in their country of origin. Some of these patients experienced relapses more than 30 years after their initial exposure, with no overt signs of infection with *R. prowazekii* between the two disease episodes. Because infestation with body lice was still a relatively common occurrence during that period, lice further disseminated the infection to other humans, causing regional outbreaks. The last outbreak of epidemic typhus in North America occurred in Philadelphia in 1877. Today, even recrudescent typhus is a rare occurrence in North America. However, this form of typhus is still common in parts of Africa, Asia, South America, and occasionally eastern Europe. Some travelers returning to Europe or North America from endemic areas have presented with classic epidemic typhus.

The southern flying squirrel (*Glaucomys volans*) has been identified as a reservoir of a less virulent strain of *R. prowazekii* in the United States, where it was first found to be infected in Virginia during vertebrate serosurveys for Rocky Mountain spotted fever. Since the initial isolations from flying squirrels in 1963, *R. prowazekii* has been recorded in flying squirrels and their ectoparasites in several states, especially eastern and southern states. Peak seroprevalence (about 90%) in flying squirrels occurs during late autumn and winter when fleas and sucking lice are also most abundant on these hosts. Although several ectoparasites can imbibe *R. prowazekii* when feeding on infected flying squirrels, only the sucking louse *Neohaematopinus sciuropteri* is known to maintain the infection and to transmit the pathogen to uninfected squirrels; nevertheless, a squirrel flea, *Orchopeas howardi*, is also a likely vector. Several North American cases of human infection have been documented in which the patients recalled having contact with flying squirrels, especially during the winter months when these rodents commonly occupy attics of houses or cabins. To distinguish this form of the disease from classic and recrudescent typhus, it is called **sporadic epidemic typhus** or sylvatic epidemic typhus. Some details such as the mode of human infection remain unresolved. Because the flying squirrel–associated louse *N. sciuropteri* does not feed on humans, it has been speculated that human disease may occur when infectious, aerosolized rickettsiae from louse feces are inhaled from attics or other sites occupied by infected flying squirrels.

Historically, epidemic typhus has been the most widespread and devastating of the louse-borne diseases. Zinsser (1935), Snyder (1966), and Allen (2014) have documented the history of this disease and highlighted how major epidemics influenced human history. For example, Napoleon's vast army of 1812 was arguably defeated more by epidemic typhus than by opposing Russian forces. Soon thereafter (c. 1816–1819), 700,000 cases of epidemic typhus occurred in Ireland. Combined with the potato famine of that period, this encouraged many people to emigrate to North America; some of these people carried infected lice or latent infections with them. During World War II, several military operations in North Africa and the Mediterranean region were hampered by outbreaks of epidemic typhus. One epidemic in Naples, Italy, in 1943 resulted in more than 1,400 cases and 200 deaths. This outbreak is particularly noteworthy because it was the first epidemic of the disease to be interrupted by human intervention through widespread application of the insecticide DDT to louse-infested persons.

Today, epidemic typhus is much less of a health threat than it once was. This is largely because few people, especially in developed countries, are currently infested by body lice. Higher sanitary standards, less overcrowding, regular laundering, frequent changes of clothes, effective pesticides, and effective antibiotics have all contributed to the demise of this disease. Nevertheless, epidemic typhus has the potential to reemerge. This is evidenced by the largest outbreak of epidemic typhus since World War II that affected about 50,000 people living in refugee camps in Burundi in 1997 and 1998. Further, more than 5,600 cases were recorded in China during 1999, and recent cases have been recorded in parts of Russia. Some people in Mexico and Texas have antibodies to *R. prowazekii*. *Rickettsia prowazekii* is currently listed as a select agent by the U.S. Centers for Disease Control and Prevention because of its ability to cause epidemics with high mortality and its bioweapon potential. Additional information about epidemic typhus is provided by Andersson et al. (1998), Anderson and Andersson (2000), and Allen (2014).

Louse-Borne Relapsing Fever

Also known as epidemic relapsing fever, this disease is caused by the spirochete bacterium *Borrelia recurrentis.* This pathogen is transmitted to humans by the human body

louse, as first demonstrated by Sergent and Foley in 1910. Clinical symptoms include the sudden onset of fever, headache, muscle ache, anorexia, dizziness, nausea, coughing, and vomiting. Thrombocytopenia (a decrease in blood platelets) also can occur which can result in bleeding, a symptom that may initially be confused with a hemorrhagic fever. Episodes of fever last 2−12 days, typically followed by periods of 2−8 days without fever, with two to five relapses being most common. As the disease progresses, the liver and spleen enlarge, leading to abdominal discomfort and labored, painful breathing as the lungs and diaphragm are compressed. At this stage, most patients remain prostrate, often shivering and taking shallow breaths. Case-fatality rates for untreated outbreaks range from 5% to 40%. Antibiotic treatment is with penicillin or tetracycline. Humans are the sole known reservoir of *B. recurrentis*.

Body lice become infected when they feed on an infected person with circulating spirochetes. Most of the spirochetes perish when they reach the louse gut, but a few survive to penetrate the gut wall, where they multiply to massive populations in the louse hemolymph, nerves, and muscle tissue. Spirochetes do not invade the salivary glands or ovarian tissues and are not voided in louse feces. Therefore, transmission to humans typically occurs when infected lice are crushed during scratching, which allows the spirochetes in infectious hemolymph to invade the body through abrasions and other skin lesions. However, *B. recurrentis* is also capable of penetrating intact skin. As with *R. prowazekii* infections, body lice are eventually killed as a result of infection with *B. recurrentis*.

An intriguing history of human epidemics of louse-borne relapsing fever is provided by Bryceson et al. (1970). Hippocrates described an epidemic of "caucus" or "ardent fever" in Thasos, Greece, which can clearly be identified by its clinical symptoms as this malady. From 1727 to 1729, an outbreak in England killed all inhabitants of many villages. An epidemic that spread from eastern Europe into Russia during 1919−1923 resulted in 13 million cases and 5 million deaths. Millions also were infected during an epidemic that swept across North Africa in the 1920s. Several major epidemics subsequently have occurred in Africa, with up to 100,000 fatalities being recorded for some of them. During and immediately after World War II, more than 1 million persons were infected in Europe alone.

An ongoing outbreak of louse-borne relapsing fever is occurring in Ethiopia, where 1,000−5,000 cases are typically reported annually accounting for about 95% of the world's recorded infections. Other smaller foci occur intermittently in other regions such as Burundi, Rwanda, Sudan, Uganda, China, Russia, Central America, and the Peruvian Andes. Localized resurgence of this disease under conditions of warfare or famine is a possibility.

Trench Fever

Also known as 5-day fever and wolhynia, trench fever is caused by infection with the bacterium *Bartonella* (formerly *Rochalimaea*) *quintana*. Like the two preceding diseases, the agent is transmitted by the human body louse. Human infections range from asymptomatic through mild to severe, although fatal cases are rare. Clinical symptoms can be nonspecific and include headache, muscle aches, fever, and nausea. The disease can be cyclic with several relapses often occurring. Previously infected persons often maintain a cryptic infection that can cause relapses years later with the potential for spread to other persons if they are infested with body lice. Effective antibiotic treatment of patients involves administering drugs such as doxycycline or tetracycline. *Bartonella quintana* DNA has been molecularly detected in a 4,000-year-old human tooth (Drancourt et al., 2005).

Lice become infected with *B. quintana* after feeding on the blood of an infected person. The pathogen multiplies in the lumen of the louse midgut and in the midgut epithelial cells. Infectious bacteria are voided in louse feces, and transmission to humans occurs via the posterior-station route when louse bites are scratched. *Bartonella quintana* can remain infectious in louse feces for several months, contributing to aerosol transmission as a rare but alternative route of transmission. Transovarial transmission does not occur in the louse vector. Infection is not detrimental to lice and does not affect their longevity.

Trench fever was first recognized as a clinical entity in 1916 as an infection of European troops engaging in trench warfare during World War I. At that time, more than 200,000 cases were recorded in British troops alone. Between the two world wars, trench fever declined in importance but reemerged in epidemic proportions in troops stationed in Europe during World War II. Because of the presence of asymptomatic human infections, the current distribution of trench fever is difficult to determine. However, since World War II, infections have been recorded in several European and African nations, Japan, China, Mexico, Bolivia, and Canada.

Until the 1990s, *B. quintana* was considered to be a rare disease of humans. However, several inner-city homeless and/or immunocompromised people, including HIV-positive individuals, particularly in North America, Europe, and Asia, have presented with *B. quintana* infections. This is manifested not as trench fever but as vascular tissue lesions (bacillary angiomatosis), liver pathology, chronically swollen lymph nodes, and/or inflammation of the heart valves (endocarditis). Because this disease typically occurs in inner cities, it has been given the name **urban trench fever.** The disease is probably widespread worldwide having being recorded, for example, in the cities of New York, Seattle, San Francisco, Marseille, Tokyo, and Moscow.

Long thought to be only a human disease (an anthroponosis), *B. quintana* has recently been reported to infect at least three species of macaques in southeast Asia and a macaque-associated sucking louse, *Pedicinus obtusus*, suggesting the possibility of an animal (zoonotic) origin for this disease (Li et al., 2013).

Other Pathogens Transmitted by Human Body Lice

Some additional bacterial pathogens can be transmitted by body lice under certain conditions. For example, *Salmonella typhi*, the agent of typhoid (salmonellosis), can be louse-borne during outbreaks of this disease. However, other modes of transmission, such as through contaminated food, account for most human cases. Another emerging, widespread bacterium, *Acinetobacter baumannii*, which can cause infections of human skin, wounds, the urinary tract, lungs, and meninges (and is often drug resistant), has been molecularly detected in both body and head lice removed from humans. In laboratory trials, body lice can successfully imbibe *A. baumannii* while feeding on experimentally infected rabbits. These lice cannot transmit *A. baumannii* via a bite, but they excrete viable bacteria in their feces, which could represent a source of human infection. Human body lice can also transmit *R. rickettsii* and *Rickettsia conorii* to rabbits under laboratory conditions; these pathogens cause Rocky Mountain spotted fever and Boutenneuse fever, respectively, and are typically transmitted by ticks. Human body lice can transmit *Yersinia pestis,* the causative agent of plague, under laboratory conditions, and it has been hypothesized that transmission of this pathogen by body lice could have been widespread during the historical plague outbreaks (Ayyadurai et al., 2010).

Lice as Intermediate Hosts of Tapeworms

Occasionally, humans become infested with the **double-pored tapeworm** (*Dipylidium caninum*). Although carnivores are the normal definitive hosts for this parasite, humans can be infested if they accidentally ingest infested dog biting lice (*Trichodectes canis*), which serve as intermediate hosts. Although this would appear to be an unlikely event, infants, especially children playing on carpets or other areas frequented by a family dog, may touch an infested louse with sticky fingers, which may then be put into their mouth to initiate an infestation.

VETERINARY IMPORTANCE

A variety of chewing lice and sucking lice parasitize domestic animals (Tables 7.1, 7.2). The potential effects of the various species of lice on livestock production are many. Even infestations that are considered relatively light may, in some way, have a measurable negative impact. Infestation by lice, presumably because of both their feeding and movement on the host, may be extremely annoying. Host animals often become restless. The host may develop dermatitis, allergic reactions, or secondary infection contributing to the pruritus. Host responses to the irritation include licking and rubbing the affected body parts, which may produce negative consequences. Hair, wool, mohair, or feather loss might hinder thermoregulation and result in unsightly animals with a lower market value. Scratches reduce the value of hides from slaughtered animals because defects must be trimmed away. Licking can lead to the formation of hair balls in the stomach, as seen especially in cats and calves. Large animals, in particular, can damage gates, fences, and other livestock equipment against which they rub to relieve irritation. Feeding by lice may directly, or through an immunological response by the host, result in blemishes in the hide.

The effect of blood-feeding parasites such as lice is more than merely robbing nutrients necessary for normal growth of the host. Feeding and salivary secretions of ectoparasites can stimulate immunologic and nonspecific defense mechanisms, ultimately influencing behavior and physiology of a host and preventing the host from reaching its full growth potential. This effect may reduce not only weight gains but also production of byproducts such as milk and eggs. Although lice may be barely detectable, they can multiply to extremely high numbers, particularly on young, old, sick, or stressed animals. Often this is because these hosts are unable to effectively groom themselves or they are immunocompromised. Sucking lice infestations, especially when they become severe, may affect the vitality of the host, promoting anemia, toxic anemia, abortion, and death and likely, as suggested by Campbell (1988), increasing susceptibility to pathogens.

Few pathogens are known to be transmitted to domestic animals by lice (Table 7.3). The most important of these are the viral agent of swinepox and the bacterial agents of murine mycoplasma infection of rats and mice, caused by *Mycoplasma muris* and *Mycoplasma coccoides*, respectively (Table 7.3). Additionally, *Mycoplasma suis,* which causes an acute febrile disease in feeder pigs, has been reported to be transmitted by the hog louse by some researchers. Other pathogens have been detected in various species of lice, but there is no current evidence that lice are vectors of these organisms.

Lice of Livestock

Lice cause major economic losses through reduced livestock productivity and diminished health. These losses include the cost of treatment, which can be considerable. Louse infestation may lead to pruritus and its side effects,

TABLE 7.3 Pathogens and Parasites Transmitted by Lice

Disease	Disease Agent	Vector(s)	Host(s)	Geographic Distribution
VIRAL				
Swinepox	Swinepox virus	*Haematopinus suis*	Hogs	Widespread
BACTERIAL				
Epidemic typhus	*Rickettsia prowazekii*	*Pediculus humanus*	Humans	Global (focal)
Sporadic epidemic typhus	*Rickettsia prowazekii*	Flying squirrel lice	Flying squirrels, humans	North America
Louse-borne relapsing fever	*Borrelia recurrentis*	*Pediculus humanus*	Humans	Global (focal)
Trench fever	*Bartonella quintana*	*Pediculus humanus*	Humans	Global
Tularemia	*Francisella tularensis*	Rodent & lagomorph lice	Rodents, rabbits	Global
Murine Mycoplasma Infection	*Mycoplasma muris*	*Polyplax spinulosa*	Domestic rats	Global
Murine Mycoplasma Infection	*Mycoplasma coccoides*	*Polyplax serrata*	Domestic mice	Global
HELMINTHIC				
Seal heartworm	*Dipetalonema spirocauda*	*Echinophthirius horridus*	Harbor seal	Northern Hemisphere
Avian filariasis	*Eulimdana* spp.	Bird chewing lice	Charadriiform birds	Widespread
Avian filariasis	*Pelecitus fulicaeatrae*	Bird chewing lice	Aquatic birds	Holarctic region
Avian filariasis	*Sarconema eurycerca*	*Trinoton anserinum*	Geese, swans	Holarctic region
Double-pored tapeworm[a]	*Dipylidium caninum*	*Trichodectes canis*	Dogs, humans	Global

[a]*Lice are intermediate hosts, not vectors, of this tapeworm.*

reduced productivity, and diminished general health. During colder months of the year, louse infestations can reach into the thousands, hundreds of thousands, or even a million per animal. Under these conditions, the most serious detrimental effects to the host occur, including death. Animals most heavily infested are often the young, ill, nutritionally deprived, and immunocompromised.

Cattle sucking lice can decrease cattle weight gains and milk production, in addition to necessitating additional feed to maintain louse populations and additional time to feed. The shortnosed cattle louse (Fig. 7.11D) in North America can be a cause of severe, and terminal, anemia in range cattle. The cattle tail louse is considered to be the most damaging cattle louse in Florida. Results of studies on the impact of the longnosed cattle louse (Fig. 7.11B), little blue cattle louse (Fig. 7.11C), and cattle biting louse (Fig. 7.11A) on weight gains have been mixed. However, Gibney et al. (1985) found significant differences between weight gains of cattle heavily infested with this complex of lice and weight gains of noninfested cattle. This complex of lice is typical on yearling cattle throughout the Great Basin and Rocky Mountain region of the United States. Weight gains are typically lower in stressed cattle and in those receiving inadequate nutrition. Nelson (1984) identified a "synergistic relationship," whereby a coexisting malnutrition and parasitic infestation are more deleterious to the host than either one is alone. Campbell (1988) further suggested a synergistic relationship between louse infestation and cold winter weather.

Damage to leather caused by cattle lice is costly. Irritation, leading to rubbing and subsequent hide damage (Fig. 7.19), may be caused by small numbers of lice in sensitive cattle. The cattle biting louse not only is responsible for increased scratches in cattle hide due to irritation (Fig. 7.19) but also is the major cause of the defect known as **light spot**, manifesting as 1- to 3-mm lesions that result from erosion of grain enamel of the hide. Light spot has been responsible for annual losses in the United Kingdom of £15 to £20 million (Coles et al., 2003). Rubbing also damages livestock facilities, a major concern in cattle feed

FIGURE 7.19 Steer infested with the cattle biting louse (*Bovicola bovis*); note areas where hair has been rubbed off by the host. *Photograph by John E. Lloyd.*

lots. Under laboratory or confined conditions, at least three pathogens can be transmitted by cattle sucking lice: the causative agents of bovine anaplasmosis, dermatomycosis (=ringworm), and, rarely, theileriosis. The importance of cattle lice in transmitting any of these pathogens in nature is unknown but presumed to be low.

Lice of horses and other equids typically do not greatly debilitate their hosts except when they are present in large numbers. As with other host species, horses that are malnourished or in poor health can become heavily infested. Horses in poor health may be infested by several thousand individuals of the horse biting louse (Fig. 7.12). Pruritus, hair loss, and coat deterioration may occur in severely infested animals. Horses with severe louse infestations, or horses that are extremely sensitive to infestation, are nervous and irritable. They stamp their hooves, lick, and rub in response to lice. They bruise and scratch themselves. Hair can be rubbed from the neck, shoulders, flanks, and tail base, resulting in an unthrifty appearance that may affect the market value of the horse. No pathogens are known to be transmitted by equid lice.

Hog lice, *Haematopinus suis* (Fig. 7.14), are extremely irritating to the host, and frequent feeding by the lice causes hogs to rub on objects to the point of bleeding. Hair is lost and the skin becomes rough and scaly with lesions. Heavily infested hogs are restless and eat less, which interferes with their growth. Hog lice can imbibe significant volumes of blood, especially from piglets, which often have larger infestations than adult pigs. Anemia may occur. Hog lice are potential vectors of several disease agents of swine. *Haematopinus suis* is a mechanical vector of the virus that causes **swinepox** (Table 7.3), a serious and potentially fatal disease characterized by large pockmark lesions mainly on the belly of infected animals. Swinepox is more commonly transmitted via direct contact between pigs. Some studies have also implicated this louse as a vector of *Mycoplasma* (formerly *Eperythrozoon*) *suis* and *Mycoplasma* (formerly *Eperythrozoon*) *parvum,* causative agents of swine mycoplasma infection, and of African swine fever virus. However, transmission of these pathogens by lice appears to be rare in nature.

Lice that parasitize sheep and goats (Tables 7.1, 7.2) can cause serious losses, even when present in relatively small numbers, because of damage to wool and mohair (Figs. 7.20, 7.21). Biting and rubbing in response to irritation damage the skin and devalue wool and mohair. The fleece becomes ragged with broken fibers, and it may slip from, or be pulled from, the skin, creating bare areas (Fig. 7.21). The fleece becomes contaminated with lice, cast exoskeletons, ova, and feces. Sucking lice can stain the wool with blood (Fig. 7.20), presumably due to undigested blood in their feces; this wool will not scour.

Significant losses due to the sheep biting louse, *Bovicola ovis,* are incurred in the major sheep-producing countries, such as Australia, New Zealand, South Africa,

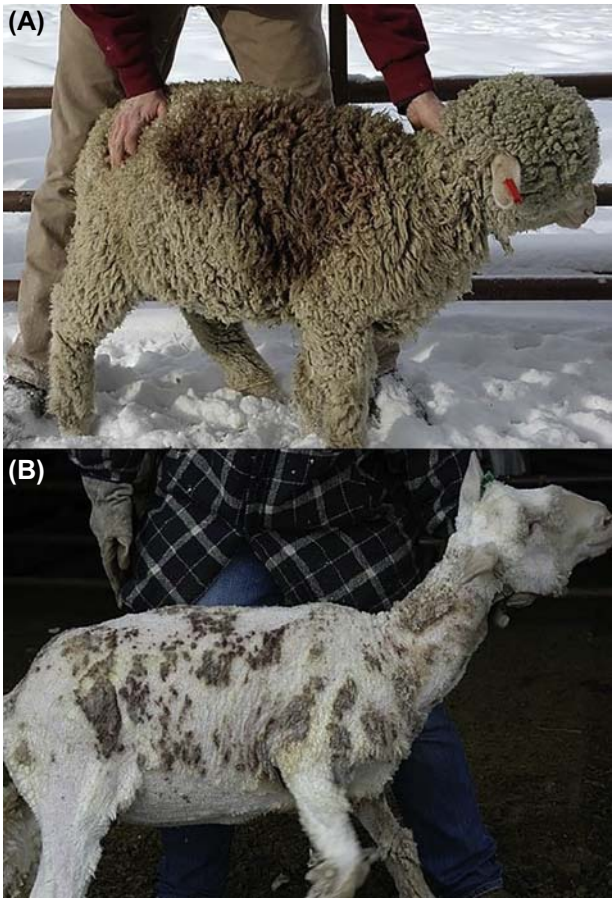


FIGURE 7.20 A lamb infested with the African blue louse (*Linognathus africanus*). (A) In full fleece, with large patch of bloody frass (fecal matter) from the lice. (B) The same lamb after shearing, with bloody patches of skin (brownish) where the lice are clustered. *Photograph by Greg Johnson.*

FIGURE 7.21 Fleece damage (wool slippage) in a ewe, caused by a severe infestation with the African blue louse (*Linognathus africanus*). *Photograph by John. E. Lloyd.*

and others. In Western Australia, louse infestation reduces production of clean wool by 0.3−0.8 kg/animal. In addition to causing fleece devaluation worldwide, the sheep biting louse is the major cause of cockle in pelts. Some sheep develop hypersensitivity to the sheep biting louse. **Cockle**, a nodular condition of the skin, arises in response to infestation by *B. ovis* and a hypersensitivity to louse antigens (Heath et al., 1996). The term **scatter cockle** has been used to describe the distribution of this defect and to distinguish it from **rib cockle,** a pelt blemish caused by sheep keds (*Melophagus ovinus*).

Sheep heavily infested with the African blue louse, *Linognathus africanus,* develop bare spots along the sides of the body (Fig. 7.21). Wool slips from the infested areas, leaving a bare area surrounded by a circle of densely infested fleece. Additional effects can include anemia and death in goats, especially kids, and skin irritation and fleece damage in Angora goats.

Wild and working elephants (Indian and African) can be infested by the elephant louse (*H. elephantis*) (Figure 7.1D, 7.5). In large infestations, this louse can cause intense irritation in its hosts, which can result in rubbing and skin lesions.

Lice of livestock, especially cattle, can also transmit fungi that cause ringworm (dermatomycosis), including *Trichophyton verrucosum*. Presumably, this involves mechanical transmission of fungal spores on louse bodies, including mouthparts. However, these fungi are more commonly transmitted via direct contact between infected animals.

Lice of Wildlife

Typically, lice of wildlife cause little or no apparent health problems unless their hosts are immunosuppressed, stressed, or unable to groom efficiently, sometimes because of injury. However, a chewing louse of North American wild deer (*Odocoileus* spp.) can multiply to huge numbers and cause severe hair loss (hair loss syndrome) and sometimes death from hypothermia in winter. Although the louse appears to be widespread on deer in the United States, most pathological cases have been reported from the Pacific Northwestern states of Oregon and Washington from Columbian black-tailed deer (*Odocoileus hemionus columbianus*) (Bildfell et al., 2004). The causative chewing louse has been identified as an undescribed species of *Damalinia*, a louse genus that is not thought to be native to North America. Similarly, massive infestations of the African blue louse, *Linognathus africanus,* have been associated with mule deer deaths in California due to exsanguination, anemia, and winter stress.

Lice of Cats and Dogs

Louse infestations of cats and dogs are most noticeable on sick, malnourished hosts, which are often unable to groom themselves, or on very young or old animals. Under these conditions, louse populations can increase dramatically. Apparently healthy pets will readily pick up lice when exposed to infested animals. Severe infestations by any of the four species of lice cause host restlessness, scratching, skin inflammation, a ruffled or matted coat, and hair loss. Heavy infestations of sucking lice may produce anemia.

The dog biting louse (*Trichodectes canis*) (Fig. 7.15A) is distributed worldwide but apparently is becoming less common in the United States (Kim et al., 1973). This species often occurs on the head, neck, and tail of the host and will aggregate around wounds and body openings. Infestations are typically most severe on puppies and older dogs in poor condition. The dog biting louse is an intermediate host of the double-pored tapeworm (*Dipylidium caninum*) (Table 7.3). Lice become infected when they ingest viable *D. caninum* eggs from dried host feces. The tapeworm develops into a cysticercoid stage in the louse where it remains quiescent unless the louse is ingested by a dog, usually during grooming. In the dog gut, the cysticercoid is liberated and metamorphoses into an adult tapeworm.

The dog sucking louse (*Linognathus setosus*) (Fig. 7.15C) occurs worldwide but is infrequently encountered. This louse occurs primarily on the neck, shoulders, and under the collar and mainly on long-haired dog breeds. The dog sucking louse may cause anemia due to blood loss. The lice may cause irritation, resulting in sleeplessness, nervousness, biting, and scratching, often leading to secondary bacterial infection and hair loss. This species has been shown to harbor immatures of the filarial nematode *Acanthocheilonema reconditum*, which parasitizes dogs, and these lice appear to be vectors.

The cat biting louse, *Felicola subrostrata*, is worldwide in distribution but relatively uncommon. Infestations occur

mainly on unhealthy, older or long-haired cats, or on cats unable to groom. The intense irritation caused by this species may cause severe scratching, dermatitis, and hair loss on the back of infested cats. Secondary bacterial infections may develop as a result of the scratching.

Lice of Laboratory Animals

Some lice that parasitize laboratory animals initiate serious health problems by causing pruritus, skin lesions, scab formation, anemia, and hair loss. Others are vectors of pathogens that can cause severe problems in animal colonies (Table 7.3). The mouse louse, *Polyplax serrata*, is a vector of the bacterium *Mycoplasma coccoides*, which causes **murine mycoplasma infection**, a potentially lethal infection of mice that occurs worldwide. Infection of this blood parasite in mice can either be inapparent or result in severe anemia. Transmission of this pathogen in louse-infested mouse colonies is usually rapid. The spined rat louse, *Polyplax spinulosa* (Fig. 7.16), is a vector of *Mycoplasma muris*, which causes another form of **murine mycoplasma infection** (Table 7.3), a potentially fatal blood infection that can cause severe anemia in laboratory rats.

Laboratory and wild guinea pigs are parasitized by two species of chewing lice: the slender guineapig louse, *Gliricola porcelli*, and the oval guineapig louse, *Gyropus ovalis.* Small numbers of these lice cause no noticeable harm, whereas large populations can cause host unthriftiness, scratching (especially behind the ears), hair loss, and a ruffled coat.

Large infestations of the rabbit louse, *Haemodipsus ventricosus*, can cause severe itching and scratching, which result in the host rubbing against its cage, often causing hair loss. Young rabbits are more adversely affected than adults and may experience retarded growth as a consequence of infestation by *H. ventricosus*. The rabbit louse is also a vector of the causative agent of tularemia among wild rabbit populations (Table 7.3).

Lice of Poultry and Other Birds

Although louse populations may be very large on domestic fowl, including domestic chickens, turkeys, guinea fowl, pea fowl, and pheasants, no pathogens are known to be transmitted by these lice. The chicken body louse (*Menacanthus stramineus*) (Fig. 7.17B) often causes significant skin irritation and reddening through its persistent feeding (Fig. 7.22). Occasionally the skin or soft quills pushing through the skin bleed from the gnawing and scraping action of the lice, with the lice readily imbibing the resultant blood. Populations of the chicken body louse are influenced by the host's ability to groom. Debeaked birds tend to become more heavily infested. In general, louse-infested chickens do not gain as much weight or produce as many eggs as do louse-free chickens, and heavily infested young chicks may die.

FIGURE 7.22 Chicken body lice (*Menacanthus stramineus*) feeding on a chicken with large egg mass of *M. stramineus* also present (on left). *Photograph by Amy C. Murillo.*

The shaft louse (*Menopon gallinae*) (Fig. 7.17F) also causes significant losses to the poultry industry, including deaths of young birds with heavy infestations. Large infestations of chicken body lice, shaft lice, and other poultry lice may be injurious to the host by causing feather loss, lameness, low weight gains, inferior laying capacity, and even death. Lice and other ectoparasites infest peridomestic and backyard chicken flocks more commonly than they do caged layer and other confined flocks (Murillo and Mullens, 2016).

The vast majority of chewing lice are parasites of wild or peridomestic birds. Several of these lice are suspected vectors of avian pathogens. Some chewing lice of aquatic and other birds, including geese and swans, coots, grebes, and whimbrels, are vectors of filarial nematodes, including *Pelecitus fulicaeatrae, Sarconema eurycera*, and species of *Eulimdana* (Bartlett and Anderson, 1989; Clayton et al., 2015) (Table 7.3). Pet parrots, parakeets, budgerigars, and other birds also are subject to infestation by chewing lice, which is usually noticed mainly by host scratching and ruffled or lost feathers. Large populations of these lice can debilitate their hosts. Ranch birds such as ostriches, emus, and rheas are also prone to similar adverse effects caused by their associated chewing lice.

PREVENTION AND CONTROL

Several techniques have been used in attempts to rid humans and animals of lice and louse-borne diseases. Preventing physical contact between lousy persons or animals and the items they contact, as well as various chemical, hormonal, and biological control mechanisms, compose the current arsenal of techniques. Chemicals used to kill lice are called **pediculicides**.

Clothes of persons with body lice should be changed frequently, preferably daily, and washed in very hot, soapy water to kill lice and nits. Washing associated bed linen in this manner is also advisable. Infested people should also receive a concurrent whole-body treatment with a pediculicide. Overcrowded and unsanitary conditions should be avoided whenever possible during outbreaks of human body lice and louse-borne diseases because it is under these situations that both can thrive.

Crab lice can often be avoided by refraining from having multiple sexual partners and changing or laundering bed linen slept on by such persons. Pediculicides should be applied to the pubic area and to any other infested body regions.

To reduce the spread of head lice, the sharing of combs, hats, earphones, and blankets, especially by children, should be discouraged. Often, parents of children with head lice are notified to keep youngsters away from school or other gatherings until the infestation has been eliminated. If the parents are also infested, this can further involve ridding lice from the entire family to prevent reinfestations. Various pediculicidal shampoos, lotions, and gels are widely available for controlling head lice. In the United States, topically applied 1% permethrin or 0.33% pyrethrins are typically used to kill human lice, and 0.5% malathion lotions are often used in cases of initial treatment failures. However, topically applied pediculicides are not very effective against body lice, which spend most of their lives in the clothing. These treatments typically kill all nymphal and adult lice but only a proportion of viable louse eggs. Therefore, treatments should be repeated at weekly intervals for up to 4 weeks to kill any recently hatched lice. Hatched or dead nits that remain glued to hair may be unsightly or embarrassing and can be removed with a fine-toothed louse comb. **Louse combs** have been used, in various forms, since antiquity to remove head lice (Mumcuoglu, 1996) and remain in widespread use today. Because lice are highly susceptible to desiccation, one technique for killing head lice and their eggs is to expose infested human scalps to a stream of hot air (Goates et al., 2006). An increasingly popular method for avoiding louse infestations is to impregnate clothing with permethrin; this action prevents, for example, health care workers or soldiers who are interacting with louse-infested refugees from becoming infested.

A wide range of pediculicides are commercially available. Although its use is now banned in many developed countries, the organochlorine DDT is widely used, especially in less developed countries, for controlling human and animal lice. Several alternative pediculicides, such as lindane, chlorpyrifos, diazinon, malathion, permethrin, or pyrethrins, are currently used in different parts of the world. Pediculicides can be used in powders, fogs, or sprays to treat furniture or premises for lice. Several general parasiticides show promise as pediculicides. Avermectins such as abamectin, doramectin, and ivermectin can kill human body lice and livestock lice. Prescribed doses of these compounds can be administered orally, via injection, or as topical applications of powders, dusts, and pour-ons. Some of these compounds have not been approved for use on humans. The development of novel control agents for lice is a constant process because resistance to various pediculicides has developed in lice in many parts of the world (Mumcuoglu, 1996; Burgess, 2004, 2008; Eremeeva et al., 2017).

Healthy, well-groomed, and well-nourished animals are less likely to develop infestations of lice requiring treatment. Because lice are spread mainly via contact, introduction of infested animals should be avoided. Exposure of noninfested animals to facilities shortly after removal of infested animals and exposure to contaminated equipment are also common routes of infestation. Nevertheless, infestations occur despite best efforts. For this reason, an almost bewildering variety of insecticide products and treatment methods are available. The following classes of insecticides have at least one compound registered for use against lice of pets and livestock: organochlorines, organophosphates, carbamates, pyrethrins, synthetic pyrethroids, macrocyclic lactones (avermetins and milbemycins), formamadines, chloronicotinyls and spinosyns, insect growth regulators, and synergists. For a more complete review of current animal pediculicides, the reader is referred to the *Merck Veterinary Manual*, 11th edition (2016) and the *Merck/Merial Manual for Pet Health* (2007).

Small animals like dogs and cats may be treated topically with shampoos, powders, aerosols, rinses, spot-ons, etc. of low mammalian toxicity. Care must be taken to follow the label directions to ensure that a product is indicated for cats as they tend to lick themselves. Lice may be controlled by clipping away matted hair and then bathing the host, followed by the use of a mild insecticidal shampoo, powder, spray, or other treatment according to label directions. A second treatment in 2 weeks may be indicated on the product label to control lice that were present as eggs at the time of initial treatment.

Because of the importance of lice control to the livestock industry, a large number of insecticidal products and treatment methods are available. Withdrawal times after treatment must be observed to avoid objectionable residues in meat or milk. A number of products are approved only for nonlactating cattle or goats. Caution must be taken in treating young, very old, or debilitated animals, as these may be more susceptible to toxicity. Some products may be labeled for several species of livestock; however, in the United States, the specific use must be approved by the Environmental Protection Agency or Food and Drug Administration and indicated on the product label. The label should always be read and understood before

treatment. As with pets, treatment of livestock may require a follow-up treatment to control lice that were present as eggs at the time of the initial treatment.

Beef cattle, dairy cattle, sheep, goats, swine, and equines may be effectively treated with a whole body mist or spray for louse control, although easier and less stressful methods may be available. Animal systemic insecticides, which have gained wide acceptance among livestock producers, are those that enter the circulatory system and eventually reach and control blood-feeding parasites like sucking lice. These insecticides may be administered topically/dermally, orally, or as a subcutaneous injection. The most popular method of application is the **pour-on**, a single line of liquid along the midline of the back (a variation is a single spot on the midline). Pour-on formulations of systemic pediculcides also control chewing lice, mainly through absorption and spread through the skin and hair coat of the treated animal. Several nonsystemic insecticides control lice in a similar manner (i.e., they spread through the skin and the hair coat from the site of application).

In the United States, more cattle are treated for lice than are any other species of livestock. While cattle may be sprayed, most commonly they are treated with the various pour-on formulations, both systemic and nonsystemic. A preventive fall treatment with a systemic insecticide is a common practice among cattle producers for control of both cattle lice and cattle grubs (*Hypoderma bovis* and *H. lineatum*). Fall treatment for lice control prevents an increase in louse infestations to potentially damaging levels over the winter.

Even when cattle are treated in the fall, it may be difficult for the cattle producer to keep his or her animals lice free. The producer will not usually see clinical manifestations of a louse infestation until winter. At this time, infested animals lick and rub in response to the lice. Bare patches may be noted (Fig. 7.19), and if the hair is parted, lice may be visible. When clinical infestations are observed, cattle may be treated with most of the products approved for louse control. Most methods can be used in cold weather; however, some, like the pour-ons, have an advantage in that they do not require elaborate equipment for spraying and the animals do not become completely wet and prone to chilling. Caution must be taken to avoid treatment of grub-infested cattle with a systemic in the winter because of the possibility of the "host−parasite reaction" occasionally seen in cattle when grubs are killed in the esophageal area or in the area of the spinal canal. Various treatment methods can be used during the winter to treat an entire herd or to treat only the animals that exhibit signs of infestation. Animals restrained in a stanchion may be individually dusted by hand. Because louse-infested animals are prone to rub, self-treatment devices like pediculicide-impregnated dust bags, oilers, and back rubbers can aid in the control of lice. Insecticidal ear tags, applied in the summer for fly control, may aid in the control of lice because they are also present on cattle in the summer.

Louse infestations of sheep, goats, and swine may be treated with whole body or pour-on treatments of insecticide. Horses may be treated with a whole-body spray, but because horses may react to sprayer noise, liquid insecticide is often applied as a wipe. For control of severe infestations of swine, an insecticidal dust may be applied to the bedding.

Contact of domestic poultry with potentially infested wild birds should be avoided. When poultry houses are vacated for a new group of birds, it is important to remove all feathers that may be infested with nits. Poultry can be treated with whole body pediculicidal sprays or dusts, which should be repeated in 7−10 days. Although host treatment is most efficacious, bedding materials and cages can also be treated to aid in louse control.

With respect to louse-borne diseases, vaccines have been developed only against epidemic typhus, and none are completely safe or currently approved for widespread use. Live attenuated vaccines have been administered to humans, particularly in certain African nations, in attempts to quell epidemic typhus outbreaks but have not been highly effective.

REFERENCES AND FURTHER READING

Allen, A. (2014). *The fantastic laboratory of Dr. Weigl: How two brave scientists battled typhus and sabotaged the Nazis*. New York: W. W. Norton & Co.

Anderson, J. O., & Andersson, S. G. E. (2000). A century of typhus, lice and *Rickettsia*. *Research in Microbiology, 151*, 143−150.

Andersson, S. G. E., Zomorodipour, A., Andersson, J. O., Sicheritz-Pontén, T., Alsmark, U. C. M., Podowski, R. M., et al. (1998). The genome sequence of *Rickettsia prowazekii* and the origin of mitochondria. *Nature, 396*, 133−140.

Ayyadurai, S., Sebbane, F., Raoult, D., & Drancourt, M. (2010). Body lice, *Yersinia pestis* Orientalis and black death. *Emerging Infectious Diseases, 16*, 892−893.

Bartlett, C. M., & Anderson, R. C. (1989). Mallphagan vectors and the avian filarioids: New subspecies of *Pelecitus fulicaeatrae* (Nematoda: Filarioidea) in sympatric North American hosts, with development, epizootiology, and pathogenesis of the parasite in *Fulica americana* (Aves). *Canadian Journal of Zoology, 67*, 2821−2833.

Beaucournu, J.-C. (1968). Les anoploures de lagomorphes, rongeurs et insectivores dans la region paléarctique occidentale et en particulier en France. *Annales de Parasitologie Humaine et Comparée, 43*, 201−271.

Bedford, G. A. H. (1932). Trichodectidae (Mallophaga) found on South African Carnivora. *Parasitology, 24*, 350−364.

Bildfell, R. J., Mertens, J. W., Mortenson, J. A., & Cottam, D. F. (2004). Hair-loss syndrome in black-tailed deer of the Pacific Northwest. *Journal of Wildlife Diseases, 40*, 670−681.

Bonilla, D. L., Durden, L. A., Eremeeva, M. E., & Dasch, G. A. (2013). The biology and taxonomy of human head and body lice - implications for louse borne disease prevention. *PLoS Pathogens, 9*(11). e1003724.

Bonilla, D. L., Kabeya, H., Henn, J., Kramer, V. L., & Kosoy, M. Y. (2009). *Bartonella quintana* in body lice and head lice from homeless persons, San Francisco, California, USA. *Emerging Infectious Diseases, 15*, 912–915.

Bryceson, A. D. M., Parry, E. H. O., Perine, P. L., Warrell, D. A., Vukotich, D., & Leithead, C. S. (1970). Louse-borne relapsing fever. a clinical and laboratory study of 62 cases in Ethiopia and a reconsideration of the literature. *Quarterly Journal of Medicine, 39*, 129–170.

Burgess, I. F. (2004). Human lice and their control. *Annual Review of Entomology, 49*, 457–481.

Burgess, I. F. (2008). 9. Human body lice. In X. Bonnefoy, H. Kampen, & K. Sweeney (Eds.), *Public health significance of urban pests* (pp. 289–301). Geneva: World Health Organization.

Butler, J. F. (1985). Lice affecting livestock. In R. E. Williams, R. D. Hall, A. B. Broce, & P. J. Scholl (Eds.), *Livestock entomology* (pp. 101–127). New York: Wiley.

Campbell, J. B. (1988). Arthropod induced stress in livestock. *Veterinary Clinics of North America: Food Animal Practice, 4*, 551–555.

Clay, T. (1938). New species of Mallophaga from *Afroparvo congensis* Chapin. *American Museum Novitates*. No. 1008.

Clayton, D. H., Bush, S. E., & Johnson, K. P. (2015). *Coevolution of life on hosts: Integrating ecology and history*. University of Chicago Press.

Dalgleish, R. C., Palma, R. L., Price, R. D., & Smith, V. S. (2006). Fossil lice (Insecta: Phthiraptera) reconsidered. *Systematic Entomology, 31*, 648–651.

Drancourt, M., Tran-Hung, L., Courtin, J., de Lumley, H., & Raoult, D. (2005). *Bartonella quintana* in a 4000-year-old human tooth. *Journal of Infectious Diseases, 191*, 607–611.

Durden, L. A., & Musser, G. G. (1994a). The sucking lice (Insecta, Anoplura) of the world: A taxonomic checklist with records of mammalian hosts and geographical distributions. *Bulletin of the American Museum of Natural History, 218*, 1–90.

Durden, L. A., & Musser, G. G. (1994b). The mammalian hosts of the sucking lice (Insecta, Anoplura) of the world: A host-parasite checklist. *Bulletin of the Society for Vector Ecology, 19*, 130–168.

Emerson, K. C. (1972). *Checklist of the Mallophaga of North America (north of Mexico). Parts I-IV*. Dugway, Utah: Deseret Test Center.

Emerson, K. C., & Price, R. D. (1985). Evolution of Mallophaga and mammals. In K. C. Kim (Ed.), *Coevolution of parasitic arthropods and mammals* (pp. 233–255). New York: Wiley.

Eremeeva, M. E., Capps, D., Winful, E. B., Warang, S. S., Braswell, S. E., Tokarevich, N., et al. (2017). Molecular markers of pesticide resistance and human pathogens in human head lice (Phthiraptera: Pediculidae) from rural Georgia, USA. *Journal of Medical Entomology, 54*, 1067–1072.

Ferris, G. F. (1919-1935). Contributions toward a monograph of the sucking lice. Parts I-VIII. *Stanford University Publications, University Series, Biological Sciences, 2*, 1–634.

Ferris, G. F. (1931). The louse of elephants *Haematomyzus elephantis* (Mallophaga: Haematomyzidae). *Parasitology, 23*, 112–127.

Ferris, G. F. (1951). The sucking lice. *Memoirs of the Pacific Coast Entomological Society, 1*, 1–320.

Foucault, C., Brouqui, P., & Raoult, D. (2006). *Bartonella quintana* characteristics and clinical management. *Emerging Infectious Diseases, 12*, 217–223.

Gibney, V. G., Campbell, J. B., Boxler, D. J., Clanton, D. C., & Deutscher, G. H. (1985). Effects of various infestation levels of cattle lice (Mallophaga: Trichodectidae and Anoplura: Haematopinidae) on feed efficiency and weight gains of beef heifers. *Journal of Economic Entomology, 78*, 1304–1307.

Goates, B. M., Atkin, J. S., Wilding, K. G., Birch, K. G., Cottam, M. R., Bush, S. E., et al. (2006). An effective nonchemical treatment for head lice: A lot of hot air. *Pediatrics, 118*, 1962–1970.

Gratz, N. G. (1997). *Human lice: Their prevalence, control and resistance to insecticides. A review 1985–1997*. World Health Organization/CTD/WHOPES/97.8., 61 pp.

Heath, A. C. G., Bishop, D. M., Cole, D. J. W., & Pfeffer, A. T. (1996). The development of cockle, a sheep pelt defect, in relation to size of infestation and time of exposure to *Bovicola ovis*, the sheep-biting louse. *Veterinary Parasitology, 67*, 259–267.

Hopkins, G. H. E. (1949). The host-associations of the lice of mammals. *Proceedings of the Zoological Society of London, 119*, 387–604.

Ignoffo, C. M. (1959). Keys and notes to the Anoplura of Minnesota. *American Midland Naturalist, 61*, 470–479.

Kaufman, P., Koehler, P. G., & Butler, J. F. (2005). *Cattle tail lice*. ENY-271 (IG127), Entomology and Nematology Department, Florida Cooperative Extension Service, Institute of Food and Agricultural Sciences, University of Florida.

Kim, K. C. (1985). Evolution and host associations of Anoplura. In K. C. Kim (Ed.), *Coevolution of parasitic arthropods and mammals* (pp. 197–231). New York: Wiley.

Kim, K. C., Emerson, K. C., & Price, R. D. (1973). Lice. In R. J. Flynn (Ed.), *Parasites of laboratory animals* (pp. 376–397). Iowa State Univ. Press.

Kim, K. C., Pratt, H. D., & Stojanovich, C. J. (1986). *The sucking lice of North America: An illustrated manual for identification*. Pennsylvania State Univ. Press.

Lancaster, J. L., Jr., & Meisch, M. V. (1986). *Arthropods in livestock and poultry production, Lice* (pp. 321–345). Chichester, England: Ellis Horwood.

Ledger, J. A. (1980). *The arthropod parasites of vertebrates in Africa south of the Sahara* (Vol. IV). Johannesburg: Phthiraptera (Insecta). South African Institute of Medical Research.

Li, G., Ortiz, G., Fournier, P.-E., Gimenez, G., Reed, D. L., Pittendrigh, B., et al. (2010). Genotyping of human lice suggests multiple emergences of body lice from local head louse populations. *PLoS Neglected Tropical Diseases, 4*(3), e641.

Li, H., Bai, J.-Y., Wang, L.-Y., Zeng, L., Shi, Y.-S., Qiu, Z.-L., et al. (2013). Genetic diversity of *Bartonella quintana* in macaques suggests zoonotic origin of trench fever. *Molecular Ecology, 22*, 2118–2127.

Light, J. E., Smith, V. S., Allen, J. M., Durden, L. A., & Reed, D. L. (2010). Evolutionary history of mammalian sucking lice (Phthiraptera: Anoplura). *BMC Evolutionary Biology, 10*, 292.

Marshall, A. G. (1981). *The ecology of ectoparasitic insects*. London: Academic press.

Matthysse, J. G. (1946). Cattle lice, their biology and control. *Bulletin No. 832. New York, Agricultural Experiment Station*.

Mumcuoglu, K. Y. (1996). Control of human lice (Anoplura: Pediculidae) infestations: Past and present. *American Entomologist, 42*, 175–178.

Murillo, A. C., & Mullens, B. A. (2016). Diversity and prevalence of ectoparasites on backyard chicken flocks in California. *Journal of Medical Entomology, 53*, 707–711.

Nelson, W. A. (1984). Effects of nutrition of animals on their ectoparasites. *Journal of Medical Entomology, 21*, 621–635.

Pajot, F.-X. (2000). *Les poux (Insecta, Anoplura) de la region afrotropicale*. Paris: IRD Éditions.

Price, M. A., & Graham, O. H. (1997). Chewing and sucking lice as parasites of mammals and birds. *United States Department of Agriculture—Agricultural Research Service, Technical. Bulletin, 1849*.

Price, R. D., Hellenthal, R. A., Palma, R. L., Johnson, K. P., & Clayton, D. H. (2003). *The chewing lice: World checklist and biological overview*. Illinois Natural History Survey, Special Publication No. 24.

Reed, D. L., Smith, V. S., Hammond, S. L., Rogers, A. R., & Clayton, D. H. (2004). Genetic analysis supports direct contact between modern and archaic humans. *PLoS Biology, 2*(11), e340.

Kahn, C. M. (Ed.). (2016). *The Merck veterinary manual* (11th ed.). Merck & Company, Inc.

Kahn, C. M. (Ed.). (2007). *The Merck/Merial manual for pet health* (1st ed.). Merck & Company, Inc.

Townsend, L., & Scharko, P. (1999). Lice infestation in beef cattle. *Compendium on Continuing Education for The Practicing Veterinarian, 21*(Suppl.), S119−S123.

Tuff, D. W. (1977). A key to the lice of man and domestic animals. *Texas Journal of Science, 28*, 145−159.

Van der Stichele, R. H., Dezeure, E. M., & Bogaert, M. G. (1995). Systematic review of clinical efficacy of topical treatments for head lice. *British Medical Journal, 311*, 604−608.

Veracx, V., & Raoult, D. (2012). Biology and genetics of human head and body lice. *Trends in Parasitology, 28*, 563−571.

Watson, D. W., Lloyd, J. E., & Kumar, R. (1997). Density and distribution of cattle lice (Phthiraptera: Haematopinidae, Linognathidae, Trichodectidae) on six steers. *Veterinary Parasitology, 69*, 283−296.

Zinsser, H. (1935). *Rats, lice, and history*. New York: Bantam, 228 pp.

Chapter 8

True Bugs (Hemiptera)

William L. Krinsky
Division of Entomology, Peabody Museum of Natural History, Yale University, New Haven, CT, United States

The order Hemiptera includes all the insects known as true bugs. Hemipterans are characterized as soft-bodied insects with piercing and sucking mouthparts and usually two pairs of wings. The order traditionally was divided into two major divisions, the Heteroptera and the Homoptera, based on wing morphology. The name Hemiptera (literally, "half-wings") is derived from the members of the Heteroptera ("different wings"), most of which have forewings called **hemelytra**. They are composed of a thickened basal portion, the **corium** and **clavus**, and a somewhat transparent or filmy distal portion, the **membrane**—hence the idea of a half-wing (Fig. 8.1). The hind wings are completely membranous. The difference in texture between the forewings and hindwings in the heteropterans gives this group its name. By comparison, the Homoptera ("same wings") have two pairs of wings that are very similar in character, both being membranous. The wings of homopterans often are held rooflike over the back of the body, whereas the wings of the heteropterans typically are held flat against the dorsum. Traditionally, the Homoptera have been divided into two suborders, the Auchenorrhyncha, having the mouthparts clearly arising from the head, and the Sternorrhyncha, in which the mouthparts appear to arise between the front coxae or are absent. The suborder Auchenorrhyncha is now thought to be polyphyletic, so the suborder has been replaced by three suborders: Fulgoromorpha (planthoppers), Cicadomorpha (cicadas, leafhoppers, treehoppers, spittle bugs, froghoppers) and Coleorrhyncha (moss bugs). Sternorrhyncha includes aphids, adelgids, mealy bugs, phylloxerans, psyllids, whiteflies, and scale insects. For simplicity, the four suborders, other than Heteroptera, are generally called homopterans.

The true bugs, with about 90,000 species worldwide, constitute the largest exopterygote order of insects. The North American fauna has about 16,000 species of hemipterans, about two-thirds of which are homopterans.

The piercing-sucking mouthparts of almost all true bugs enable these insects to feed on a diversity of fluids. The homopterans feed exclusively on plant juices and all of them are terrestrial. The heteropterans include phytophagous, predaceous, and hematophagous species. Common heteropterans include seed bugs, mirid plant bugs, stink bugs, assassin bugs, water striders, backswimmers, water boatmen, and giant water bugs, as well as the medically important kissing bugs and bed bugs.

Various homopterans and some predaceous and phytophagous heteropterans are known to bite humans. Predaceous or phytophagous bugs in at least 20 families of Hemiptera have been reported as occasionally biting or annoying humans by probing with their mouthparts (Table 8.1). Published reports of these bites have been reviewed by Myers (1929), Usinger (1934), Ryckman (1979), Ryckman and Bentley (1979), Alexander (1984), and Schaefer (2003).

Homopteran species known to cause occasional irritation or pain are leafhoppers (Cicadellidae), treehoppers (Membracidae), spittle bugs (Cercopidae), planthoppers (Fulgoridae), and cicadas (Cicadidae). Unlike the generally painless bites by hematophagous species, predaceous and phytophagous bug bites often cause pain or a burning sensation, presumably the result of enzymes and other substances in the saliva that normally digest insect or plant materials. Most of these bites cause only transient discomfort associated with toxic reactions to foreign proteins and the localized erythema and edema that may result.

Common terrestrial heteropterans known to probe human skin in North America are the wheel bug (*Arilus cristatus*) and other assassin bugs (*Reduvius personatus*, *Sinea diadema*, *Melanolestes picipes*), the two-spotted corsairs (*Rasahus biguttatus* and *R. thoracicus*), and certain anthocorids (*Anthocoris musculus*, *Lyctocoris campestris*, and *Orius insidiosus*). There are fewer reports of nabid, lygaeid, mirid, tingid, and rhopalid bugs biting people. A small number of cases involving bites by coreids, enicocephalids, and pyrrhocorids have been reported from other continents.

Medical and Veterinary Entomology. https://doi.org/10.1016/B978-0-12-814043-7.00008-X

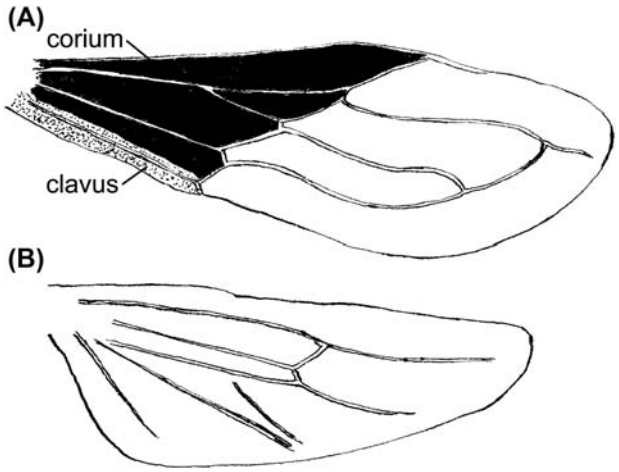


FIGURE 8.1 Wings of a typical triatomine bug (*Triatoma rubrofasciata*). (A) Forewing (hemelytron). (B) Hindwing. *Redrawn and modified from Lent and Wygodzinsky, 1979.*

Humans most often are bitten by predaceous species when they enter habitats in which active predation is occurring or when the predatory species are attracted to house lights and enter dwellings. Some of the larger aquatic predaceous hemipterans can stab with their mouthparts, causing pain similar to that of a wasp sting. Species that most often bite in this way are belostomatids (**giant water bugs**, sometimes called "toe-biters") and notonectids (**backswimmers**). The bite of an assassin bug (*Holotrichius innesi*) found in the Sinai and Negev deserts of Israel is considered more neurotoxic and hemotoxic than the bite of venomous snakes in that region (Caras, 1974).

The painless bites of the bloodsucking species pose the greatest threat to the health and well-being of humans and other animals because of the pathogens that are often transmitted and the blood loss associated with their feeding. The hematophagous heteropteran species of major medical and veterinary importance are the kissing bugs (triatomines) and the bed bugs (cimicids). These ectoparasitic insects are obligate blood feeders, requiring blood for growth and reproduction. The kissing bugs are vectors of the causative agent of Chagas disease, a significant medical problem in Central and South America. Bed bugs are not known to play a role in the transmission of any human disease agents. However, their bites may cause considerable discomfort, and their continued feeding may result in significant blood loss from a host. Some bed bugs feed primarily on nonhuman hosts, such as bats and swallows. The polyctenids, bat bugs that feed exclusively on bat blood, have never been associated with any medical or veterinary problems.

TABLE 8.1 Nonhematophagous Hemiptera That Occasionally Bite Humans

	Common Names	Locations of Published Cases
Cicadomorpha		
Cicadellidae	Leafhoppers	United States (California, Texas); Trinidad; England; North Africa; India; China; Japan; Philippines
Cercopidae	Spittle bugs	India
Membracidae	Treehoppers	United States (eastern)
Heteroptera		
Anthocoridae	Minute pirate bugs	North America; Panama; Brazil; England; Germany; Czech Republic; Sudan; South Africa
Coreidae	Leaf-footed bugs	Chile, Hungary
Enicocephalidae	Gnat bugs	India
Lygaeidae	Seed bugs	Hawaii; Brazil; North Africa; Kuwait; India
Miridae	Leaf or plant bugs	North America; Brazil; Europe; Sudan
Nabidae	Damsel bugs	United States; Canada; Brazil; NewZealand; India
Pyrrhocoridae	Red bugs or stainers	Brazil; North Africa
Reduviidae	Assassin bugs	North America; Brazil; North Africa; Israel; India;
Rhopalidae	Scentless plant bugs	North America
Belostomatidae	Giant water bugs	North America
Corixidae	Water boatmen	Chile
Notonectidae	Backswimmers	North America

KISSING BUGS (REDUVIIDAE)

The **kissing bugs** are so named because most of them are nocturnal species that feed on humans, often biting the faces of their sleeping victims. Another common name for them is **conenoses**, referring to the shape of the anterior part of the head (Figs. 8.2 and 8.3). Various common names in South and Central America and where they are used locally include **barbeiro**, **bicudo**, or **chupão** (Brazil); **vinchuca** (Bolivia, Uruguay, Chile, Argentina); **bush chinche** (Belize); **chipo** (Venezuela); **pito** (Colombia); **chinchorro** (Ecuador); and **chirimacha** (Peru). They are all members of the subfamily Triatominae in the family Reduviidae.

Lent and Wygodzinsky (1979) wrote an excellent monograph on kissing bugs that includes a survey of the external morphological structures, descriptions of triatomine species, and notes on the vector importance of each species. Triatomine biosystematics, including an assessment of the evolutionary history of the subfamily, was reviewed by Schofield (1988). A complete list of triatomine genera and species was given by Jurberg et al. (2015). Biology, taxonomy, public health importance, and control were reviewed by Schofield and Dolling (1993), Schofield (1994), Yamagata and Nakagawa (2006), and Doggett et al. (2015).

FIGURE 8.3 Adult *Triatoma pallidipennis*. *From Centers for Diseases Control and Prevention Public Health Image Library; Photograph by James L. Gathany.*

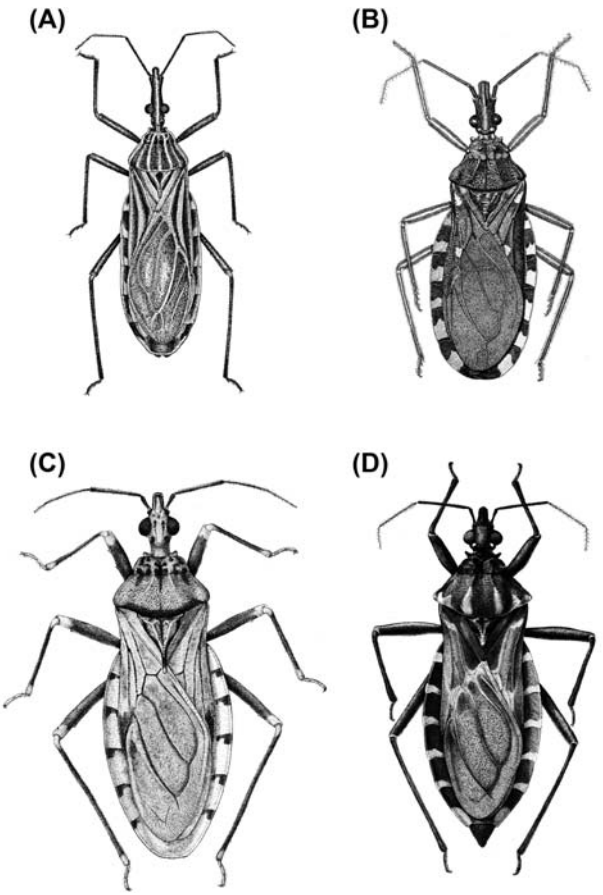


FIGURE 8.2 Triatomine species. (A) *Rhodnius prolixus*. (B) *Triatoma infestans*. (C) *Panstrongylus geniculatus*. (D) *Panstrongylus megistus*. *Courtesy of the American Museum of Natural History.*

Taxonomy

Members of the heteropteran family Reduviidae are commonly called **assassin bugs** because most species attack and feed on other insects. There are 23 subfamilies in the Reduviidae, including the Triatominae, or kissing bugs. Keys for the identification of triatomine species are given by Lent and Wygodzinsky (1979).

The Triatominae is divided into five tribes and 18 genera; most species are known only from the New World, six species (*Linshcosteus*) are found only in India, and seven species (*Triatoma*) occur in southern and Southeast Asia and northern Australia. The only species found in Africa is *Triatoma rubrofasciata*; it is found throughout the tropics, presumably having spread worldwide via ships.

The New World triatomine species occur from just south of the Great Lakes region of the United States to southern Argentina, with all but a few species concentrated in subtropical and tropical regions. The latter areas are considered the likely places of origin for the subfamily. All triatomines have the potential to transmit *Trypanosoma cruzi*, the etiologic agent of Chagas disease. Of the 146 described triatomine species, about half have been shown to be vectors, and fewer than two dozen of these are considered vectors of major epidemiological importance (Table 8.2) (Jurberg et al., 2015; Stevens et al., 2015).

Morphology

Triatomines range in length from 5 to 45 mm, with the majority of species falling in the range of 20–28 mm. Most

TABLE 8.2 Major Triatomine Vectors of *Trypanosoma cruzi* and Their Geographic Occurrence

Species	Geographic Occurrence
Rhodnius prolixus	El Salvador, Guatemala, Honduras, Nicaragua, Colombia, Venezuela, Guyana, Suriname, French Guiana
Triatoma infestans	Peru
Triatoma dimidiata	Mexico south to Ecuador and Peru
Meccus phyllosomus complex	Mexico
Rhodnius pallescens	Panama
Triatoma maculata	Colombia, Venezuela, Guyana, Suriname, French Guiana
Triatoma brasiliensis	Brazil (northeastern)
Triatoma carrioni	Ecuador (southern), Peru (northern)
Panstrongylus chinai	Ecuador, Peru
Rhodnius ecuadoriensis	Ecuador and Peru (northern)
Panstrongylus rufotuberculatus	Costa Rica, Panama, Colombia, Venezuela, Ecuador, Peru, Brazil, Bolivia
Panstrongylus lignarius	Peru
Panstrongylus megistus	Brazil (especially coastal), Bolivia, Paraguay, Argentina, Uruguay
Triatoma guasayana	Bolivia, Paraguay, Argentina
Triatoma patagonica	Argentina
Triatoma sordida	Bolivia, Brazil, Paraguay, Uruguay, Argentina

The two species with the widest geographic distribution are listed first, followed by species arranged generally by their distribution from north to south. In cases where a species is not considered a major vector over the entire range given, countries where the triatomine species is an important vector are underlined.
Based on Lent and Wygodzinsky, 1979; Schofield and Dujardin, 1997; Caravallo et al., 2000; and Jurberg et al., 2015.

species are black or dark brown, often with contrasting patterns of yellow, orange, or red, notably on the **connexivum** (the prominent abdominal margin at the junction of the dorsal and ventral plates) (Figs. 8.2 and 8.3).

The head of an adult triatomine is constricted posteriorly to form a distinct "neck" behind the paired ocelli. Prominent hemispherical compound eyes are situated just in front of the ocelli. The region in front of the eyes is cylindrical to conical, hence the name "cone-nosed" bugs. The antennae are filiform and four-segmented. The beak, or rostrum, is three-segmented and is formed by the labium, which encloses the stylet-like mouthparts. These stylets are modified portions of the maxillae and mandibles that lie within a dorsal channel of the rostrum and are grooved to form a food canal and a salivary canal. When the bug is not feeding, the straight rostrum is held under, and nearly parallel to, the head (Fig. 8.4). In many nontriatomine reduviids, the rostrum is curved and strongly sclerotized.

The dorsal portions of the thorax include a collar, or "neck," a somewhat triangular pronotum, and a scutellum. The undersurface of the prothorax (prosternum) has a stridulatory groove that has fine transverse sculpturing. When the tip of the rostrum is moved anteriorly to posteriorly in this groove, sound is produced, the function of which is mainly defensive.

The forewings, or **hemelytra**, have a leathery basal portion (corium and clavus) and an apical membranous portion (apical clavus and membrane) typical of most heteropterans. The membrane is dusky in most species but may be spotted, or only darkened along the wing veins. The wing veins of the membrane form two elongate closed cells. The hindwings are completely membranous (Fig. 8.1). The hindwings are rarely absent but may be greatly shortened in some species. The relatively long, slender legs are used for walking. In addition to paired simple claws on each tarsus that allow the bug to crawl over rough surfaces, many species have a spongy structure, the fossula, at the apex of the tibia on one or more pairs of legs. The fossulae have adhesive setae on their surfaces that enable the bugs to climb on smooth surfaces, such as leaves and glass.

The triatomine abdomen is 11-segmented, often pointed or lobed in the female but smoothly rounded in the male. In many species, around the periphery of the abdomen, both dorsally and ventrally, are segmental plates (**connexival plates**) connected to the abdominal segments by intersegmental membranes. These membranes allow for expansion of the abdomen during engorgement. The membranes in

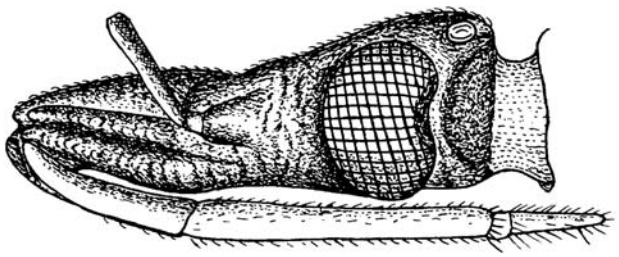


FIGURE 8.4 Lateral view of head of *Triatoma dimidiata*. *From Lent and Wygodzinsky, 1979, courtesy of the American Museum of Natural History.*

different species are folded on themselves in various ways, allowing the plates and membranes to expand in accordion fashion during feeding.

Life History

As in all Hemiptera, triatomines undergo hemimetabolous development. After the egg stage, development occurs through five nymphal instars. Nymphs are distinguished from adults by their smaller eyes, lack of ocelli and wings, and the presence of thoracic lobes where wings will develop. Both sexes of adults and all nymphal instars require blood for their survival and development.

Female bugs are ready to mate 1–3 days after the final molt. Mating involves transfer of a spermatophore from the aedeagus while the male is positioned dorsolaterally to the female with his claspers grasping the end of the female's abdomen from below. Copulation lasts about 5–15 min. Although both sexes usually have had at least one blood meal before mating, unfed males also will mate with fed females.

Oviposition by females begins 10–30 days after copulation. Each female typically deposits only one or two eggs daily, producing a total of 10–30 eggs between bloodmeals. Depending on the species, a single female may produce up to 1,000 eggs in her lifetime, but about 200 is average. Virgin, fed females may lay small numbers of infertile eggs. Each oval egg is about 2–2.5 × 1 mm. The eggs may be white or pink. Most species deposit eggs singly, but some females lay eggs in small clusters or masses. Different species lay eggs freely or glue them to a substrate. Gluing eggs to the substrate is seen in at least two species of *Triatoma* and many species of *Rhodnius*, *Psammolestes*, *Cavernicola*, and *Parabelminus*. In those species that glue their eggs to the substrate, the eggs may be single or in clusters. Eggs of some species turn pink or red before hatching 10–37 days after oviposition, depending on temperature.

The newly emerged nymphs are pink and take a bloodmeal 48–72 h after the eggs hatch. Nymphs must engorge fully to molt (Fig. 8.5), often requiring more than one bloodmeal during all but the first instar. The entire life cycle from egg to adult may be as short as 3–4 months but more commonly takes 1–2 years. The variable developmental times within and between species are related to many factors, including environmental temperature, humidity, host availability, host species, feeding intervals, and the length of nymphal diapause.

Behavior and Ecology

The New World triatomines are found in stable, sheltered habitats that are used by reptiles, birds and a wide variety of mammals for their nests, roosts, or burrows. Kissing bugs

FIGURE 8.5 Triatomine nymphs engorging on human foot. *Courtesy of Robert B. Tesh.*

can be divided into three general habitat groups: sylvatic, peridomestic, and domestic. **Sylvatic** forms inhabit nests and burrows, as well as a wide array of natural hiding places such as caves, rock piles, fallen logs, tree holes, hollow trees, palm fronds, bromeliads, and other epiphytes. These habitats attract amphibians, lizards (e.g., iguanas), opossums, rodents (e.g., porcupines), armadillos, sloths, bats, and other mammals, on which the triatomines feed. The **peridomestic** species use domestic animals as hosts by living in chicken coops and other bird enclosures, stables, and corrals and in rabbit and guinea pig houses. Because *Triatoma infestans* infests the latter, as well as wild guinea pig habitats, this species may have entered the domestic habitat thousands of years ago when people in South America began breeding guinea pigs for use as food. The **domestic** (domiciliary) species, exemplified by *T. infestans*, have colonized human habitations where they depend on human or domestic animal blood as their source of nourishment. The domestic triatomine species are almost exclusively associated with humans and their pets and are often carried from one region to another in vehicles or concealed in household materials.

Many of the so-called peridomestic species, as well as a few domestic ones, have maintained sylvatic adaptations and may migrate from wild hosts to domestic animals and humans, depending on the availability of suitable habitats and hosts. Peridomestic species sometimes fly to lights of houses and thereby are attracted at night to feed on sleeping humans. Passive transport of certain species to human dwellings may occur when palm fronds containing attached triatomine eggs are used as roofing material. This is commonly the case with *Rhodnius prolixus* and other avian-feeding species that cement their eggs to the leaves in and around arboreal bird nests. The significance of birds in dispersing triatomines is not known, although eggs and

young nymphs of *R. prolixus* have been found among the feathers on storks, and *Triatoma sordida* nymphs have been found in the plumage of sparrows.

In whatever habitat triatomines are found, they tend to be secretive, hiding in cracks and crevices of natural and artificial materials (e.g., debris of nests and burrows, in rock crevices and piles of vegetation); in building materials such as wood, shingles, thatch, and palm fronds; and in human dwellings in cracks in the walls, behind pictures or other wall-hangings, in bedding and mattresses, in furniture, boxes, suitcases, piles of papers or clothes, and other accumulated materials that provide shelter during the day. Shaded crevices that provide extensive bodily contact with a rough, dry surface are preferred. Nymphs of many species are camouflaged by dirt and debris with which they cover themselves.

Most species of triatomines are nocturnal and actively seek blood from diurnal hosts that are resting or sleeping at night. In some cases, bugs will feed in daylight, typically on hosts that are nocturnal. Kissing bugs can survive for months without a bloodmeal, making them well adapted to nest habitats in which hosts may be present only intermittently with long intervals in between. When hosts are available, bugs commonly feed every 4−9 days. Individual species show definite host preferences and may favor bats, birds, armadillos, wood rats, or humans. Those favoring the latter species in South America are most important in the epidemiology of Chagas disease.

As in other hematophagous arthropods, feeding behavior is initiated by a combination of physical and chemical factors. Heat alone stimulates *Rhodnius prolixus* to probe, the heat receptors being located on the antennae. Carbon dioxide, which induces feeding responses in various hematophagous arthropods, causes increased activity in triatomines and may alert them to the presence of a host. The possible role of aggregation pheromones in attracting bugs to a host is unclear, but a pheromone in the feces of nymphal and adult *Triatoma infestans* and in nymphal *R. prolixus* attracts unfed nymphs. These species defecate soon after feeding on or by the host, so that such a pheromone might attract other bugs to a source of blood.

The probing response begins when the rostrum is swung forward. The third segment is flexed upward so that optimal contact with a host occurs when the bug is at the side and just below a host. The serrated mandibular stylets are used to cut through the epidermis of the host and then anchor the mouthparts while the maxillary stylets probe for a blood vessel. When a vessel is penetrated, the left maxillary stylet slides posteriorly on the right stylet, disengaging the two stylets so that the left folds outward from the food canal. The purpose of this action is not known; it may allow a larger opening for ingestion of blood cells, or it may be a mechanism for holding the capillary lumen open (Lehane, 2005).

The amount of blood ingested depends on the size of the bug and the duration of feeding. This, in turn, is governed by the presence of chemicals in the blood of the host that stimulate onset of feeding and by stretch receptors in the abdomen of the bug that stimulate cessation. Known phagostimulants of triatomines include various nucleotides and phosphate derivatives of nucleic acids. Triatomine saliva contains an anticoagulant to help maintain blood flow during feeding and nitric oxide, which has antiplatelet and vasodilatory effects.

The time required to engorge fully varies from 3 to 30 min. During feeding, the abdomen becomes visibly distended. Adult bugs may imbibe blood equivalent to about three times their body weight, while nymphs may imbibe 6−12 times their unfed weight. Bloodmeals are stored in the anterior, widened portion of the midgut before the blood is passed to the narrower, posterior portion where digestion occurs. After engorging, the bug removes the rostrum from the host and, in most species, defecates on or near the host before crawling away to seek shelter. The interval between feeding and defecation is a major factor in determining the effectiveness of a species as a vector of *T. cruzi*. Schofield (1979) reviewed the behavior of triatomines, with particular attention to their role in trypanosome transmission.

Public Health Importance

Triatomine species that are efficient vectors tend to cause little or no pain when they feed. At least one substance in triatomine saliva has analgesic properties. The bugs stealthily approach their sleeping hosts and engorge without causing much, if any, awareness (Fig. 8.6). However, immediate and delayed skin reactions to bites of *Triatoma infestans* and *Dipetalogaster maxima* have been observed. These reactions were not clearly correlated with

FIGURE 8.6 Adult triatomine (*Rhodnius* sp.) feeding. *Courtesy of William L. Krinsky.*

previous exposure to bugs. Pruritic (itchy) skin reactions following triatomine bites tend to enhance transmission of *T. cruzi* by stimulating the bitten individuals to scratch infective feces into the bite wounds.

Some individuals react to triatomine feeding with mild **hypersensitivity reactions** such as pruritus, edema, and erythema. These reactions occur most often in response to triatomine species that are not efficient vectors of *T. cruzi* to humans. Within the latter group of species are *Triatoma protracta* in California and *T. rubida* in Arizona. These triatomines fly to light and have been known to invade homes situated within natural wood rat habitats. In a small number of cases, individuals have developed severe systemic reactions, including anaphylaxis, following bug bites. Immunotherapy involving multiple injections of *T. protracta* salivary gland extract has been successful in ameliorating the effects of the bite (Marshall and Street, 1982; Rohr et al., 1984).

Chagas Disease (American Trypanosomiasis)

In 1907, while on an antimalarial campaign in Minas Gerais, Brazil, Carlos Chagas was introduced to the bloodsucking triatomines ("barbeiros"). He found what is now known to be *T. cruzi* in the hindguts of several bugs; within 2 years, he recognized this same flagellate protozoan in domestic animals and in a sick 2-year-old girl. Chagas disease and its epidemiology were thus first discovered in reverse fashion from that of most diseases. In this case the vector was found first, the nonhuman vertebrate hosts of the parasite second, and the human pathology last. The first report of the disease by Chagas was published in 1909, only 20 months after Chagas became aware of the existence of bloodsucking bugs. Chagas disease became known as American trypanosomiasis to differentiate it from African trypanosomiasis (African sleeping sickness), the disease caused by trypanosomes transmitted by tsetse flies in Africa.

Several bibliographies on the vast literature on Chagas disease and its epidemiology have been published, including those by Olivier et al. (1972) and Ryckman and Zackrison (1987). An excellent review of the etiologic agent and its biological associations is provided by Hoare (1972).

Triatomine species that are important **vectors** of *T. cruzi* are listed with their geographic occurrences in Table 8.2. *Triatoma infestans* is probably most often responsible for transmission of the trypanosome to humans because this species has colonized human dwellings over a wide geographic range in South America. *Rhodnius prolixus* is the second most important vector because it is widely distributed in sylvatic and domestic habitats in northern South America and is found only in domestic populations in Central America. *Triatoma dimidiata* is found in southern Mexico, throughout Central America and into northern South America. *Panstrongylus megistus* is generally considered a major vector in the humid coastal regions of eastern Brazil, and *T. braziliensis,* occurring in 12 populous Brazilian states, is the most important vector in the semiarid areas of northeastern Brazil. These five triatomine species are thought to be associated with the majority of Chagas disease cases in Central and South America.

The basic features of the life cycle of *T. cruzi* are shown in Fig. 8.7. Broad and slender trypanosomes (**trypomastigotes**) circulating in the blood of an infected vertebrate host (Fig. 8.8) are imbibed by the triatomine during feeding. In the proventriculus of the bug, the broad trypomastigotes transform into **sphaeromastigotes** and slender **epimastigotes**. The latter multiply via binary fission in the bug midgut, and as early as the fifth or sixth day after feeding, they occur in tremendous numbers that carpet the walls of the rectum of the bug. As early as the seventh or eighth day after feeding, these epimastigotes become infective **metacyclic forms** (trypomastigotes) that pass out of the bug in the feces and Malpighian tubule secretions. Although alternative *T. cruzi* developmental schemes have been proposed, the life cycle generally is thought to be limited to the gut of the bug, and infective forms occur only in the hindgut and rectum.

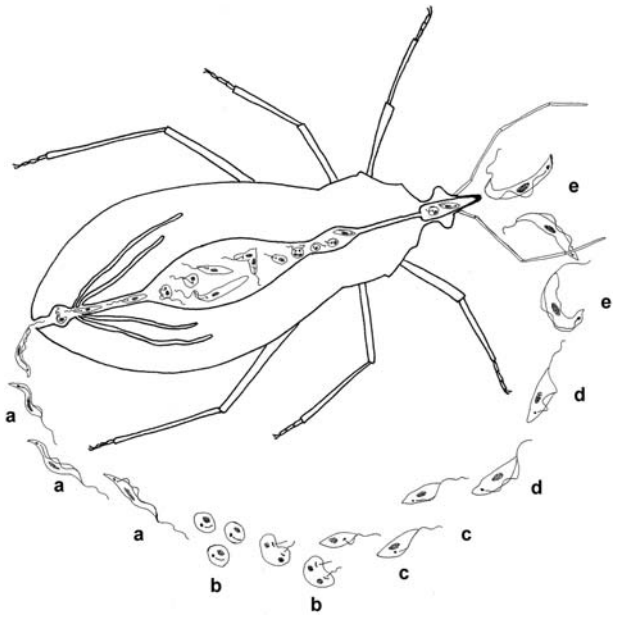


FIGURE 8.7 Life cycle of *Trypanosoma cruzi* in a triatomine bug and vertebrate host. (A) Metacyclic forms excreted by triatomine. (B) Amastigotes (in vertebrate host). (C) Epimastigotes (in vertebrate host). (D) Trypomastigotes (in vertebrate host). (E) Bloodstream forms ingested by triatomine. *Courtesy of W. L. Krinsky.*

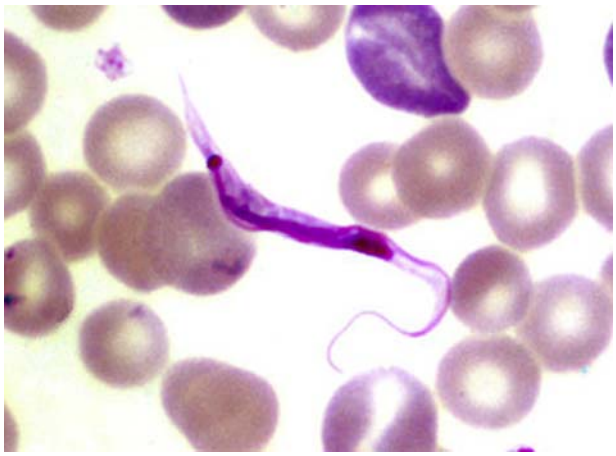


FIGURE 8.8 *Trypanosoma* species in blood. *Centers for Disease Control and Prevention, Public Health Image Library; Photograph by Mae Melvin.*

Transmission to another vertebrate host occurs by this posterior-station (stercorarian) route. The trypanosomes infect the vertebrate when infective feces are rubbed into the bite site or other breaks in the skin. Transmission probably occurs most often when infective feces come in contact with the mucosal membranes of the nose or mouth or the conjunctivae of the eyes. Infected triatomines of those species that defecate while engorging or soon after feeding, while still on the host, are the most likely vectors of *T. cruzi*.

The entire development of *T. cruzi* in the lumen of the triatomine gut takes about 6−15 days or longer, depending on the ambient temperature and developmental stage of the bug (6−7 days in first-instar nymphs and 10−15 days in older nymphs and adults). Once a nymph or an adult is infected, it is infective for life. Trypanosomes do not pass via the eggs or spermatophore to the next generation. Cannibalism and coprophagy have both been observed in laboratory colonies of triatomines and have been suggested as possible modes of transmission from bug to bug. However, the minimal infection rate among uninfected bugs housed with infected ones indicates that such transmission is of minor consequence under natural conditions. Coprophagy is the means by which symbiotic bacteria essential for development of triatomines are transferred from adults to newly hatched nymphs (Beard et al., 2002).

Besides contamination of the skin or mucosal tissues, an alternative mode of transmission from bug to humans is ingestion of food contaminated with triatomine fecal matter. Various tropical fruit juices (made from guava and acai and other palm berries) in which infected triatomines have been accidentally ground up have been the sources of infection (Coura, 2013). Such ingestion has been the cause of several hundred Chagas cases in the Amazon in areas where the triatomine species are not known to invade dwellings. Wild and domestic vertebrate animals may ingest whole infected bugs (Miles, 1983), and that is thought to be the most common mode of transmission to wood rats in North America. The possibility of additional trophic levels in such transmission has been demonstrated by the infection of rodents and dogs after their ingestion of house flies that previously had ingested feces of infected triatomines (Hoare, 1972). Carnivores may become infected by feeding on infected prey (Miles, 1983). Human infection has occurred after accidental contact with freshly squashed triatomine bugs. Trypanosomes in the hindguts of dead triatomines may maintain their infectivity for up to 30 days. The "fecal rain" from triatomine-infested ceilings that falls on inhabitants of some tropical houses may be another source of infection (Miles, 1983). Although inadvertent contamination with infective triatomine feces is probably the most common route of human infection, some cultural practices involve deliberate contacts with triatomines. These include the eating of *Triatoma picturata* ("chinche de compostela") for their supposed aphrodisiac properties in Nayarit, Mexico, and the rubbing of feces of *T. barberi* ("chinche voladora") onto the skin to cure warts on children in Oaxaca, Mexico. Both of these activities expose individuals to a high risk of trypanosome infection (Salazar-Schettino, 1983).

Other modes of transmission from person to person include infection via blood transfusion, organ transplantation, laboratory accidents, transplacental infection, and infection during childbirth. Although infection of children via the breast milk of infected mothers is rare, the risk of infection during nursing is significantly increased when bleeding of the nipples occurs.

Arthropods other than triatomines have been infected with *T. cruzi* in laboratory and field studies. Both the common bed bug (*Cimex lectularius*) and the African argasid tick *Ornithodoros moubata* have been infected by feeding on infected hosts and have maintained infective metatrypanosomes in their guts after normal cyclical development of the parasite. However, these arthropods are not known to have any role in natural transmission cycles.

Many individuals who become infected with *T. cruzi* do not develop symptoms early in the course of infection. These subclinical cases may or may not develop chronic disease. In a small number of cases, especially in children, an acute clinical form of Chagas disease occurs after the initial infection. Significant mortality (5%−15%) occurs among those showing acute disease.

Acute Chagas Disease

The acute form of Chagas disease begins with an area of erythematous and indurated skin, called a **chagoma**, at the site of parasite entry. If the infective material is rubbed into the eye, periorbital edema called **Romaña's sign** appears (Fig. 8.9). This swelling, which may be accompanied by

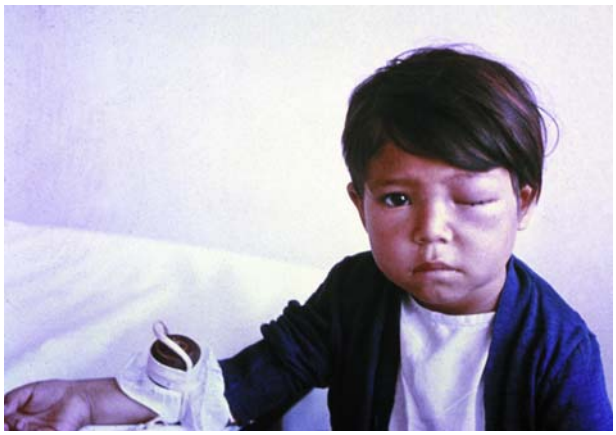


FIGURE 8.9 Romaña's sign in boy undergoing xenodiagnosis, the feeding of laboratory-reared triatomine bugs on a patient as a means of detecting infection with trypanosomes. *Courtesy of U.S. Public Health Service.*

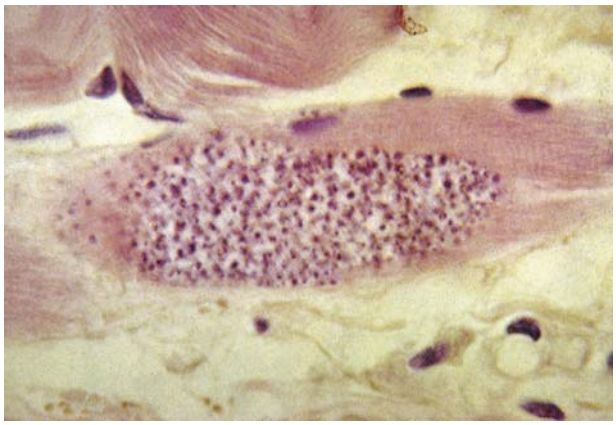


FIGURE 8.10 *Trypanosoma cruzi* amastigotes developing in heart muscle. *From Peters and Gillies, 1981.*

regional lymph node enlargements, may last for 2–6 weeks. The tissue changes result from intracellular development of amastigote trypanosomes in subcutaneous tissue and muscles. The amastigotes multiply and transform into trypomastigotes that enter the bloodstream. Other signs of acute Chagas disease include fever, general enlargement of lymph nodes, enlarged liver and spleen, and skin rashes. In children less than two years old who develop myocarditis, cardiac insufficiency, and meningoencephalitis, the disease is almost always fatal. Most persons with the acute disease survive and enter the **indeterminate phase** of the disease, a stage in which the person appears healthy but still has the potential to develop serious chronic disease.

The indeterminate phase begins when antibodies to *T. cruzi* become detectable by serological testing, and trypanosomes, if detectable at all, may be demonstrated only by special methods, such as culturing blood or xenodiagnosis (Fig. 8.9). Persons in this phase may transmit *T. cruzi* via blood, organ transplants, or congenitally. The indeterminate phase may last indefinitely without further signs or symptoms of disease; in 20%–30% of cases, however, chronic Chagas disease develops within the next few years or as long as 25 years after the initial infection.

Chronic Chagas Disease

This is most often characterized by cardiac symptoms including palpitations, dizziness, chest pain, and peripheral edema and sometimes fainting. The causes of these symptoms are various forms of arrhythmias, thromboembolism, and heart failure, which may lead to sudden death or persist for several years. The underlying pathology for the cardiac abnormalities is the development of amastigote trypanosomes in the cardiac muscles, accompanied by the degeneration of cardiac muscle fibers (Fig. 8.10), followed by fibrosis. The second most often seen type of chronic Chagas disease that occurs south of the Amazon involves enlargement of the esophagus or, less often, the colon. These conditions are known as **megasyndromes**. Enlargement of these organs is accompanied by gastrointestinal discomfort, including pain on eating and prolonged constipation in the case of megacolon. The often mammoth enlargement of the esophagus and colon is the result of pathologic destruction of myenteric ganglion cells, so that autonomic parasympathetic innervation (Auerbach plexus) is greatly diminished. Individual patients may develop chronic cardiac disease, megaesophagus, and megacolon, or only one or two of these syndromes. The most common outcome in patients with chronic Chagas disease is damage to heart muscle (cardiomyopathy) and conduction fibers, which lead to various forms of heart block and, in South America, congestive heart failure. Several years of suffering with cardiac symptoms may precede cardiac failure, which may, after as long as a few more years, result in death.

The restlessness, agitation, irritability, insomnia, and various other vague discomforts experienced by patients with chronic Chagas disease have led medical historians to speculate on the basis of Charles Darwin's writings that he may have suffered from this disease. Furthermore, Darwin's palpitations and chest pains were brought on by emotional rather than physical stress, a phenomenon noted in Chagas' first patient when she was a middle-aged woman. Evidence that Darwin may have become infected with Chagas disease trypanosomes while he was in Argentina comes from his own description of being attacked by "the *Benchuca*, a species of *Reduvius*, the great black bug of the Pampas" (Voyage of the H.M.S. Beagle, March 26, 1835). It is impossible to make a definitive diagnosis of Darwin's illness in the absence of pathologic material. The fact that Darwin had some of the same complaints before his voyage on the "Beagle" further complicates the speculation.

In suspected acute Chagas disease, direct examination of anticoagulated blood, buffy coat preparations, or concentrated serum may reveal living trypanosomes. Blood culture and fixed blood smears are most useful for confirming the diagnosis. PCR (polymerase chain reaction) techniques provide the most sensitive assays for the diagnosis of acute phase and early congenital Chagas disease, but PCR is not a reliable diagnostic tool for chronic Chagas disease. Enzyme-linked immunosorbent assay (ELISA) and immunofluorescent antibody assay are being used more extensively for diagnosing the disease. The reliability of these tests varies because there is a lack of standardization of the testing materials used in different countries and different laboratories. The use of at least two testing methods is often recommended to increase the accuracy of diagnosis. PCR assays have been used in chronic Chagas cases to evaluate treatment success or failure after drugs have been administered.

The most sensitive procedure for recovering trypanosomes from both acute and chronic Chagas patients is **xenodiagnosis** (Fig. 8.9). This involves feeding uninfected, laboratory-reared triatomines on a patient, holding the bugs in the laboratory for about 30 days, and then dissecting the hindguts of the bugs to look for trypanosomes. This procedure uses the bugs as living culture chambers. It takes advantage of the natural transmission cycle in which even very small numbers of trypanosomes ingested by a triatomine multiply in great numbers in the alimentary tract. Xenodiagnosis has been most successful when triatomine species and geographic strains from the area in which a person has been infected are used. As in all parasitic diseases, a careful history of travel or activities that may have led to infection is essential for differential diagnosis of the disease.

Diagnosis of chronic Chagas disease requires demonstration of *T. cruzi*-specific antibodies in a patient who has the characteristic cardiac dysfunction and/or megasyndromes. Positive xenodiagnosis and antibody testing alone may only indicate that an individual has been exposed to the parasite and is in the indeterminate phase.

Chagas disease is now considered the most serious parasitic disease of the Americas. It is endemic in 21 Latin American countries. *Trypanosoma cruzi* is enzootic in the southern United States. As a result of travel, Chagas disease is present in Canada, Europe, Australia, and Japan. An estimated eight million people in Central and South America are infected with *T. cruzi*, with about 28,000 new cases and 12,000 deaths occurring each year. An estimated 75 million people in the Americas live in areas of exposure and risk infection with *T. cruzi*. More than half the populations of some rural villages are antibody positive. Although still enzootic in many Central and South American countries (Fig. 8.11), the nations now most affected by infection and disease are Brazil, Mexico, Argentina, Bolivia, and Colombia. The type of disease observed varies from country to country. Cardiomyopathy and megasyndromes are common in Brazil, but cardiac disease alone is common in Venezuela. Cardiac abnormalities are present, but less prevalent, in Colombia and Panama. Cardiac disease is even less common among seropositive people in other parts of Central America and Mexico, where cardiac problems, if they occur, present later in life. Recent genetic studies have enabled the identification of six discrete taxonomic units of *T. cruzi* and recognition of various genotypes. Genetic typing of *T. cruzi* from patients with different clinical presentations is being studied to look for correlations between *T. cruzi* pathology and parasite genotypes (Ramirez et al., 2010).

FIGURE 8.11 Distribution map of human Chagas disease based on 2010 data from various health organization sources. *Revised and re-drawn; courtesy of William L. Krinsky.*

The social and economic burdens caused by Chagas disease are primarily associated with morbidity rather than with mortality. Chronically infected individuals often suffer for decades from weakness and fatigue that interfere with their productive enjoyment of life.

Chagas disease affects mostly the poorest people in the population. Typically the incidence of infection is directly

associated with poor housing construction and proximity to domestic animal quarters or sylvatic habitats. Substandard houses such as rough-walled huts made of mud and sticks or adobe mud bricks, often roofed with thatch, provide abundant cracks and crevices in which triatomines can hide during the day and crawl out at night to feed on sleeping people and domestic animals. *Rhodnius prolixus*, naturally occurring on living palms, is especially abundant in palm roofs, whereas *P. megistus*, a species that is naturally found in hollow trees, favors the interstices of timber-framed, mud houses. Thousands of triatomines may inhabit individual houses, causing each inhabitant to be bitten by dozens of bugs each night. *Triatoma infestans*, the most important vector of *T. cruzi* in southern South America, also may occur inside of houses with plastered walls and tiled roofs. The ability of triatomines to fly has even led to their intrusion into luxury high-rise buildings.

More than 100 species of mammals have been found infected with *T. cruzi*. Although triatomines feed on a variety of birds, reptiles, and amphibians, only mammals can be infected. Within domestic settings, humans, dogs, cats, and mice often are involved. Cats may become infected after the ingestion of mice, whereas all domestic mammals have the potential to become infected after the ingestion of triatomines. In peridomestic cycles, chickens are excellent sources of blood for the triatomines. In sylvatic cycles, opossums and various rodents are often important reservoir hosts that are readily fed upon by bugs. Squirrels are strongly associated with triatomines in peridomestic surveys. The broad host range of the parasite and the large number of potential triatomine vectors over a vast geographic area contribute to the complexities of the natural cycles of *T. cruzi* in any given region. *Trypanosoma cruzi* genotyping is being used to identify which hosts or triatomines are involved in the transfer of the parasite from one cycle to another. Rats and opossums have been implicated in the transfer of sylvatic *T. cruzi* to domestic cycles (Pennington et al., 2015; Messenger et al., 2015).

In general, the greatest transmission to humans occurs in regions where domestic triatomines are abundant. Not only are these species adapted to survive in close proximity to man, but most of these species have feeding patterns that cause them to defecate very soon after engorgement while still on or near the host. The lack of truly domestic species of triatomines found north of Mexico has been cited as a major reason why there have been only seven cases of Chagas disease acquired from triatomines in the United States.

Triatomine species repeatedly found to harbor natural infections with *T. cruzi* in the United States are *Triatoma sanguisuga* in Pennsylvania, Ohio, Maryland, and south to Florida; *T. lecticularia* in Pennsylvania, Illinois, Maryland, and south to Florida; *T. gerstaeckeri* in Texas and New Mexico; *T. protracta* in Texas, New Mexico, Arizona, and California; *T. rubida* in Texas and west to California; and *T. recurva* in Arizona. All of these species except *T. recurva* are commonly associated with wood rats (*Neotoma* spp.), which are also naturally infected with *T. cruzi*. Other potential reservoirs of infection for *T. sanguisuga* are raccoons, armadillos, and opossums. Triatomines rarely colonize houses in the United States, and the bugs are largely restricted to sylvatic habitats. The low incidence of transmission of *T. cruzi* to humans by triatomines in North America is attributed to these factors as well as the longer time delay between feeding and defecation seen in North American triatomine species compared with that of important Latin American vector species (Wood, 1951; Martinez-Ibarra et al., 2007; Zeledon et al., 1977; Klotz et al., 2009).

In the United States, triatomine-associated Chagas disease is much less likely than is transfusion-acquired infection. Correlation of seroprevalence of *T. cruzi* in Latin American countries with the numbers of immigrants who originated in those countries where Chagas disease is endemic indicates that an estimated 300,000 persons infected with *T. cruzi* now live in the United States. Twenty-one cases of Chagas disease have been documented as resulting from blood transfusions in the United States. Consequently, careful screening of blood donors, begun within the past 10 years, is essential to prevent the use of blood from chronically infected, asymptomatic individuals. An Enzyme Linked Immunosorbent Assay is now often used to detect *T. cruzi* antibodies in donated blood, followed by radioimmunoprecipitation assay as a confirmatory test (Bern et al., 2011).

Other Human Parasites Associated With Kissing Bugs

Trypanosoma rangeli, a nonpathogenic trypanosome found in Central and South America, is also transmitted by triatomines. *Rhodnius prolixus* is the chief vector. *Trypanosoma rangeli* is morphologically and serologically distinguishable from *T. cruzi* and, unlike the latter, may be transmitted via the saliva of the bug. It is also found naturally in a wide array of mammals, including monkeys, dogs, opossums, anteaters, raccoons, and humans.

As with all hematophagous arthropods, incidental infections with various blood-borne pathogens can occur when these invertebrates feed on viremic, bacteremic, or parasitemic hosts. Although interest in Hepatitis B virus (HBV) led to the suggestion that triatomines might at times disseminate this virus, epidemiological evidence is lacking. Experimental feeding by fifth-instar nymphs of *T. infestans* on asymptomatic Human immunodeficiency virus (HIV)-infected patients demonstrated that the HIV can survive in the bugs 3–7 days after engorgement, but the bugs do not transmit the virus.

Veterinary Importance

Triatomines transmit *T. cruzi* to a variety of domestic and wild mammals, including opossums, armadillos, rodents, carnivores, and monkeys. Depending on the strain of the trypanosome, the species and age of the host infected, and other poorly understood factors, the infection can lead to disease. Myocarditis and megaesophagus, similar to the conditions seen in humans, have been observed in dogs. **Canine trypanosomiasis** is of veterinary importance in Central and South America, and many cases have been recognized in southern Texas (USA). Clinical indications of infection in dogs include dyspnea and ascites. There is little evidence that nonhuman wild animal hosts that serve as natural reservoirs of *T. cruzi* develop any pathology.

Trypanosoma rangeli, which unlike *T. cruzi* is usually transmitted by salivary inoculation, occurs within the distribution of *Rhodnius prolixus*, its chief vector. The public health importance of *T. rangeli* in veterinary medicine lies in differentiating this common parasite from the pathogenic *T. cruzi*. *Trypanosoma conorhini* is a nonpathogenic parasite of rats transmitted by *Triatoma rubrofasciata* in many tropical regions of the Old and New World. It appears to have a tropicopolitan distribution identical to that of *T. rubrofasciata*, a domiciliary species of the tropics and subtropics. *Trypanosoma conorhini* is spread by posterior-station transmission (Hoare, 1972).

Heavy infestations of triatomines in poultry houses in Central America can cause chronic blood loss in chickens. Even though no avian pathogens are involved, the impact of constant blood-feeding may result in significant morbidity and, in the case of young birds, mortality.

Prevention and Control

The goal of any prevention or control program is to reduce contact between humans and kissing bugs in order to prevent discomfort from bites and the more serious problem of Chagas disease. Control of Chagas disease involves using insecticides, improving housing conditions, and screening blood used for transfusions. Residual insecticides sprayed onto houses or applied to walls in paints are effective in controlling triatomines for a few months after application, but control of populations of insecticide-resistant bugs requires selective use of different pesticides. Long-term control of any of the triatomines requires careful surveillance and changes in and around traditional homes. Some simple surveillance techniques include use of sensor boxes for passive collecting and placing pieces of colored paper on the inside walls of houses to assess the presence of triatomines by observing their fecal patterns on the paper, providing an indicator of triatomine activity. Triatomine populations in houses have been reduced by eliminating hiding places by covering rough walls and floors with plaster (recently made from local materials [i.e., mud, sand, and volcanic ash]) and replacing thatched roofs with tin or tile. Around houses, the removal of wall hangings, firewood, accumulated debris or vegetation, and animal (chicken, guinea pig) enclosures not only excludes triatomines but also prevents houses treated with insecticides from subsequently being colonized by peridomestic or sylvatic species.

Defecation by *T. infestans* and *R. prolixus* soon after feeding is dependent on full engorgement by the bugs, which, in turn, appears to be related to the density of the bug population in a given habitat; the greatest chance of engorgement is in low-density populations. Presumably, high bug densities with constant host resources lead to smaller bloodmeal sizes and a slower rate of defecation. Therefore, the chance of inhabitants becoming infected with *T. cruzi* tends to be greatest in newly colonized houses where the bug population is rising or in houses being repopulated after vector control has been instituted.

During the past 40 years, *R. prolixus* has almost completely disappeared from Costa Rica, El Salvador, and parts of Mexico. This decline has been attributed to improved housing and widespread insecticide use by malaria eradication campaigns. Since 1991, multinational campaigns specifically aimed at reducing Chagas disease have been launched. The Southern Cone Initiative is a two-pronged program that includes large-scale use of residual insecticides in domestic and peridomestic structures in infested villages and universal blood screening to eliminate transmission from infected donors. Within a decade of the start of the program, the incidence of Chagas disease was reduced by greater than 65% in the countries involved (Argentina, Brazil, Chile, Paraguay, and Uruguay). Transmission by the major vector, *T. infestans*, was eliminated in Uruguay and Chile and in parts of the other countries. Similar campaigns have been established for Chagas disease control in Central America and Mexico and in the Andean and Amazonian countries (Coura, 2013; Yamagata and Nakagawa, 2006).

The drugs available for the treatment of acute Chagas disease, such as nifurtimox and benznidazole, are reasonably effective in preventing the development of chronic disease. However, they require long treatment regimens and neither is known to affect the course of chronic Chagas disease once it develops. Routine screening of the blood supply is necessary to prevent transmission of *T. cruzi* via blood donors in endemic areas and in countries with large Latin American immigrant populations. Seropositive blood can be decontaminated with crystal violet (250 mg/L) and storage at 4°C for at least 24 h.

Various biological control approaches, including the use of juvenile hormone mimics, predatory arthropods, and parasitic wasps (e.g., the scelionid *Telenomus fariai*), have been studied for the control of triatomines. However, no

biological control method has been found for effective, widespread use in Central and South America.

An experimental approach for controlling *T. cruzi* involves genetically modifying triatomine bacterial symbionts so that triatomines infected with them produce antitrypanosomal gene products (Hurwitz et al., 2012).

BED BUGS (CIMICIDAE)

The family Cimicidae includes species known by several common names, including **bed bugs**, **bat bugs,** and **swallow bugs**. All species in this family are wingless, obligate hematophagous ectoparasites. Their medical and veterinary importance relates primarily to the loss of blood and discomfort caused by their feeding on vertebrate hosts. The monograph on the Cimicidae by Usinger (1966) is still the most complete work on the taxonomy of the group, and the modern treatise by Doggert et al. (2018) provides a comprehensive review of the history of infestations, their global resurgence, basic biology, medical impacts, control and management of bed bugs, and legal implications associated with infestations.

The scientific name for the common human bed bug, *Cimex lectularius*, literally means "bed bug" (*L. cimex* = "bug" and L. *lectularius* = "of the bed"). More than 50 common names have been given to bed bugs in different countries; some of these are mahogany-flat (Baltimore), heavy dragoon (Oxford), red coat (New York), wall louse (*Wandlaus, Wegluis*, and *Wanze* [German]), *Wägglus* (Swedish), *Vaeggelus* (Danish), *Piq-seq* (Chinese), *Chinche* (Old Spanish), *Chinga* (Gallic), *Nachtkrabbler* (night crawler, German), *Tapetenflunder* (wallpaper flounder, German), *Punaise* (stinker, French), *Perceveja* (pursuer, Portuguese), *Lude* (Finnish), *Plostice* (flat, Czech), *klop* (Russian), *bug* (ghost, goblin, British), *Buk* (Arabic), *Fusfus* (Syrian), *Pishpesh* (Hebrew), *Ekukulan* (Douala-Bantu), *Kunguni* (Swahili), *Uddamsa* (biter, Sanskrit), *Rep* (Vietnamese), *Nankinmusi* (Nanking bug, Japanese), and *Tokozirami* (bed louse, Japanese). These and other names were reviewed by Usinger (1966).

Taxonomy

The family Cimicidae is divided into six subfamilies with 23 genera and 91 described species. The family is related to the predaceous family Anthocoridae, which includes species that feed on insects and mites and occasionally bite humans and other warm-blooded vertebrates. A related family, the **Polyctenidae**, includes species that are all ectoparasitic on bats and, like some cimicids, are also commonly called bat bugs.

The Cimicidae includes 12 genera with species associated with bats and nine genera with species associated with birds. In addition, some species in the genus *Cimex* are found on bats and others on birds. Three species are considered ectoparasites of humans. *Leptocimex boueti*, a member of the subfamily Cacodminae, occurs on bats and people in West Africa. The other two are members of the subfamily Cimicinae, the bed bugs. The tropical bed bug, *Cimex hemipterus* (Fig. 8.13), is parasitic on humans and chickens in the Old World and New World tropics and has recently been recorded in Florida; the bed bug, *Cimex lectularius* (Figs. 8.12, 8.14, and 8.15), is a cosmopolitan species associated primarily with humans, bats, and chickens.

Both *Cimex* species that feed on humans originated in the Old World. The origin of *C. hemipterus* is uncertain; however, there is evidence that *C. lectularius* originated in the Middle East, probably being associated with bats and humans living in caves. *Cimex lectularius* apparently spread into Europe during historic times, being recorded from Greece by 400 BC, from Italy by AD 77, and from Germany for the first time in the eleventh century. The bed bug was known in France in the thirteenth century and is recorded as occurring in England in 1583. Therefore, the wide dissemination of *C. lectularius* throughout the world probably did not begin until after the sixteenth century.

Morphology

The most striking feature of cimicids is their dorsoventral flattening. Adults of the oval, mahogany-colored *Cimex* species generally range in length from about 5.5 to 7.0 mm, with abdomens 2.5−3.0 mm wide. The females are larger than the males. The bat bug *Leptocimex boueti* differs from *C. lectularius* and *C. hemipterus* in having a very narrow pronotum, only slightly wider than the head, and very long legs. It is a smaller species, the total body length being 2.8 mm in males and 4.0 mm in females.

FIGURE 8.12 Human bed bug, *Cimex lectularius*; female, left; male, right. *From Usinger, 1966.*

FIGURE 8.13 Tropical bed bug, *Cimex hemipterus*. *Photograph by Brittany Campbell, University of Florida.*

The cimicid head (Fig. 2.2E) is small and cylindrical with two knoblike, multifaceted eyes. Ocelli are not present. The antennae are four-segmented and inserted between the eye and the clypeus. The labium is three-segmented and, as in the triatomines, dorsally encloses the maxillary and mandibular stylets; they, in turn, enclose a relatively large dorsal food canal and a very small ventral salivary canal. The labium has two sensory lobes at its tip. When the bug is not feeding, the **rostrum**, or beak, composed of the labium and associated mouthparts, is bent below the head with the tip extending to the middle of the prosternum.

The thorax consists of a narrow canoe-shaped pronotum, a mesonotum that is covered dorsolaterally by reduced forewings called hemelytral pads, and a metanotum hidden below the latter. Nymphs do not have hemelytral pads. *Cimex hemipterus* can be distinguished from *C. lectularius* by the former's narrower pronotum (Fig. 8.14). The hemelytral pads are oval in *Cimex* species and are reduced to small elevated ridges in *Leptocimex*. Hindwings are never present. The legs are slender, with two-segmented tarsi in the nymphs and two-segmented tarsi in the adults.

The abdomen is 11-segmented and capable of tremendous expansion during blood-feeding. In nymphs, membranous areas on the entire ventral surface and on the first, second, and part of the third abdominal terga enable expansion of the abdomen while feeding. In adults, the intersegmental membranes are wide, and the middle of the ventral surface of the second to fifth segments of the abdomen is likewise membranous. Female *Cimex* adults are readily distinguished from males by the presence of an indentation on the hind margin of the fifth abdominal sternite (Fig. 8.15). This narrow cleft, called the paragenital sinus, is surrounded by bristles and is the point at which the male inserts his aedeagus to intra-abdominally inseminate the female. No paragenital sinus occurs in *Leptocimex boueti*.

Life History

Mating occurs with the male bug straddling the female's back at an oblique angle. In this position the tip of his abdomen is strongly curved against the right side of the venter of the female where the paragenital sinus is located. The male inseminates the female by injecting sperm into the sinus. This form of **traumatic insemination** (= **hypodermic** insemination) that involves introduction of the sperm into an extragenital site occurs in many species of the superfamily Cimicoidea. Specialized structures for reception of the sperm, variously called the **spermalege**, **organ of Ribaga**, and **organ of Berlese**, are present in

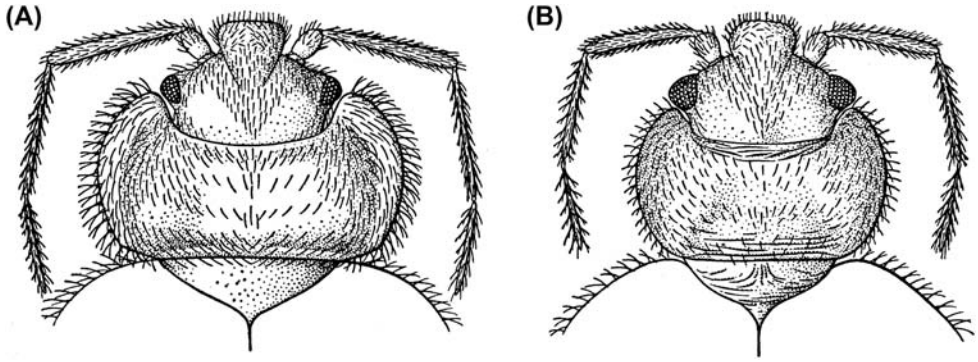


FIGURE 8.14 Head and prothorax of adult bed bugs. (A) *Cimex lectularius*. (B) *C. hemipterus*. *From Smart, 1943; courtesy of the British Museum (Natural History).*

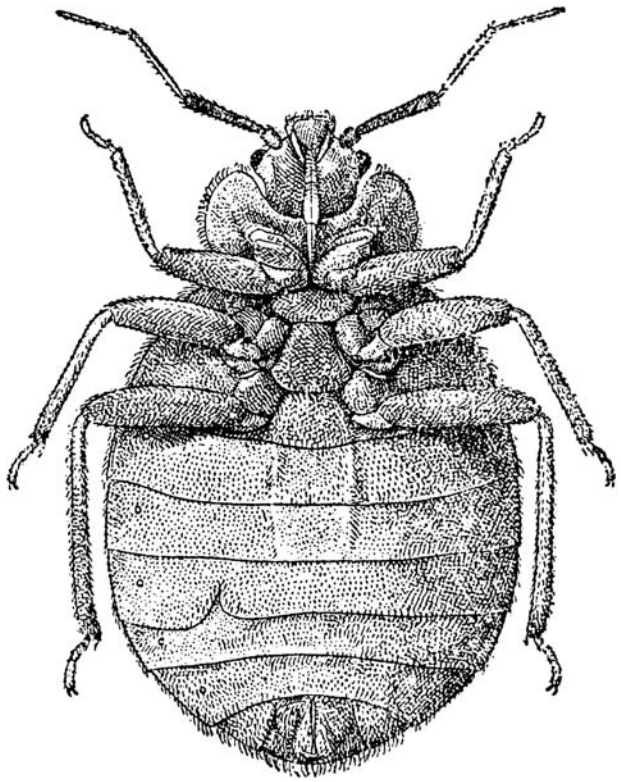


FIGURE 8.15 Human bed bug (*Cimex lectularius*), female, ventral view; the cleft on the hind margin of the fifth abdominal sternite denotes the point of entry (paragenital sinus) for the male aedeagus during insemination. *From Busvine, 1966.*

these species. Copulation usually lasts from one to several minutes but may take up to half an hour. Females that have been inseminated retain permanent scars that are visible in the integument. The sperm pass from the spermalege into the hemocoel, from which they enter paired outpouchings of the walls of the oviducts called sperm conceptacles. From there the sperm travel within the walls of the oviducts via an intraepithelial network of tubular canals called spermodes to the bases of the ovarioles.

Mated females usually feed to repletion and then begin to lay eggs 3–6 days later. Oviposition lasts for about 6 days, during which 6–10 eggs are deposited. Depending on ambient temperature and relative humidity, female bugs may feed every 3–4 days. Eggs are laid continuously, with the mean number of eggs per week typically varying from three to eight. Some females have been observed to lay as many as 12 eggs in one day and up to 540 eggs in their lifetimes. A female is capable of producing viable eggs for 5–7 weeks after feeding and mating. After that time, an increasing number of eggs are sterile.

The eggs are elongate-oval, about 1 mm long, and pearly white. They are laid singly and are coated with a transparent cement that causes them to adhere to various surfaces. The eggs are usually deposited in groups or clusters. Hatching usually takes place in 4–12 days depending on the temperature. There are five nymphal stages, each lasting 2.5–10 days depending largely on bloodmeal availability and ambient temperature. The temperature threshold for development is about 15°C, with optimal development at 30°C. Humidity, except at the extremes, has little or no effect on development. The total developmental time from egg to adult for *C. lectularius* varies from 24 days (at 30°C) to 128 days (at 18°C), and for *C. hemipterus*, from 25 days (at 30°C) to 265 days (at 18°C).

The nymphs are pale straw-colored before they feed but bear a resemblance to red berries after they have fed. Feeding generally occurs within 24 h after hatching or molting. At low temperatures, nymphs may survive for 5–6 months without feeding, whereas adults can survive even longer. This makes them efficient nest parasites that are able to survive long periods when a host is absent. Nymphs feed at least once during each instar. Engorgement usually takes about 3 min for first-instar nymphs and 10–15 min for older nymphs and adults. After fully engorging, nymphs are 2.5–6 times heavier than unfed nymphs, and the adults are 1.5–2 times heavier than unfed individuals. As in triatomines, liquid fecal matter is excreted soon after feeding. Half the weight of the entire bloodmeal is lost within the first 5 h after feeding.

Behavior and Ecology

Cimicids are similar to triatomines in their choices of hiding places, the nature of the substrates selected, and their feeding patterns. They hide in cracks and crevices in human and animal habitations and in nests, caves, and tree holes in natural settings. They prefer rough, dry substrates that allow maximal contact of the bugs with the surface. Their attraction to such harborages between feedings often results in large aggregations. In domestic situations, bed bugs prefer hiding in wood and paper accumulations rather than in materials made of stone, plaster, metal, or textiles. Both *Cimex lectularius* and *C. hemipterus* may infest mattresses, box springs, and upholstered furniture. Other common sites of infestations include upholstered seats in buses and public facilities such as theaters and office waiting rooms. Cimicids will crawl into the narrowest crevices, such as those formed behind loose wallpaper, pictures, or electrical switch or socket plates. The harborages and infested premises are often stained with conspicuous fecal spots that range in color from white to yellow to brown to reddish-brown to black. Areas infested by *C. lectularius* may be identified by a characteristic sweet odor.

Bugs leave their hiding places primarily to feed. They are negatively phototactic, tending to feed mostly in darkness or subdued light when the temperature is above 10°C. Warmth and carbon dioxide, as in many other hematophagous

arthropods, appear to be major factors in attracting bed bugs to a host. A temperature differential of only 1−2°C is sufficient to induce probing (Lehane, 2005).

When the bug has located a host, it approaches with its antennae outstretched and its beak directed downward at a 90-degree angle. It grabs the host with the tarsal claws of the front legs. After contact is made, the antennae are pulled backward, and the entire bug makes rocking, pushing movements as the vertically directed stylets are embedded in the skin. As the stylets penetrate the skin, the labium becomes more and more bent at the skin surface. The mandibles, which have retrorse teeth at their tips, move in and out of the skin in alternating fashion, producing a passage for the maxillae. The bundle of feeding stylets probes actively within the skin until a blood vessel of suitable size is penetrated. Only the maxillae, and possibly only the right maxilla, actually enter the lumen of the vessel.

The salivary secretion contains an anticoagulant that prevents the blood from clotting and nitric oxide, which is an antiplatelet and vasodilatory compound.

After engorging, the bug withdraws its stylets; sometimes this requires considerable effort owing to the teeth on the mandibles. Once the stylets are again encased in the straightened labial sheath, the beak is folded back under the head. The bugs are quick to retreat when disturbed at the beginning or end of feeding; however, while the stylets are fully embedded in the skin, the bug is unable to withdraw its mouthparts even if handled or rotated.

The two species of *Cimex* most commonly associated with humans have been dispersed over wide areas of the globe, with *C. lectularius* most often being found in temperate regions and *C. hemipterus* in the tropics. These species are carried concealed in luggage, furniture, and all manner of packing materials. They have been transported on land vehicles, ships, and planes.

Public Health Importance

Usinger (1966) listed 27 human pathogens, including viruses, bacteria, protozoa, and helminths, that have been shown to survive for varying lengths of time in *C. lectularius* and *C. hemipterus*. However, there is little or no evidence to incriminate bed bugs as vectors of these or any other disease agents.

Recent attempts to explain transmission of Hepatitis B virus and, to a lesser extent, HIV in otherwise unexplained situations have focused on the possibility of cimicid transmission. Hepatitis B antigens (and HBV DNA) persist for several weeks in cimicid tissues and feces under laboratory conditions after the bugs have fed on infected blood. Replication of the virus, however, does not occur. HBV DNA persists from one instar to the next (transstadial transmission) but not transovarially. Past attempts to transmit the virus from infected bugs to chimpanzees failed. These results and those from transmission studies with mosquitoes suggest that it is unlikely that HBV transmission occurs via either infective feces or interrupted feedings; however, definitive studies, attempting transmission with cimicid tissues or feces known to have infectious virus, have not been done. Hepatitis C RNA was not found in cimicid tissues after bugs fed on a viremic patient (Silverman et al., 2001).

Although transmission studies with bed bugs and HIV indicate that these insects may harbor the virus for up to eight days, replication of the virus does not occur and the virus is not present in cimicid feces. These observations, together with failed attempts to experimentally transmit HIV by interrupted feedings, suggest that cimicids are neither biological nor mechanical vectors of HIV.

Despite the fact that cimicids do not play a significant role as vectors of human pathogens, bed bugs are medically important because they cause unpleasant bite reactions and significant blood loss in people living in dwellings that are chronically infested. The actual feeding by bed bugs generally does not produce any pain. If interrupted, a bug will often bite again close to the previous site, thereby creating a linear array of punctures that is characteristic of cimicid bites. People are most often bitten on the limbs, trunk, and face.

Sensitivity reactions to bed bug bites are the result of substances injected during feeding. These reactions may be localized cutaneous responses or generalized and systemic. The most common local reactions are wheals similar to uncomplicated mosquito bites or, in some individuals, large fluid-filled bullae. Erythema is not a common response but may occur as a result of multiple feedings that cause extensive hemorrhaging under the skin. Individual reactions to cimicid bites vary from no response to severe immediate or delayed sensitivity reactions, including **anaphylaxis**. In most cases, swelling and itching associated with the bites can be relieved by the application of ice and use of an oral antihistamine. Chronic bed bug bites are sometimes misdiagnosed as allergic dermatitis or other skin disorders. Accurate clinical assessment often requires careful epidemiological evaluation of a patient's living quarters.

People living with chronic infestations of bed bugs often are subject to nightly attacks, resulting in a marked loss of blood. Children who are marginally nourished are especially vulnerable to developing anemia and other medical problems as a result of such chronic blood loss. Individuals subjected to continued feeding by bed bugs may also develop extreme irritability that results from restless nights and chronic sleep deprivation. If the source of the disturbance goes undetected, the emotional stress caused by such infestations may be misdiagnosed as a neurosis.

A significant increase in reports of bed bug infestations in major U.S. cities (Atlanta, New York, Philadelphia, San Francisco) and in the United Kingdom, Australia, and

Brazil has occurred in recent years. The tropical bed bug, *C. hemipterus,* which had not been seen in Florida in the past 60 years, was identified as the cause of an infestation in a house in Brevard County in 2015. Various explanations for the apparent resurgence of bed bug infestations have been suggested, including increased international travel and transport of materials globally as well as recent changes in pest control methods. Use of specific baits, instead of broad-acting insecticides for control of cockroaches, ants, and other pest species, may have allowed bed bugs to persist and increase. For several years before the current outbreaks, the bed bug was not a major pest in the developed nations. Its absence may have led to a decline in familiarity with the insect by pest control and medical personnel, resulting in incorrect diagnoses and thereby the use of inadequate control measures. Whatever the cause, the economic impact of increased infestations on the tourist industry has been significant, resulting from reduced clientele, increased pest control costs, and litigation expenses incurred by affected patrons.

Other Cimicids That Occasionally Attack Humans

In addition to the three cimicid species directly associated with human habitations, there are several species that occasionally feed on people. These include **swallow bugs** of the genus *Oeciacus*; the **bat bugs** *Cimex pilosellus* and *Cimex adjunctus* (New World) and *C. pipistrelli* (Europe); and the **bird bugs**, such as the **Mexican chicken bug** *Haematosiphon inodorus* and *Cimexopsis nyctalis* from the nests of chimney swifts. Human bites by these species generally occur only in the vicinity of the nesting or roosting sites of their natural hosts.

The swallow bugs that occur in mud nests of swallows include two species: *Oeciacus hirundinis* in Eurasia south to Morocco and *O. vicarius* in North America south to Durango in Mexico. Both species are members of the subfamily Cimicinae and will bite people who disturb infested bird nests. Swallows may be heavily infested with the bugs, and nestlings often die as a result of blood loss. The eggs of *Oeciacus* species are attached to the outer surfaces of swallow mud nests, often being so abundant that they can be seen from a distance. There is some evidence that *Oeciacus* species are carried as nymphs by the birds from nest to nest.

Two arboviruses have been isolated from the cliff swallow bug *O. vicarius* and nestling cliff swallows and house sparrows, one originally from Colorado and one originally from Oklahoma. They are both **alphaviruses**, part of the Western equine encephalitis complex. The one isolated in Colorado (USA) is called Fort Morgan virus, and the one from Oklahoma is called Buggy Creek virus. A third alphavirus, a variant of Buggy Creek virus called Stone Lakes virus, was isolated multiple times from *O. vicarius* from California. The isolates represent the first records of this alphavirus group west of the Continental Divide. The virus was not isolated from cliff swallows and no antibodies to the virus were detected. These viruses from *O. vicarius* appear to be primary infections of the cimicids, with the cliff swallows acting as bloodmeal sources rather than as amplification hosts. The viruses are not known to cause pathology in their avian hosts or in humans. Occurrence of these viruses in bugs suggests that viruses can overwinter in swallow bugs that occupy nests left vacant by their migrating hosts. Isolation of Buggy Creek virus from field-collected eggs of *O. vicarius* supports the idea that the cliff swallow alphaviruses are maintained by vertical transmission in the bugs.

Veterinary Importance

Cimicids can be significant pests in commercial poultry production. Cimicids that attack domestic poultry include *Cimex lectularius* in North America, Europe, and the former Soviet Union; *Haematosiphon inodorus* in Central America; and *Ornithocoris toledoi* in Brazil.

Raised slats and wood shavings in nest boxes in broiler breeder houses provide harborage for the bugs. Indications of cimicid infestations include fecal spots on eggs, nest boxes (Fig. 8.16) and wooden supports, skin lesions on the breasts and legs of birds, reduced egg production, and increased consumption of feed. Chicken bugs are not known to transmit any avian pathogens. However, chickens and other fowl raised in poultry houses heavily infested with chicken bugs are irritable and often anemic. Morbidity in such cases may be high, and young birds may succumb from blood loss. Economic loss on infested poultry farms also may be increased by the reduced productivity of workers allergic to the bugs.

FIGURE 8.16 Fecal spots, indicative of cimicid activity, along seams of a nesting box of laying hens in a poultry house heavily infested with *Cimex lectularius. Photograph by Gary R. Mullen.*

Two species of nonpathogenic trypanosomes that undergo development in cimicids have been isolated from bats in North America. *Trypanosoma hedricki* and *T. myoti*, both closely related to *T. cruzi*, have been found in big brown bats and little brown bats in southern Ontario, Canada. Developmental stages infective to bats form in the rectum of *C. brevis* and *C. lectularius*, which suggests that these trypanosomes are transmitted by the bugs via the posterior-station route. Because bats also are known hosts for *T. cruzi*, the differentiation of other bat trypanosomes and the elucidation of their transmission are important. Furthermore, because of the similarities of the life cycles and transmission of these nonpathogenic trypanosomes to that of *T. cruzi*, they could be suitable candidates for developing laboratory models of the Chagas disease parasite.

Prevention and Control

Measures to prevent cimicid infestations should begin with household sanitation. Removing accumulations of paper and wood trash eliminates hiding places and harborages for the bugs. However, once an infestation occurs, eliminating cimicids generally requires a multipronged chemical and nonchemical approach that includes the use of residual insecticides (sprays and desiccant dusts), inspection, and thorough cleaning or destruction of infested clothing and other materials. Pyrethroids and neonicotinoids formerly provided good control but now are becoming less effective as bugs develop resistance to these insecticides. Inspection (visual and use of bed bug–detecting dogs), monitoring (use of bed bug interceptor devices, such as bed leg traps and CO_2 traps), as well as vacuuming, steaming, and isolation of infested materials (use of mattress covers and encasements) in combination with chemical controls are all effective in eliminating an infestation.

Noninsecticide methods of killing bed bugs include use of thermal devices (to heat or cool infested material to lethal temperatures) and CO_2 fumigation. Nonchemical treatments alone are not sufficient to control infestations but are important adjuncts to chemical control. For temporary control, such as needed by a traveler occupying an infested room for one or a few nights, any of various insecticides applied as aerosols can be used to thoroughly spray bed frames, mattresses, and box springs.

Control of cimicids in premises in which people are bothered by the bites of bird bugs or bat bugs requires identification and removal of the source of the bugs. Such sources include bats roosting in attics or eaves, bird nests on window ledges or air conditioners, and birds roosting in chimneys. Removal of the nonhuman vertebrate hosts must be accompanied by use of an insecticide, or the hungry bugs will seek human blood more aggressively in the absence of their natural hosts.

Various arthropods are natural predators of cimicids. These include the masked bed bug hunter *Reduvius personatus*, other hemipterans, ants, pseudoscorpions, and spiders. None of these, however, has been effectively used for controlling bed bugs.

Doggett et al. (2018) address advances in the biology and management of bed bugs.

REFERENCES AND FURTHER READING

Alexander, J. O. (1984). *Arthropods and human skin*. Berlin: Springer-Verlag.

Asin, S. N., & Catalá, S. S. (1991). Are dead *Triatoma infestans* a competent vector of *Trypanosoma cruzi*? *Memórias do Instituto Oswaldo Cruz, 86*, 301–305.

Axtell, R. C. (1999). Poultry integrated pest management: Status and future. *Integrated Pest Management Reviews, 4*, 53–73.

Beard, C. B., Cordon-Rosales, C., & Durvasula, R. V. (2002). Bacterial symbionts of the Triatominae and their potential use in control of Chagas disease transmission. *Annual Review of Entomology, 47*, 123–141.

Bérenger, J.-M., Almeras, L., Leulmi, H., & Parola, P. (2015). A high-performance vacuum cleaner for bed bug sampling: A useful tool for medical entomology. *Journal of Medical Entomology, 52*, 513–515.

Bern, C., Kjos, S., Yabsley, M. J., & Montgomery, S. P. (2011). *Trypanosoma cruzi* and Chagas disease in the United States. *Clinical Microbiology Reviews, 24*, 655–681.

Blow, J. A., Turell, M. J., Silverman, A. L., & Walker, E. D. (2001). Stercorarial shedding and transtadial transmission of Hepatitis B virus by the common bed bugs (Hemiptera: Cimicidae). *Journal of Medical Entomology, 38*, 694–700.

Bower, S. M., & Woo, P. T. K. (1981). Development of *Trypanosoma* (*Schizotrypanum*) *hedricki* in *Cimex brevis* (Hemiptera: Cimicidae). *Canadian Journal of Zoology, 59*, 546–554.

Brambila, J., & Hodges, G. S. (2004). Bugs (Hemiptera). In J. L. Capinera (Ed.), *Encyclopedia of entomology* (Vol. 1, pp. 354–371). Dordrecht: Springer.

Brault, A. C., Armijos, V., Wheeler, S., Wright, S., Fang, Y., Langevin, S., et al. (2009). Stone Lakes virus (family Togaviridae, genus Alphavirus), a variant of Fort Morgan virus isolated from swallow bugs (Hemiptera: Cimicidae) west of the Continental Divide. *Journal of Medical Entomology, 46*, 1203–1209.

Brown, C. R., Moore, A. T., Young, G. R., Padhi, A., & Komar, N. (2009). Isolation of Buggy Creek virus (Togaviridae: Alphavirus) from field-collected eggs of *Oeciacus vicarius* (Hemiptera: Cimicidae). *Journal of Medical Entomology, 46*, 375–379.

Brumpt, E. (1922). *Précis de Parasitologie*. Paris: Masson & Co.

Busvine, J. R. (1966). *Insects and Hygiene*. London: Methuen & Co. Ltd.

Calisher, C. H., Monath, T. P., Muth, D. J., Lazuick, J. S., Trent, D. W., Francy, D. B., et al. (1980). Characterization of Fort Morgan virus, an alphavirus of the western equine encephalitis virus complex in an unusual ecosystem. *The American Journal of Tropical Medicine and Hygiene, 29*, 1428–1440.

Campbell, B. E., Koehler, P. G., Buss, L. J., & Baldwin, R. W. (2016). Recent documentation of the tropical bed bug (Hemiptera: Cimicidae) in Florida since the common bed bug resurgence. *Florida Entomologist, 99*, 549–551.

Caracavallo, R. U., Jurberg, J., Lent, H., Noireau, F., & Galvão, C. (2000). Phylogeny of the Triatominae (Hemiptera: Reduviidae). Proposals for taxonomic arrangements. *Entomologia y Vectores, 7*(Suppl. 1), 1–99.

Caras, R. (1974). *Venomous animals of the world*. Englewood Cliffs: Prentice-Hall, Inc.

Costa, C. H. N., Costa, M. T., Weber, J. N., Gilks, G. F., Castro, C., & Marsden, P. D. (1981). Skin reactions to bug bites as a result of xenodiagnosis. *Transactions of the Royal Society of Tropical Medicine and Hygiene, 75*, 405–408.

Coura, J. R. (2013). Chagas disease: Control, elimination and eradication. Is it possible? *Memórias do Instituto Oswaldo Cruz, 108*, 962–967.

Doggett, S. L., Geary, M. J., & Russell, R. C. (2004). The resurgence of bed bugs in Australia: With notes on their ecology and control. *Environmental Health, 4*, 30–38.

Doggett, S. L., Miller, D. M., & Lee, C.-Y. (2018). *Advances in the biology and management of modern bed bugs*. Wiley-Blackwell.

Faundez, E. I., & Rojas-Porras, N. A. (2016). First case of a human being bitten by a water boatman (Hemiptera: Heteroptera: Corixidae) from Chile. *Journal of Medical Entomology, 53*, 210–211.

Faundez, E. I. (2016). A case of biting humans by *Nabis americoferus* (Heteroptera: Nabidae) with comments on bites by other species of the genus *Nabis* in the United States. *Journal of Medical Entomology, 53*, 230–232.

Foil, L. D., & Issel, C. J. (1991). Transmission of retroviruses by arthropods. *Annual Review of Entomology, 36*, 355–381.

Grijalva, M. J., Villacis, A. G., Ocaña-Mayorga, S., Yumiseva, C. A., & Baus, E. G. (2011). Limitations of selective deltamethrin application for triatomine control in central coastal Ecuador. *Parasites and Vectors, 4*, 20.

Grijalva, M. J., Terán, D., & Dangles, O. (2014). Dynamics of sylvatic Chagas disease vectors in coastal Ecuador is driven by changes in land cover. *PLoS Neglected Tropical Diseases, 8*, e2960.

Hoare, C. A. (1972). *The trypanosomes of mammals*. Oxford: Blackwell Sci. Publ.

Hopla, C. E., Francy, D. B., Calisher, C. H., & Lazuick, J. S. (1993). Relationship of cliff swallows, ectoparasites, and an alphavirus in west-central Oklahoma. *Journal of Medical Entomology, 30*, 267–272.

Hornok, S., & Kontschán, J. (2017). The western conifer seed bug (Hemiptera: Coreidae) has the potential to bite humans. *Journal of Medical Entomology, 54*, 1073–1075.

Hurwitz, I., Fieck, A., Klein, N., Jose, C., Kang, A., & Durvasula, R. (2012). A paratransgenic strategy for the control of Chagas disease. *Psyche, 202*, 10. https://doi.org/10.1155/2012/178930.

Illinois Department of Public Health. (2017). *Bed bugs*. http://www.dph.illinois.gov/topics-services/environmental-health-protection/structural-pest-control/bed-bugs#resources.

Jupp, P. G., Purcell, R. H., Phillips, J. M., Shapiro, M., & Gerin, J. L. (1991). Attempts to transmit hepatitis B virus to chimpanzees by arthropods. *South African Medical Journal, 79*, 320–322.

Jurberg, J., Galvão, C., Weirauch, C., & Moreira, F. F. F. (2015). Hematophagous bugs (Reduviidae, Triatominae). In A. R. Panizzi, & J. Grazia (Eds.), *True bugs (Heteroptera) of the Neotropics*. Dordrecht: Springer.

Kells, S. A. (2006). Nonchemical control of bed bugs. *American Entomologist, 52*, 109–110.

Kirchhoff, L. V. (2017). *Chagas disease (American trypanosomiasis) workup*. https://emedicine.medscape.com/article/214581-overview.

Kirk, M. L., & Schofield, C. J. (1987). Density-dependent timing of defaecation by *Rhodnius prolixus*, and its implications for the transmission of *Trypanosoma cruzi*. *Transactions of the Royal Society of Tropical Medicine and Hygiene, 81*, 348–349.

Klotz, S. A., Dorn, P. L., Klotz, J. H., Pinnas, J. L., Weirauch, C., Kurtz, J. R., et al. (2009). Feeding behavior of triatomines from the southwestern United States: An update for transmission of Chagas disease. *Acta Tropica, 111*, 114–118.

Klotz, J. H., Dorn, P. L., Logan, J. L., Stevens, L., Pinnas, J. L., Schmidt, J. O., et al. (2010). "Kissing bugs": Potential disease vectors and case of anaphylaxis. *Clinical Infectious Diseases, 50*, 1629–1634.

Lacey, L. A., D'Alessandro, A., & Barreto, M. (1989). Evaluation of a chlorpyrifos-based paint for the control of Triatominae. *Bulletin of the Society for Vector Ecology, 14*, 81–86.

Lehane, M. J. (2005). *Biology of blood-sucking insects* (2nd ed.). Cambridge: Cambridge Univ. Press.

Lent, H., & Wygodzinsky, P. (1979). Revision of the Triatominae (Hemiptera, Reduviidae), and their significance as vectors of Chagas' disease. *Bulletin of the American Museum of Natural History, 163*, 123–520.

Lewinsohn, R. (1979). Carlos Chagas (1879–1934): The discovery of *Trypanosoma cruzi* and of American trypanosomiasis (foot-notes to the history of Chagas's disease). *Transactions of the Royal Society of Tropical Medicine and Hygiene, 73*, 513–523.

Lowenstein, W. A., Romaña, C. A., Ben Fadel, F., Pays, J. F., Veron, M., & Rouzioux, C. (1992). Survie du virus de l'immunodéficience humaine (VIH-1) chez *Triatoma infestans* (Klug 1834). *Bulletin de la Société de Pathologie Exotique, 85*, 310–316.

Marshall, N. A., & Street, D. H. (1982). Allergy to *Triatoma protracta* (Heteroptera: Reduviidae). I. Etiology, antigen preparation, diagnosis and immunotherapy. *Journal of Medical Entomology, 19*, 248–252.

Martinez-Ibarra, J. A., Paredes-González, E., Licón-Trillo, A., Montañez-Valdez, O. D., Rocha-Chávez, G., & Nogueda-Torres, B. (2012). The biology of three Mexican-American species of Triatominae (Hemiptera: Reduviidae): *Triatoma recurva*, *Triatoma protracta* and *Triatoma rubida*. *Memórias do Instituto Oswaldo Cruz, 107*, 659–663.

Maudlin, I., Holmes, P. H., & Miles, M. A. (Eds.). (2004). *The trypanosomiases*. Cambridge: Massachusetts:CABI Publishing.

Messenger, L. A., Garcia, L., Vanhove, M., Huaranca, C., Bustamante, M., Torrico, M., et al. (2015). Ecological host fitting of *Trypanosoma cruzi* Tcl in Bolivia: Mosaic population structure, hybridization and a role for humans in Andean parasite dispersal. *Molecular Ecology, 24*, 2406–2422.

Miles, M. A. (1983). The epidemiology of South American trypanosomiasis—biochemical and immunological approaches and their relevance to control. *Transactions of the Royal Society of Tropical Medicine and Hygiene, 77*, 5–23.

Moore, A. T., Edwards, E. A., Brown, M. B., Komar, N., & Brown, C. R. (2007). Ecological correlates of Buggy Creek virus infection in *Oeciacus vicarius*, southwestern Nebraska, 2004. *Journal of Medical Entomology, 44*, 42–49.

Myers, J. G. (1929). Facultative blood-sucking in phytophagous Hemiptera. *Parasitology, 21*, 472–480.

Olivier, M. C., Olivier, L. J., & Segal, D. B. (Eds.). (1972). *A bibliography on Chagas' disease (1909–1969). Index-catalogue of medical and veterinary zoology special publication No. 2*. Washington: U.S. Govt. Print. Off, 633 p.

Pennington, P. M., Messenger, L. A., Reina, J., Juárez, J. G., Lawrence, G. G., Dotson, E. M., et al. (2015). The Chagas disease domestic transmission cycle in Guatemala: Parasite-vector switches and lack of mitochondrial co-diversification between *Triatoma dimidiata* and *Trypanosoma cruzi* subpopulations suggest non-vectorial parasite dispersal across the Motagua valley. *Acta Tropica, 151*, 80−87.

Peters, W., & Pasvol, G. (2007). *Atlas of Tropical medicine and parasitology* (6th ed.). Elsevier Mosby.

Ramirez, J. D., Guhl, F., Rendón, M. L., Rosas, F., Marin-Neto, J. A., & Morillo, C. A. (2010). Chagas cardiomyopathy manifestations and *Trypanosoma cruzi* genotypes circulating in chronic chagasic patients. PLoS Neglected *Tropical Diseases, 4*(11), e899.

Reinhardt, K., & Siva-Jothy, M. T. (2007). Biology of bed bugs (Cimicidae). *Annual Review of Entomology, 52*, 351−374.

Ribeiro, J. M. C., & Francischetti, I. M. B. (2003). Role of arthropod saliva in blood feeding: Sialome and post-sialome perspectives. *Annual Review of Entomology, 48*, 73−88.

Rohr, A. S., Marshall, N. A., & Saxon, A. (1984). Successful immunotherapy for *Triatoma protracta*-induced anaphylaxis. *Journal of Allergy and Clinical Immunology, 73*, 369−375.

Romero, A., & Anderson, T. D. (2016). High levels of resistance in the common bed bug, *Cimex lectularius* (Hemiptera: Cimicidae), to neonicotinoid insecticides. *Journal of Medical Entomology, 53*, 727−731.

Ryckman, R. E., & Bentley, D. G. (1979). Host reactions to bug bites (Hemiptera, Homoptera): A literature review and annotated bibliography, Part II. *California Vector Views, 26*, 25−49.

Ryckman, R. E., & Zackrison, J. L. (1987). Bibliography to Chagas' disease, the Triatominae and Triatominae-borne trypanosomes of South America (Hemiptera: Reduviidae: Triatominae). *Bulletin of the Society of Vector Ecologists, 12*, 1−464.

Ryckman, R. E., Bentley, D. G., & Archbold, E. F. (1981). The Cimicidae of the Americas and Oceanic Islands, a checklist and bibliography. *Bulletin of the Society of Vector Ecologists, 6*, 93−142.

Ryckman, R. E. (1962). Biosystematics and hosts of the *Triatoma protracta* complex in north America (Hemiptera: Reduviidae) (Rodentia: Cricetidae). *University of California Publications in Entomology, 27*, 93−240.

Ryckman, R. E. (1979). Host reactions to bug bites (Hemiptera, Homoptera): A literature review and annotated bibliography, Part I, Part II (with Bentley, D. G.). *California Vector Views, 26*, 1−49.

Ryckman, R. E. (1985). Dermatological reactions to the bites of four species of Triatominae (Hemiptera: Reduviidae) and *Cimex lectularius* L. (Hemiptera: Cimicidae). *Bulletin of the Society of Vector Ecologists, 10*, 122−125.

Salazar-Schettino, P. M. (1983). Customs which predispose to Chagas' disease and cysticercosis in Mexico. *The American Journal of Tropical Medicine and Hygiene, 32*, 1179−1180.

Schaefer, C. W. (2003). Heteropteran adventitious biters (Hemiptera): Primitively predaceous? *Entomological News, 114*, 211−216.

Schofield, C. J. (1979). The behaviour of Triatominae (Hemiptera: Reduviidae): A review. *Bulletin of Entomological Research, 69*, 363−379.

Schofield, C. J. (1988). Biosystematics of the Triatominae. In M. W. Service (Ed.), *Biosystematics of haematophagous insects* (pp. 285−312). Oxford: Clarendon Press.

Schofield, C. J. (1994). *Triatominae: Biology and control*. West Sussex: Eurocommunica Publications.

Schofield, C. J., & Dias, J. C. (1999). The southern cone initiative against Chagas disease. *Advances in Parasitology, 42*, 1−27.

Schofield, C. J., & Dolling, W. R. (1993). Bedbugs and kissing bugs. In R. P. Lane, & R. W. Crosskey (Eds.), *Medical insects and arachnids* (pp. 483−516). London: Chapman & Hall.

Schofield, C. J., & Dujardin, J.-P. (1997). Chagas disease vector control in Central America. *Parasitology Today, 13*, 141−144.

Schofield, C. J., & White, G. B. (1984). Engineering against insect-borne diseases in the domestic environment/House design and domestic vectors of disease. *Transactions of the Royal Society of Tropical Medicine and Hygiene, 78*, 285−292.

Schofield, C. J., Minter, D. M., & Tonn, R. J. (1987). XIV. The triatomine bugs−biology and control. In *Vector control series−triatomine bugs−training and information guide*. World Health Organization Vector Biology and Control Division 87.941.

Shuh, R. T., & Slater, J. A. (1995). *True bugs of the world (Hemiptera: Heteroptera) classification and natural history*. Ithaca: Cornell University Press.

Silverman, A. L., Qu, L. H., Blow, J., Zitron, I. M., Gordon, S. C., & Walker, E. D. (2001). Assessment of hepatitis B virus DNA and hepatitis C virus RNA in the common bedbug (*Cimex lectularius* L.) and kissing bug (*Rhodnius prolixus*). *American Journal of Gastroenterology, 96*, 2194−2198.

Smart, J. (1943). *A handbook for the identification of insects of medical importance*. London: British Museum (Natural History).

Stevens, L., Rizzo, D. M., Lucero, D. E., & Pizarro, J. C. (2013). Household model of Chagas disease vectors (Hemiptera: Reduviidae) considering domestic, peridomestic, and sylvatic vector populations. *Journal of Medical Entomology, 50*, 907−915.

Stevens, L., Monroy, M. C., Rodas, A. G., Hicks, R. M., Lucero, D. E., Lyons, L. A., et al. (2015). Migration and gene flow among domestic populations of the Chagas insect vector *Triatoma dimidiata* (Heteroptera: Reduviidae) detected by microsatellite loci. *Journal of Medical Entomology, 52*, 419−428.

Ter Poorten, M. C., & Prose, N. S. (2005). The return of the common bedbug. *Pediatric Dermatology, 22*, 183−187.

Trumper, E. V., & Gorla, D. E. (1991). Density-dependent timing of defecation by *Triatoma infestans*. *Transactions of the Royal Society of Tropical Medicine and Hygiene, 85*, 800−802.

Usinger, R. L. (1934). Blood sucking among phytophagous Hemiptera. *The Canadian Entomologist, 66*, 97−100.

Usinger, R. L. (1944). *The Triatominae of North and Central America and the West Indies and their public health significance. Public Health Bulletin 288*. Washington: U.S. Govt. Print. Off.

Usinger, R. L. (1966). *Monograph of Cimicidae (Hemiptera-Heteroptera). Thomas Say Foundation* (Vol. VII). Entomological Society of America.

Wang, C., Gibb, T., & Bennett, G. W. (2009). Evaluation of two least toxic integrated pest management programs for managing bed bugs (Heteroptera: Cimicidae) with discussion of a bed bug intercepting device. *Journal of Medical Entomology, 46*, 566−571.

Wang, C., Lü, L., & Xu, M. (2012). Carbon dioxide fumigation for controlling bed bugs. *Journal of Medical Entomology, 49*, 1076−1083.

Wang, C., Singh, N., Zha, C., & Cooper, R. (2016). Bed bugs: Prevalence in low-income communities, resident's reactions, and implementation of a low-cost inspections protocol. *Journal of Medical Entomology, 53*, 639–646.

Welch, K. A. (1990). First distributional records of *Cimexopsis nyctalis* List (Hemiptera: Cimicidae) in Connecticut. *Proceedings of the Entomological Society of Washington, 92*, 811.

Woo, P. T. K. (1991). Mammalian trypanosomiasis and piscine cryptobiosis in Canada and the United States. *Bulletin of the Society for Vector Ecology, 16*, 25–42.

Wood, S. F. (1951). Importance of feeding and defecation times of insect vectors in transmission of Chagas' disease. *Journal of Economic Entomology, 44*, 52–54.

Yamagata, Y., & Nakagawa, J. (2006). Control of Chagas disease. *Advances in Parasitology, 61*, 129–165.

Yoon, K. S., Kwon, D. H., Strycharz, C. S., Hollingsworth, C. S., Lee, S. H., & Clark, J. M. (2008). Biochemical and molecular analysis of deltamethrin resistance in the common bed bug (Hemiptera: Cimicidae). *Journal of Medical Entomology, 45*, 1092–1101.

Zapata, M. T. G., Schofield, C. J., & Marsden, P. D. (1985). A simple method to detect the presence of live triatomine bugs in houses sprayed with residual insecticides. *Transactions of the Royal Society of Tropical Medicine and Hygiene, 79*, 558–559.

Zeledón, R., & Vargas, L. G. (1984). The role of dirt floors and of firewood in rural dwellings in the epidemiology of Chagas' disease in Costa Rica. *The American Journal of Tropical Medicine and Hygiene, 33*, 232–235.

Zeledón, R., Alvarado, R., & Jirón, L. F. (1977). Observations on the feeding and defecation patterns of three triatomine species (Hemiptera: Reduviidae). *Acta Tropica, 34*, 65–77.

This page intentionally left blank

Chapter 9

Beetles (Coleoptera)

William L. Krinsky
Division of Entomology, Peabody Museum of Natural History, Yale University, New Haven, CT, United States

Beetles constitute the largest order of insects but are of relatively minor public health or veterinary importance. Adults and larvae of a few species occasionally bite, but, of greater significance, some species secrete chemicals that can irritate the skin and eyes of humans and other animals. Beetles found in stored products can cause inhalational allergies, and some species found in dung and stored products act as intermediate hosts for helminths that cause pathology in domestic and wild animals. Many dung-inhabiting beetles are beneficial in interrupting the life cycles of mammalian parasitic worms and as predators or parasitoids of pestiferous flies that breed in excrement. A few beetle species are ectoparasites or mutualistic symbionts on mammals, and a few are known to temporarily invade the skin of mammals.

TAXONOMY

The order Coleoptera is divided into four suborders: **Archostemata**, considered the most primitive; **Adephaga**, named for its carnivorous members; **Myxophaga**, which are algae-eaters; and **Polyphaga**, the largest suborder, encompassing 90% of beetle families, composed of species with diverse feeding habits. Beetles are currently grouped in about 165 families (Crowson, 1981; Arnett and Thomas, 2001). About 130 families include species that occur in North America.

More than 380,000 species of beetles have been described, representing about 40% of all known insects. More than 30,000 species of beetles occur in the United States and Canada (White, 1983; Arnett et al., 2002). Fewer than 100 species worldwide are known to be of public health or veterinary importance; most of these are in the suborder Polyphaga. The species that have the greatest impact on the health of humans and domestic animals are in the following families: Meloidae (blister beetles), Oedemeridae (false blister beetles), Staphylinidae (rove beetles), Tenebrionidae (darkling beetles), Dermestidae (larder beetles), and Scarabaeidae (scarab or dung beetles).

MORPHOLOGY

Adult beetles are distinguished from all other insects by the presence of hardened forewings called **elytra** (sing., elytron) that cover and protect the membranous hindwings (Fig. 9.1). Coleoptera means "sheath-winged" in Greek. The size range of beetles is impressive, varying from 0.4 to 167 mm; however, most species are 2–20 mm long. Black and brown are the most common colors seen in the Coleoptera, but exquisite bright colors, including metallic and iridescent hues, occur especially in tiger beetles, ground beetles, plant beetles, flower beetles, metallic wood-boring beetles, long-horned beetles, and lady beetles. Beetles vary in shape from elongate, flattened, or cylindrical to oval or round. Their bodies are often hardened, like the elytra, but some families, such as the blister and false blister beetles, have soft elytra and soft body parts that are pliable and sometimes described as leather-like.

The head of a beetle is usually conspicuous, and almost all beetles have some form of biting or chewing mouthparts. Even in specialized species adapted for piercing and sucking plants, the mandibles are retained and are functional. The antennae vary greatly in shape from filiform to pectinate to clavate or clubbed and are usually composed of 11 visible segments. Two compound eyes are present in most species, and ocelli are rarely present.

Part of the thorax is visible dorsally as the pronotum, just posterior to the head (Fig. 9.1). The divisions of the thorax are usually evident only on the ventral surface. The legs vary greatly in shape from thick paddles in swimming species to slender, flexible forms in running species. The paired elytra cover the folded, membranous pair of hindwings. They usually overlay the dorsum of the abdomen and often are all that are visible in the abdominal

Medical and Veterinary Entomology. https://doi.org/10.1016/B978-0-12-814043-7.00009-1

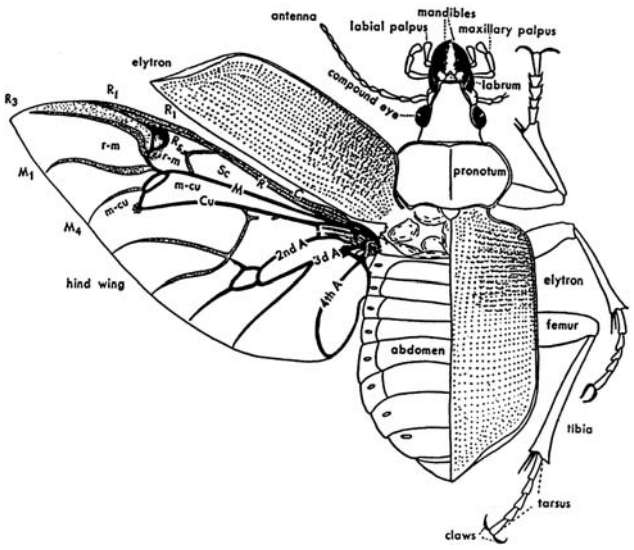


FIGURE 9.1 A representative adult beetle (Carabidae), dorsal view, with left elytron and wing spread. A, anal vein (second, third, fourth); C, costa; M, media (M_1 = first branch, etc.); R, radius (R_1, first branch, etc.); R_s, radial sector; Sc, subcosta; m-cu, mediocubital cross-vein; r-m, radiomedial cross-vein. *Modified from Essig (1942).*

region, when a beetle is viewed from above. In most species, the elytra are raised during flight. Some beetles have no hindwings and are flightless, and some beetles have very short elytra so that the abdominal tergites are visible dorsally (e.g., rove beetles and some blister beetles). Most beetles have eight visible abdominal tergites that can be seen when the elytra and hindwings are raised.

Defensive glands that secrete substances to repel predators are best developed in beetles in the suborder Adephaga. They are generally present as pygidial glands that open dorsally near the end of the abdomen. Secretions from these glands in the Adephaga are not known to cause notable ill effects in mammals. Within the Polyphaga, pygidial glands occur in a few families, such as the Tenebrionidae. In tenebrionids, the pygidial glands produce secretions that can deter small mammals and cause human skin irritation. The pygidial secretions of most other polyphagan species are not known to affect vertebrates.

Beetles that contain chemicals that are especially irritating to humans and other animals have toxic substances dispersed throughout their bodies rather than sequestered in specialized glands. The blister beetles, paederine rove beetles, and lady beetles fall within this group.

LIFE HISTORY

All beetles exhibit holometabolous development. Eggs are laid singly or in clusters on or in soil, living or dead plant matter, fabrics, water, carrion, and, rarely, living animals. The larvae of most beetles have a distinct head with simple eyes (ocelli) and chewing, mandibulate mouthparts, and the abdomen has 8–10 segments. Beetle larvae exhibit diverse morphological types, from elongate-flattened forms (**campodeiform**), to cylindrical-flattened forms (**elateriform**), caterpillar-like forms (**eruciform**), and somewhat C-shaped soft forms (**scarabaeiform**). The larval body type usually is consistent in a particular family of beetles. In a few families, however, the larval form may vary from instar to instar in a given species, a life history progression called hypermetamorphosis. Certain blister beetle larvae, including scavengers in bee nests and ectoparasites or endoparasites of other insects, are hypermetamorphic. They emerge from the eggs as active campodeiform larvae, then molt into eruciform and scarabaeiform stages. Most beetle larvae molt at least three times before transforming into pupae.

Although most temperate species undergo only one generation a year, species in warmer climates are often multivoltine. Depending on the species, any developmental stage may overwinter, but overwintering most often occurs in the pupal or adult stage. Most species exhibit diapause in one or another stage, and those that have developmental cycles exceeding one warm season usually have an obligatory diapause, initiated by changes in photoperiod and temperature. Most adult beetles live for weeks to as long as a year. However, adults of some species may live for years, spending much of their lives in diapause during periods when food is scarce.

BEHAVIOR AND ECOLOGY

Beetles live in all terrestrial and freshwater habitats. The great variation in beetle feeding behavior, whether saprophagous, herbivorous, carnivorous, or omnivorous, reflects the extremely diverse habitats in which these insects live. Their biting mouthparts play a minor role in causing discomfort to humans and other animals. Beetle defense mechanisms, which involve the shedding or secretion of physically or chemically irritating materials, and beetle behavior that puts the insects in contact with developmental stages of parasitic helminths and vertebrates can lead to public health and veterinary problems.

Larder or pantry beetles (Dermestidae) are ubiquitous in human and domestic animal environments, where the larval and adult beetles eat stored food, food debris, dead insects, and other organic matter. Setae that cause human skin irritation or act as respiratory allergens are loosely affixed to the larvae of many species. The setae are elaborately barbed so that their firm adherence to many substrates, including human skin, causes them to be dislodged from the crawling, living larvae. In some species, the larvae actively raise the abdomen and make striking movements in response to touch. Other active defensive behaviors are seen in blister beetles, some chrysomelid plant beetles, long-horned beetles, and lady beetles that exude irritant

chemicals from the femorotibial joints of the legs or from glandular openings around the mouthparts when the beetles are handled or threatened. This reflex bleeding repels predators. One of the most dramatic defensive maneuvers is the explosion of boiling hot, acrid quinones from the anal glands of carabid beetles called bombardier beetles. These forceful expulsions, which are aimed with extreme accuracy at potential predators, cause minimal damage to humans and other large animals but can cause physical and chemical burns in insects and small vertebrates (Eisner et al., 2005).

Most of the beetles that serve as intermediate hosts for helminths parasitic in domestic animals and humans are grain or dung feeders. These species ingest helminth eggs present in animal feces or feces-contaminated food. Because of the proximity of the beetles to feeding animals, whole adult beetles are often incidentally ingested by potential vertebrate hosts.

The tendency of many beetles to fly to artificial lights puts them in contact with human and domestic animal habitats and increases the chances of vertebrate contact with species that may be the sources of skin irritations, allergies, or helminthic infestations.

PUBLIC HEALTH IMPORTANCE

Human health problems caused by beetles include skin, eye, ear, and nose irritations; respiratory allergies; and minor gastrointestinal discomfort. Beetle families known to cause public health problems are listed in Table 9.1.

The greatest human discomfort associated with beetles is caused by vesicating species that secrete irritating chemicals when the insects are handled or accidentally contact human skin or sense organs. Blister beetles, false blister beetles, some rove beetles, and some darkling beetles have these irritants in their secretions, hemolymph, or body parts. Larvae of larder beetles are covered with hairs that can act as skin or respiratory allergens.

Invasion of body tissues by beetle larvae is called **cantheriasis**, whereas invasion of such tissues by adult beetles is called **scarabiasis**. These forms of infestation occur most often in tropical regions. Most clinical cases involve **enteric cantheriasis** that results from the ingestion of foodstuffs infested with beetles or the accidental ingestion of infested materials by children. Dermestid larvae, such as those of *Trogoderma glabrum* and *T. ornatum*, have been associated with enteric cantheriasis in infants who showed signs of extreme digestive discomfort, which in one case was the result of ulcerative colitis. It is unlikely that larvae were the cause of the latter condition, although larval hairs may have exacerbated the symptoms. Larvae were recovered from the stools of these patients and from the dry cereal they ingested. Multiple larvae of the cigarette beetle (Ptinidae: Anobiinae: *Lasioderma serricorne*) were recovered over the course of 3 days from the stools of a febrile 1-year-old girl. Other than fever, the child showed no signs of illness. The source of the larvae, which may have come from beetle eggs in cereal or dog food, was not found. Other grain-infesting beetles, such as *Tenebrio molitor* and *T. obscurus*, have been accidentally ingested without causing noticeable symptoms.

Rarely, adult and larval beetles have been recovered from human nasal sinuses and larvae from the urethra. Small beetles in various families have been known to fly or crawl into human eyes and ears. Some of these cause minor physical irritation, while others may cause extreme burning sensations, presumably due to chemicals exuded by the insects.

Painful, but temporary, eye lesions caused by tiny *Orthoperus* species (<1 mm long) in the family Corylophidae have been seen in eastern Australia, where the condition has been reported under several names: **Canberra eye**, **Christmas eye,** and **harvester's keratitis**.

Tingling and numbing of the lips and face, and burning of the eyes and skin, are caused by contact with beetles of the family Melyridae (genus *Choresine*) in New Guinea. Melyrid beetles also appear to be the source of batrachotoxins found in toxic passerine birds in New Guinea and possibly of similar toxins found in South American poison-dart frogs. The name *nanisani* is used to describe one of the New Guinea birds with toxic feathers, as well as the facial sensations arising from contact with the bird or the melyrid beetles (Dumbacher et al., 2004).

An unusual apparent chemical defense mechanism has been described in a South American long-horned beetle. The cerambycid *Onychocerus albitarsis* has the last segment of each antenna modified into a pointed sting, which is somewhat analogous to the last segment of the tail of a scorpion in structure and causes pain and swelling when jabbed into the skin (Berkov et al., 2008).

More than 40 species of beetles have been associated with human allergic reactions that result from inhaling beetle parts (e.g., larval setae) or excreta (Bellas, 1989). Agricultural and research workers are most often affected by inhalational allergies, because most of the beetle species involved occur in large numbers in stored products. Dermestid beetles (*Trogoderma angustum*), tenebrionids (*Tenebrio molitor* and *Tribolium* spp.), and grain weevils (*Sitophilus granarius*) have been incriminated in many cases of respiratory distress, such as asthma.

Beetles serve as intermediate hosts for more than 50 parasitic worms, including tapeworms (Cestoda), flukes (Trematoda), roundworms (Nematoda), and thorny-headed worms (Acanthocephala) (Hall, 1929; Cheng, 1973). These worms primarily parasitize nonhuman hosts. Only a few species, such as the rodent tapeworms *Hymenolepis*

TABLE 9.1 Beetle Families of Medical-Veterinary Importance, Listed in Order of Relative Importance

Family	Common Names	Clinical Importance
Meloidae	Blister beetles	Cause eye irritation and blisters on skin; can poison and kill horses and birds that ingest them
Staphylinidae	Rove beetles	Paederine species cause skin and eye lesions, and can poison livestock that ingest them; large species are known to bite humans; species attracted to dung feed on fly eggs, larvae, and pupae and are thereby beneficial in reducing pestiferous fly populations
Scarabaeidae	Dung beetles and chafers	Spines cause irritation when adults enter ears; intermediate hosts of helminths; dung feeders are potential disseminators of pathogens; some dung feeders are beneficial in removing dung that is the source of pestiferous flies and that is infested with intermediate stages of vertebrate worm parasites
Tenebrionidae	Darkling beetles and grain beetles	Cause skin and eye irritation; larvae and adults contain inhalational allergens; grain-feeding species are intermediate hosts of helminths and potential disseminators of pathogens
Dermestidae	Larder beetles, pantry beetles, hide beetles, carpet beetles	Larval setae can cause skin, eye, ear, and nose irritation or gastrointestinal discomfort if ingested; larvae and adults can cause inhalational allergies; grain-feeding species are intermediate hosts of helminths; carrion-feeding species are potential disseminators of pathogens
Histeridae	Hister beetles	Beneficial as predators of fly eggs and larvae developing in avian and mammalian manure
Oedemeridae	False blister beetles	Cause skin and eye irritation
Carabidae	Ground beetles	Intermediate hosts of poultry tapeworms
Silphidae	Burying beetles or carrion beetles	Potential disseminators of pathogens
Corylophidae	Minute fungus beetles	Cause eye lesions
Melyridae	Soft-winged flower beetles	Cause skin tingling, and numbing and burning of the skin and eyes
Coccinellidae	Ladybird beetles or ladybugs	Secretions can cause skin discoloration and irritation
Cleridae	Checkered beetles	Can bite humans, causing temporary distress
Cerambycidae	Long-horned beetles	Larger species can bite humans and other animals, causing temporary discomfort
Merycidae	Old World cylindrical bark beetles	Can bite humans, causing temporary distress
Curculionidae	Weevils	Grain-inhabiting species can cause inhalational allergies

nana and *H. diminuta*, and the *Macracanthorhynchus* species of acanthocephalan parasites, occasionally infest children. The intermediate hosts of *Hymenolepis* species are grain beetles (Tenebrionidae), and *Macracanthorhynchus* species undergo development in dung beetles (Scarabaeidae). Children become infested because of their poor hygienic practices or via accidental ingestion of the beetles. Intentional ingestion of living tenebrionids for medicinal purposes in Malaysia is also a potential route for human infestation with rodent tapeworms (Chu et al., 1977).

Many beetles, such as scarabs, silphids, and dermestids, that feed on dung and carrion have the potential to be mechanical vectors of pathogens, such as the bacilli that can cause salmonellosis and anthrax. Although there is experimental evidence for maintenance and excretion of some of these microbes by beetles, given the limited sizes of the inocula and the limited contact between humans and scavenger beetles, there is no indication that these beetles play a role in direct transmission to humans.

Many families of beetles include species known to occasionally bite humans. This may happen when the beetles are accidentally handled or when the beetles occur in such large numbers that many fly or crawl onto the body. Entomologists and others who pick up beetles are the persons most often bitten. Long-horned beetles (Cerambycidae), checkered beetles (Cleridae), rove beetles (Staphylinidae), and ladybird beetles (= ladybugs) (Coccinellidae) are

among those that have been reported as biting. The somewhat painful bites usually leave little or no skin marks and do not cause any long-lasting discomfort. Long-horned beetles feed on wood in their immature stages and are found as adults on flowers, dead and dying trees, and freshly cut timber. The larger species of rove beetles that can bite are predaceous on fly larvae and are often found on carrion or dung. Checkered beetles are found under bark associated with wood−boring insects or fungus.

Population increases and mass migrations of checkered beetles (Cleridae), flat grain beetles (Silvanidae), and ground beetles (Carabidae) have all caused annoyance at times by their sheer numbers and, in some cases, by the strong odors of their defensive secretions.

Meloidae (Blister Beetles)

Blister beetles (Fig. 9.2 and see Fig. 2.4A) occur worldwide. Most, if not all, contain the terpene **cantharidin** ($C_{10}H_{12}O_4$), which can cause skin irritations. People usually develop blisters within 24 h of contacting the secretions of these beetles or the body fluids from crushed beetles (Fig. 9.3). Often this is accompanied by tingling or burning sensations. The blisters may progress to vesicular dermatitis with itching and oozing lesions. There are about 3,000 species of meloids, and at least 20 species are known to cause blistering (Table 9.2). Cantharidin is present in the hemolymph and in the clear, yellow secretion that is exuded at the joints of the legs of these beetles by reflex bleeding. Reptiles and some predaceous insects are repelled by the fluid. Although cantharidin is irritating to humans, the chemical acts as a meloid courtship stimulant that is secreted by male accessory glands and passed to the female during copulation. The males, being the only source of cantharidin, generally have the highest concentrations of the chemical, with levels in female beetles varying with their mating histories. The meloid spermatophore is rich in cantharidin, and the eggs also contain the substance, presumably to deter predators.

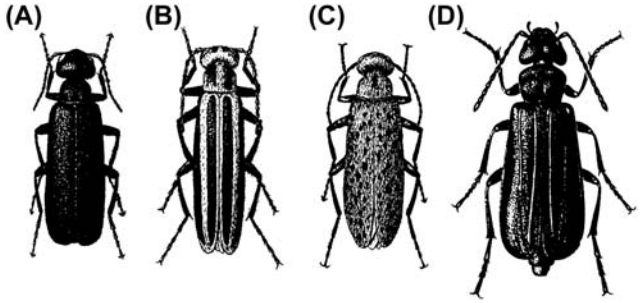


FIGURE 9.2 Blister beetles (Meloidae). (A) Black blister beetle (*Epicauta pennsylvanica*). (B) Striped blister beetle (*E. vittata*). (C) Spotted blister beetle (*E. maculata*). (D) European "Spanish fly" (*Lytta vesicatoria*). *(A−C) Modified from White (1983); (D) from Harde (1984).*

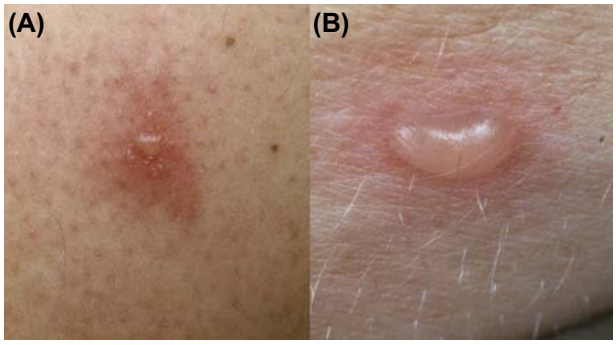


FIGURE 9.3 Contact dermatitis caused by blister beetles (Meloidae, *Epicauta* spp.). *Photographs by Gary R. Mullen (A) and Jerry F. Butler (B).*

Blister-beetle dermatitis has been reported in Europe, Asia, Africa, North America, and Central America. The most famous blister beetle is the "**Spanish fly**" *Lytta vesicatoria* of the Mediterranean region, an insect that has been erroneously touted as a human aphrodisiac. Cantharidin is poisonous to humans and other animals when ingested and may cause kidney damage and death. Ingestion of powder made by grinding up dried beetles or any other source of cantharidin produces extremely toxic effects on the urogenital system. The resulting inflammation causes painful urination, hematuria, and persistent penile erection (priapism), a condition mistakenly associated with increased sexual stimulation. Like many other naturally occurring toxins, cantharidin has been prescribed for centuries as a cure for various ailments, but it has never been proved to have a therapeutic effect.

Meloid species most often associated with skin lesions in the United States and Mexico are members of the genus *Epicauta*. These include the striped blister beetle (*Epicauta vittata*) in the Eastern states, the black blister beetle (*E. pennsylvanica*) found throughout most of the country, and the spotted blister beetle (*E. maculata*) found in the Western states (Fig. 9.2). Other species of *Epicauta* cause similar problems in India and Africa (Table 9.2). Members of the genus *Cylindrothorax* occur over a vast area of the Old World, including Africa, the Near East, India, and parts of Southeast Asia. African species that cause blistering include *C. bisignatus*, *C. dusalti*, *C. melanocephalus*, and *C. picticollis*. A *Lytta* species in China also has been associated with human dermatitis (Table 9.2).

Blister beetles are found often on flowers or foliage where the beetles feed on pollen and other plant tissues and where they are near the nests of solitary bees that serve as hosts for the larvae of many species. *Epicauta* species are usually abundant where grasshoppers flourish because the larvae of these meloids feed on grasshopper eggs. Most people who develop blister beetle lesions are agricultural workers or soldiers on maneuvers in areas where the beetles are common. Retention of cantharidin in frogs and birds

TABLE 9.2 Species of Meloidae Reported to Cause Blistering of Human Skin

Species	Geographic Occurrence of Clinical Reports
Lytta vesicatoria	Europe
L. phalerata	China
Epicauta cinerea	United States (southwestern)
E. flavicornis	Senegal
E. hirticornis	India
E. maculata	United States (western)
E. pennsylvanica	United States, Mexico
E. sapphirina	Sudan
E. tomentosa	Sudan
E. vestita	Senegal
E. vittata	United States (eastern)
Cylindrothorax bisignatus	South Africa
C. dusalti	Senegal, Mali
C. melanocephalus	Gambia, Senegal
C. picticollis	Sudan
C. ruficollis	Sudan, India
Mylabris bifasciata	Nigeria
M. cichorii	India
Psalydolytta fusca	Gambia
P. substrigata	Gambia

Modified from Alexander (1984), with information from Selander (1988).

that prey on meloids may lead to human poisoning when these predators are used as human food. Nineteenth-century medical reports of priapism in French legionnaires traced the cause of this clinical problem to the soldiers' ingestion of frogs that had eaten meloids. Humans have also developed signs of cantharidin poisoning following the ingestion of cooked wild geese (Eisner et al., 1990).

Oedemeridae (False Blister Beetles)

False blister beetles in the genera *Oxycopis* (Fig. 9.4), *Oxacis*, and *Alloxacis* are known to cause vesicular or bullous dermatitis in the United States, Central America, and Caribbean region. *Sessinia kanak*, a species that is commonly attracted to lights in the Solomon Islands, causes similar irritating lesions. Blistering has been observed in people exposed to large numbers of swarming *Eobia apicifusca* in Australia. False blister beetles are attracted to flowers where they feed on pollen. Immediate burning of the skin following contact with *Sessinia* sp. swarming around coconut flowers has been reported on the Line Islands, south of Hawaii. Both *Thelyphassa apicata* in Hawaii and *T. lineata* in New Zealand also have been reported to cause blistering when they contact skin. As in meloids, **cantharidin** is the toxic substance in all of these oedemerids.

Staphylinidae (Rove Beetles)

Rove beetles in the genus *Paederus* contain **pederin** ($C_{25}H_{45}O_9N$), a toxin more potent than that of *Latrodectus* spider venom and the most complex nonproteinaceous insect defensive secretion known. Pederin is synthesized by endosymbiotic gram-negative bacteria (*Pseudomonas* species) occurring in female *Paederus* spp. The beetles, which are mostly 7−13 mm long, are found in North, Central, and South America; Europe; Africa; Asia; and Australasia. Unlike most rove beetles that are dull-colored, many *Paederus* sp. have an orange pronotum and orange basal segments of the abdomen, which contrast sharply with the often blue or green metallic elytra and brown or black coloration of the rest of the body (Fig. 9.5). This color

FIGURE 9.4 False blister beetle, *Oxycopis mcdonaldi* (Oedemeridae), North America. *Photograph by David E. Reed.*

FIGURE 9.5 Rove beetle, *Paederus riparius* (Staphylinidae), Russia. *From Bouchard (2014).*

pattern may be a form of warning (aposematic) coloration, but a defensive function for pederin has not been demonstrated.

At least 20 of the more than 600 described species of *Paederus* have been associated with *Paederus* dermatitis (Table 9.3). Skin reactions to the beetles, called *Ch'ing yao ch'ung*, were described in China as early as AD 739. Most cases of dermatitis have involved tropical species, including *Paederus fuscipes* (widespread from the British Isles east across Central Asia to Japan and southeast to Australia), *P. sabaeus* (Africa, where it is called **Nairobi fly** and **champion fly**), *P. cruenticollis* and *P. australis* (Australasia), *P. signaticornis* (Central America), and *P. columbinus* and *P. brasiliensis* (South America). Species in South American countries are known by various names, such as *bicho de fuego, pito, potó, podó*, and *trepa-moleque*.

Unlike blister beetles, rove beetles do not exhibit reflex bleeding as a defensive reaction. Pederin contacts human skin only when a beetle is brushed vigorously over the skin or crushed. Because of their general appearance or misunderstandings about their etiology, the resulting skin lesions have been called **dermatitis linearis**, **spider-lick** (India and Sri Lanka), and **whiplash dermatitis**. The dermatitis may develop on any part of the body; however, exposed areas such as the head, arms, hands, and legs are most often affected. Mirror-image lesions may form where one pederin-contaminated skin surface touches another.

Unlike meloid-induced dermatitis that develops within 18–24 h after contact, the pederine-induced reaction of itching and burning usually occurs 24–72 h after contact with the beetle's body fluid. The affected skin appears reddened and vesicles form about 24 h after the initial response (Fig. 9.6). The vesicles may coalesce into blisters and become purulent, producing a reaction that is often more severe than that seen after exposure to meloids. The itching may last for a week, after which the blisters crust over, dry, and peel off, leaving red marks or lightened skin areas that may persist for months. Rubbing the eyes with beetle fluid or contaminated hands or beetles flying or crawling into eyes can cause pain, marked swelling of the eyelids and conjunctivae, excessive lacrimation, clouding of the cornea, and inflammation of the iris (iritis). Such ocular lesions seen in East Africa have been called **Nairobi eye**. Although eye involvement often is very irritating, permanent damage is not common.

Rove beetles live in vegetable debris and under stones and other materials, such as leaf litter. They are predaceous on insects and other arthropods or may eat plant debris. Paederine staphylinids are most abundant in areas of moist soil, such as irrigated fields and other crop lands, where the adult beetles feed on various herbivorous insects. Consequently, agricultural workers and others working in fields and grassy areas are often affected. Because the beetles are attracted to lights, workers on brightly lit oil rigs and people occupying lighted dwellings in tropical areas are also commonly affected with what has been called **night burn**.

Tenebrionidae (Darkling Beetles)

Darkling beetles (Fig. 9.7) produce defensive secretions containing **quinones**. Adults of *Blaps* sp. found in the Middle East and Europe secrete these chemicals that cause burning, blistering, and darkening of the skin. Adult beetles of some cosmopolitan *Tribolium* sp., including *T. confusum* and *T. castaneum*, have been associated with severe itching. North American desert species in the genus *Eleodes*, when threatened, take a characteristic headstand pose and exude various quinones that repel small predators and cause mild irritation to humans who handle these beetles. Darkling beetles are found in diverse habitats, including under logs and stones, in rotting wood and other vegetation, in fungi, in termite and ant nests, and among debris in and outside of homes. Most species live in dry, often desert, environments, while pest species are found in stored products, such as grain and cereals. Most tenebrionids are scavengers on decaying or dry plant material, but a few feed on living plants.

TABLE 9.3 Species of *Paederus* (Staphylinidae) Reported to Cause Skin Lesions in Humans

Species	Geographic Occurrence of Clinical Reports
Paederus alternans	India, Vietnam, Laos
P. amazonicus	Brazil
P. australis	Australia
P. brasiliensis	Brazil, Argentina
P. columbinus	Brazil, Venezuela
P. cruenticollis	Australia
P. eximius	Kenya
P. ferus	Argentina
P. fuscipes	Italy, Russia, Iran, India, China, Taiwan, Japan, Thailand, Vietnam, Laos, Indonesia
P. nr. *fuscipes*	Papua New Guinea
P. ilsae	Israel
P. nr. *intermediua*	Philippines
P. laetus	Guatemala
P. melampus	India
P. ornaticornis	Ecuador
P. puncticollis	Uganda
P. riparius	Russia
P. rufocyaneus	Malawi
P. sabaeus	Sierra Leone, Nigeria, Zaire, Cameroon, Namibia, Tanzania, Uganda
P. signaticornis	Guatemala, Panama
P. tamulus	China
Paederus spp.	Northern Iran, Pakistan, Malaysia, Sri Lanka

Modified from Frank and Kanamitsu (1987).

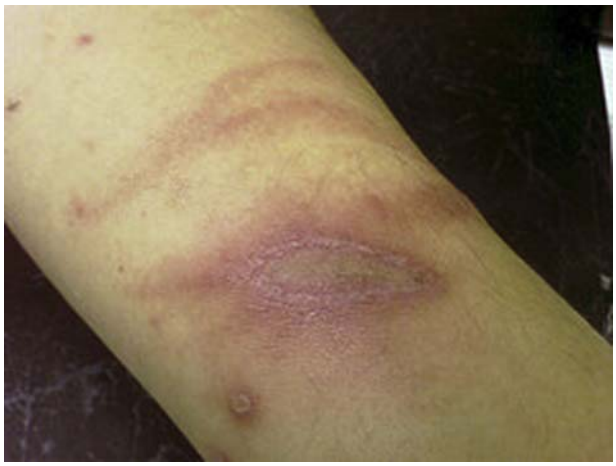


FIGURE 9.6 *Paederus* dermatitis on human forearm, caused by reaction of the skin on contact with toxin, called pederin, in the body fluids and tissues of rove beetles of the genus *Paederus* (Staphylinidae). *Courtesy of Chan Chee Keong, MD.*

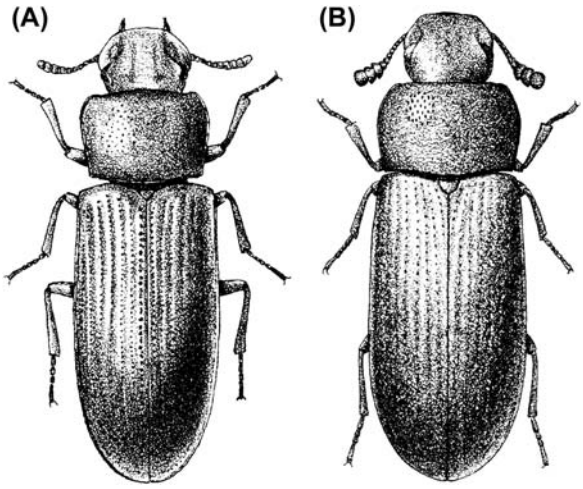


FIGURE 9.7 Darkling beetles (Tenebrionidae). (A) Confused flour beetle (*Tribolium confusum*). (B) Red flour beetle (*T. castaneum*). Defensive secretions containing quinones can cause skin irritation. *From Gorham (1991).*

Dermestidae (Larder Beetles)

Larvae of larder beetles, or pantry beetles (Fig. 9.8), are covered with barbed and spearlike setae that may cause allergic reactions in the form of pruritic, papulovesicular skin lesions. Dermestid larvae often are found living in household furnishings, such as carpets, rugs, and upholstery or stored clothing of individuals suffering from these reactions. Larder beetles are named for their common occurrence as pantry pests, but they may also be found in grain storage facilities, in bird and mammal nests and burrows, and on carrion. The larvae and adults are mostly scavengers on decaying or dry plant and animal matter.

Dermestid larvae and adults are known to have crawled into human ears, causing itching and pain. The spear-headed setae of dermestid larvae have also been observed on numerous occasions on cervical (Papanicolaou) smear slides and in sputum samples. In all of these cases the setae appear to have been contaminants that were not associated with any pathological changes in the patients (Bryant and Maslan, 1994).

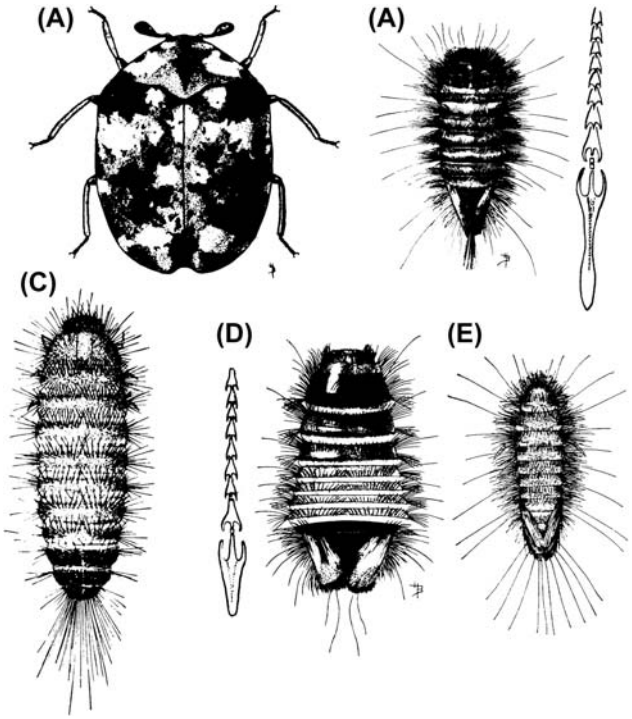


FIGURE 9.8 Dermestid beetles (Dermestidae). (A) Furniture carpet beetle (*Anthrenus flavipes*), adult. (B) Same, larva, with hastate seta (enlarged). (C) Cabinet beetle (*Trogoderma* sp.), larva. (D) Varied carpet beetle (*Anthrenus verbasci*), larva, with hastate seta (enlarged). (E) Common carpet beetle (*Anthrenus scrophulariae*), larva. *From Sweetman (1965).*

Scarabaeidae (Scarab Beetles)

In some tropical regions where human and animal excrement are abundant in the vicinity of dwellings, scarab beetles living in the dung are sometimes accidentally ingested by young children. These beetles appear in the newly passed stools of children and may disperse from the excrement in a noisy fashion that has been described in Sri Lanka as **beetle marasmus** ("kurumini mandama"). Although some of these beetles may infest the fecal matter as it is passed or after it reaches the ground, it is quite likely, as local physicians claim, that the scarab beetles (e.g., *Copris* spp., Fig. 9.9) pass through the alimentary tract and remain alive, causing little or no discomfort to the children. Evidence for such durability among the scarabs comes from cases in which frogs, horses, and cattle have ingested scarabs, which then worked their way through the stomach wall and remained alive until the hosts were killed. In Asia and Africa, humans sometimes become infested with dung beetles (*Onthophagus* and *Caccobius* spp.) when these scarabs enter the anus and live within the rectum, causing damage to the mucosa and physical discomfort.

Large numbers of the adult scarab beetles *Cyclocephala borealis* and *Autoserica castanea* invaded the ears of 186 Boy Scouts sleeping on the ground at a jamboree in Pennsylvania (USA) in 1957. The beetles caused pain and some slight bleeding as a result of the tearing action of their tibial spines. After the beetles were removed, there were very few cases of secondary infection (Mattuck and Fehn, 1958).

FIGURE 9.9 Scarab beetle, *Canthon pilularius* (Scarabaeidae), rolling dung ball, North America. *Photograph by Rachel Stone.*

Coccinellidae (Lady Beetles)

Lady beetles, also called ladybird beetles, have been cited most often as causing prickling or slight stinging sensations, followed by the formation of mild erythematous

lesions. These beetles may nip at the skin; however, given their small size, it is more likely that their defensive secretions are most often the cause of the discomfort. Under experimental conditions, where lady beetles were given direct access to skin, very small percentages of five species of lady beetles were observed biting (Ramsey and Losey, 2012). In contrast, in cases where lady beetles happen to crawl or land on persons, the alkaloid secretions produced by reflex bleeding from the legs and around the mouthparts will stain human skin and can cause mild irritation. Lady beetles are common in gardens in warm weather, where the beetles feed on aphids and scale insects on a variety of plants. Lady beetles also occur in large aggregations as they are crawling and flying from overwintering clusters.

VETERINARY IMPORTANCE

Although not generally appreciated even by entomologists, beetles are involved in a variety of problems of a veterinary nature. These include being toxic to domestic animals on ingestion, mechanically transmitting disease agents, serving as intermediate hosts for helminthic parasites, directly injuring animals as ectoparasites, and causing structural damage to poultry facilities. Beetles also can be beneficial by playing an important role in the recycling of animal dung and as natural control agents, especially for dung-breeding flies.

Ingestion of Toxic Beetles

Several blister beetles in the family Meloidae pose a hazard to livestock that feed on forage in which the living beetles are abundant. Horses that ingest quantities of these beetles are especially susceptible to **cantharidin poisoning**. Dead beetles and beetle parts retain their cantharidin content so that forage crops that are harvested for livestock feed continue to be a source of the toxin. Cantharidin is not readily degraded by heating or drying. Blister beetles in alfalfa (*Medicago sativa*) fields contain enough cantharidin to provide lethal doses to horses that feed on this material when it is used as hay. Species that pose problems in the United States include the striped blister beetle (*Epicauta vittata*), the black blister beetle (*E. pennsylvanica*) (Fig. 9.2), the margined blister beetle (*E. pestifera*), the three-striped blister beetle (*E. lemniscata*), as well as *E. fabricii*, *E. occidentalis*, and *E. temexa*. Individual beetles contain <0.1 to >11 mg of cantharidin, equivalent to $<0.1\%$ to $>12\%$ of their dry weights, with males of several species averaging $>5\%$. The minimum lethal dose for a horse is about 1 mg/kg, which means that, depending on the size of a horse and the cantharidin content of the beetles ingested, anywhere from 25 to 375 beetles are sufficient to cause death (Capinera et al., 1985).

Horses have been poisoned by eating forage in the southern, midwestern, and western United States. Poisoning is not limited to a particular geographic area because contaminated forage is transported over long distances. Affected animals exhibit moderate to severe clinical signs, ranging from depression to shock that may be followed by death. Abdominal distress (colic), anorexia, depression, fever, dehydration, gastritis, esophagitis, and oral ulcers are commonly observed. These signs are accompanied by markedly decreased serum calcium and magnesium. Accelerated heart rate (tachycardia), increased respiratory rate (tachypnea), and increased creatinine kinase activity are indicative of severe toxicosis that is likely to lead to death. In most cases in which death has occurred, a horse has succumbed within 48 h of the onset of clinical signs. Horses are the most commonly affected animals because the kinds of forage they are fed are most often contaminated with beetles. Although the ruminant digestive tract is less susceptible to cantharidin poisoning, goats, sheep, and cattle have also died from cantharidin toxicosis. Horses and cattle have also been poisoned by **pederin** following accidental ingestion of the tropical staphylinid *Paederus fuscipes*. Pederin can cause severe damage to the mucosa of the alimentary tract.

Cantharidin poisoning of emu chicks in Texas, following ingestion of the meloid *Pyrota insulata,* has been reported. Meloids feeding on nearby mesquite blossoms were attracted to lights inside a chicken barn. Severely affected chicks became ataxic, vomited, and died. Chickens are the only birds, other than emus, known to have succumbed following the ingestion of blister beetles.

Historically, there are reports of chickens, ducklings, goslings, and young turkeys dying as a direct result of ingesting the rose chafer (*Macrodactylus subspinosus*), a member of the family Scarabaeidae (Lamson, 1922). Although this North American species is abundant in the summer months, modern enclosed poultry production facilities may have greatly reduced the incidence of such poisonings.

Transmission of Pathogens

Darkling beetles (Tenebrionidae) inhabiting farm buildings can be mechanical vectors of animal pathogens. Tenebrionid beetles infesting feed in chicken houses may become infected with *Salmonella* bacteria passed in feces from infected chickens. Both larval and adult forms of the **lesser mealworm beetle** (*Alphitobius diaperinus*) (Fig. 9.10) have been found to maintain viable pathogens (e.g., *Salmonella typhimurium* and *S. chester*) on their external surfaces and in their digestive tracts. The bacteria survive for days after infection and are disseminated via beetle excreta. In chicken breeding facilities, grain beetles

FIGURE 9.10 Lesser-mealworm beetle, *Alphitobius diaperinus* (Tenebrionidae), larvae and adults; develop in litter and, as adults cause structural damage in poultry houses; can disseminate pathogenic agents via their excreta, and is an intermediate host for tapeworm *Choanotaenia infundibulum*. *Photograph by D. Wesley Watson.*

FIGURE 9.11 Tapeworm, *Choanotaenia infundibulum* (Cestoda: Dilepididae), parasite in small intestine of chickens, turkeys, pheasants, and guinea fowl; uses tenebrionid and dermestid beetles as intermediate hosts. *Photograph by Nancy C. Hinkle.*

are potential disseminators of these pathogens that can infect both chicks and adult birds. These organisms can cause gastroenteritis in human consumers. The lesser mealworm also is regarded as a potential disseminator of other bacteria (*Escherichia*, *Bacillus*, *Streptococcus*), fungi (*Aspergillus*), and the viruses causing **Marek disease**, **Newcastle disease**, **fowlpox**, **avian influenza**, **enteritis**, and **infectious bursitis** (Gumboro disease). In addition, oocysts formed by protozoans in the genus *Eimeria* are ingested by lesser mealworm beetles, and when the infected beetles are ingested by birds, the latter develop **avian coccidiosis**, a serious disease of poultry.

Ten years after *Alphitobius diaperinus* was eradicated from a chicken farm, an infestation of the hairy fungus beetle, *Typhaea stercorea*, a mycetophagid, was found to act as a reservoir of *Salmonella* in a chicken house and to be the source of continuing infections in chicks.

Mycobacterium avium subspecies, the agents of avian tuberculosis, remain viable in larval and adult tenebrionids (*Tenebrio molitor)* after the beetles have been experimentally infected. Although the bacteria have not been recovered from naturally infected beetles, there is the potential for mechanical transmission of mycobacteria to birds after the ingestion of beetles.

Intermediate Hosts of Parasites

Tapeworms (cestodes), flukes (trematodes), roundworms (nematodes), and thorny-headed worms (acanthocephalans) of many species that infest domestic and wild animals use beetles as intermediate hosts. Animals become infested by ingesting parasitized beetles that contaminate feed or bedding (tenebrionids, carabids) or that are attracted to animal dung (scarabaeids) or by ingesting water in which infective beetles have disintegrated.

Two tapeworms that infest the small intestines of poultry are the broad-headed tapeworm (*Raillietina cesticillus*) and *Choanotaenia infundibulum* (Fig. 9.11). Both parasites cause enteritis and hemorrhaging in chickens, turkeys, pheasants, and guinea fowl. A few tenebrionids and scarabaeids and more than 35 species of carabid beetles, notably in the genera *Amara* and *Pterostichus*, are intermediate hosts for *R. cesticillus* (Cheng, 1973). Some tenebrionid and dermestid species, including the lesser mealworm beetle, are intermediate hosts for *C. infundibulum*. Proglottids or tapeworm eggs ingested by beetle larvae or adults develop into cystercerci (encysted larvae) that can then infest birds that eat the beetles. Chicks are most susceptible to serious infestations and often die from worm burdens.

The beef tapeworm (*Taenia saginata*) can use dung beetles and carabids as intermediate hosts, although they are not essential for transmission. Beetles associated with infective dung or debris can ingest proglottids or eggs as in the case of poultry worms. Cattle and humans infested with the tapeworm may exhibit mild symptoms such as weight loss, abdominal pain, and increased appetite.

The dwarf tapeworms (*Hymenolepis nana* and *H. diminuta*) that usually infest rodents, especially rats and mice, can infest humans when the intermediate host beetles are accidentally ingested. *Tenebrio molitor* may act as an intermediate host for *H. nana*, although this worm is readily transmitted directly from one vertebrate host to another. Several species of tenebrionids (*Tenebrio* spp. and *Tribolium* spp.) are required intermediate hosts for *H. diminuta*. Larval and adult beetles infesting grain and cereals ingest worm eggs that develop into cysticercoid stages that infest rodents or humans, usually children, who ingest the beetles. Dwarf tapeworms produce minimal symptoms in rodents and people, although heavy infestations in children may cause abdominal pain, diarrhea, convulsions, and dizziness.

Beetles are known to be intermediate hosts for only a few trematodes. These are parasites of frogs that become infested by ingesting parasitized dytiscid beetles and pose no problem for other vertebrate animals.

Many nematodes infest livestock and wildlife, but only a few use beetles as intermediate hosts. Spirurid nematodes of various species infest livestock and, rarely, humans. *Physocephalus sexalatus* and *Ascarops strongylina* eggs develop in many species of scarabaeid dung beetles (*Geotrupes* spp., *Onthophagus* spp., and *Scarabaeus* spp.) that then may be ingested by pigs. Both wild and domestic swine can be infested with these stomach worms that cause digestive problems in heavily infested young animals. *Gongylonema pulchrum* is a parasite of the upper digestive tract of sheep, cattle, goats, and other ruminants as well as horses, dogs, and humans. The worms burrow in the mucosa and submucosa of the oral cavity and esophagus and may cause bleeding, irritation, numbness, and pain in the mouth and chest. Scarabaeid and tenebrionid beetles serve as intermediate hosts for the larvae. Scarabaeid dung beetles are also the intermediate hosts for *Spirocerca lupi*, the esophageal worm of dogs and wild canids. *Physaloptera caucasica*, another spirurid, often parasitizes monkeys in tropical Africa, where humans are also commonly infested. This nematode causes digestive distress by infesting the alimentary tract from the esophagus to the terminal ileum. Scarabaeid dung beetles are its intermediate hosts.

The acanthocephalans, aptly named for their thorny heads, include species found worldwide infesting swine, rodents, and carnivores such as dogs. *Macracanthorhynchus hirudinaceus*, which attaches to the small intestines of swine, causes enteritis and produces intestinal nodules that lower the value of these tissues when they are sold to make sausage casings. Eggs of this parasite are ingested by scarab beetle larvae of species belonging to various genera (*Phyllophaga*, *Melolontha*, *Lachnosterna*, *Cetonia*, *Scarabaeus*, and *Xyloryctes*), including May and June beetles, leaf chafers, dung beetles, and rhinoceros beetles. Infested beetle larvae, as well as the pupae and adults that develop from them, are infective to both pigs and humans. Humans and pigs often show no symptoms. However, in cases of heavy infestations, both human and porcine hosts may experience digestive problems, such as abdominal pain, loss of appetite, and diarrhea that can lead to emaciation.

Two other acanthocephalan worms that parasitize the small intestines of their hosts use scarab beetles or tenebrionids as intermediate hosts. They are *Macracanthorhynchus ingens*, which infests raccoons and occasionally dogs and humans, and *Moniliformis moniliformis*, a parasite of rodents and dogs.

Nest Associates and Ectoparasites

In addition to those beetles that occasionally invade the alimentary tracts or sense organs of animals, other species have evolved in close association with mammals as nest dwellers or ectoparasites. These species typically have reduced eyes and wings or have lost these structures completely. Some members of the family Leiodidae, known as mammal nest beetles, include *Platypsyllus* sp. that live as larvae and adults on beavers and *Leptinus* and *Leptinillus* spp. that live as adults on various small rodents (these three genera were formerly included in the family Leptinidae). These species feed on skin debris or glandular secretions and have been associated with skin lesions on their hosts. *Loberopsyllus* spp. (Erotylidae, Cryptophilinae) found in Costa Rica also live on rodents and feed on particles of skin, hair, and other organic debris (Bouchard, 2014). Some rove beetles (Staphylinidae: *Amblyopinus* spp.) appear to be mutualistically symbiotic with their mammalian hosts. These beetles infest mammalian fur to gain access to their prey, which are mammalian ectoparasites, such as fleas and mites that live in rodent nests (Ashe and Timm, 1987; Durden, 1987).

Some scarabaeid beetles are adapted to living in the fur around the anus of certain mammals. These beetles cling to the fur except when they leave to oviposit in the dung. Some of these scarabs (*Trichillum* spp.) are found on sloths and monkeys in South America, and others (*Macropocopris* spp. and *Onthophagus* spp.) are found on marsupials in Australia.

Larvae and adults of the **lesser mealworm beetle** (*Alphitobius diaperinus*) have been found boring into and living in the scrotum of a rat and feeding on sick domestic chicks and young pigeons. Similarly, the **hide beetle** (*Dermestes maculatus*) can feed on living poultry and has caused deep wounds in adult turkeys. In laboratory experiments, lesser mealworm beetles killed snakes and a salamander, all of which were devoured by the mealworms. The voracious and aggressive behavior of this commonly abundant tenebrionid makes it a significant pest in poultry houses.

In addition to their direct attacks on birds, lesser mealworm and hide beetle larvae are major causes of structural damage to poultry houses. After reaching their final instar, the larvae migrate into the insulation of poultry houses to seek pupation sites. The larval tunnels and holes produced in insulation and wood framing cause enough damage to alter temperature regulation in the houses, which reduces the efficiency of poultry production (Axtell and Arends, 1990).

Dung Beetles and Biocontrol

Many beetles that are attracted to avian and mammalian excrement should be viewed as beneficial insects. Scarabaeid beetles (including coprines) remove large quantities of mammalian dung by scattering or burying the material during feeding or reproduction. The rapid removal of dung helps to reduce the development of parasitic worms and

pestiferous cyclorrhaphan flies that require dung for their survival and reproduction and opens up grazing land that would be despoiled by the rotting excrement. Staphylinid beetles and histerid beetles that are attracted to mammalian and avian dung directly reduce muscoid fly populations by feeding on the immature stages of these flies and indirectly by introducing their phoretic mites that prey on fly eggs in the excrement.

Dung beetle diversity is greatest in tropical regions, such as Africa, with its abundance of herbivores. More than 2,000 scarabaeid species in many genera (e.g., *Onthophagus*, *Euoniticellus*, and *Heliocopris*) are known to feed and reproduce in dung in Africa. Less diverse dung-feeders, such as members of the genera *Aphodius*, *Onthophagus*, *Canthon,* and *Phanaeus*, provide the same benefits in the United States. In Australia, the development of extensive cattle farming resulted in the production of millions of tons of dung that was not naturally removed, because the native coprophagous beetles were adapted only to feeding on marsupial dung. Within the past four decades, introductions of African beetles by sterile breeding programs have established several coprine species that have helped to open up grazing lands previously ruined by dung accumulation and to reduce the breeding source of the pestiferous bush fly (*Musca vetustissima*).

Staphylinid beetles of several species in the genus *Philonthus* feed as both larvae and adults on fly larvae living in animal excrement. These beetles are maintained as components in biological control programs against the face fly and horn fly. Staphylinid species in the genus *Aleochara* are also helpful in reducing dung-breeding fly populations because the parasitoid larvae of these beetles penetrate fly puparia and destroy the fly pupae. Histerid beetles, especially *Carcinops* spp. are found in confined animal production facilities, such as poultry houses. The larvae and adults of these beetles feed on eggs and larvae of muscoid flies. Any beetle species observed in animal production facilities should be identified to assess whether its presence is beneficial or detrimental to the maintenance of sanitary conditions and animal health.

PREVENTION AND CONTROL

Preventing public health and veterinary problems associated with beetles requires education about which species are harmful. Recognition of meloid, paederine, and oedemerid beetles allows one to immediately wash skin surfaces and eyes that come into contact with the beetles, thereby removing the chemicals that cause dermatitis or inflammation of the eyes. With the exception of the smallest species, beetles that are attracted to lights may be prevented from reaching humans or other animals by using screens and bed netting.

Control of vesicatory beetles occurring in natural and cultivated vegetation can be achieved with pesticides; however, the wide area over which these chemicals must be broadcast and the potential for killing beneficial insects generally make such control impractical. Human exposure can be prevented by combining education about the problem with personal protective measures and removal of extraneous vegetation and decaying organic matter from around agricultural fields and dwellings.

Prevention of blister beetle toxicosis of farm animals involves care in the handling of forage crops. Harvesting hay at times when meloid beetles are rare, such as in late fall in temperate climates, helps prevent the contamination of dried, stored forage with dead beetles. Similarly, harvesting alfalfa before it produces the blooms that attract meloids or raking hay more frequently after it is cut and allowing it to dry longer before it is conditioned or crimped will allow beetles to leave the hay before it is baled.

Preventing and controlling dissemination of pathogens and transmission of helminths of veterinary importance can be achieved with a combination of strict sanitary and cultural practices. Removal of dung and organic waste from animal enclosures, as well as sterilization of manure before it is used as fertilizer, helps to interrupt the transmission cycles of parasites by reducing the chances of beetles ingesting worm eggs. Rotation of pastured animals also can limit contact between the definitive hosts and intermediate beetle hosts. Increased abundance of scarabaeid dung beetles that aids in the rapid removal of dung, via both ingestion and burial, has been found beneficial in reducing infestations with intestinal nematodes that do not use beetles as intermediate hosts but that are transmitted from animal to animal via dung ingestion (Fincher, 1975).

Control of destructive poultry-house beetles requires constant monitoring for the insects and strict sanitation. Pesticides provide only temporary control and are most beneficial when applied to soil into which larvae may burrow to pupate. Recent interest in plant extracts for insect control has led to the demonstration of *Cunila angustifolia* essential oil as an effective larvicide and insecticide against the lesser mealworm, *Alphitobius diaperinus* (Prado et al., 2013).

Careful personal hygiene, use of gowns and masks in beetle-rearing facilities, and regular vacuuming of floors, floor coverings, and furniture in domestic settings help prevent exposure to dermestids and other beetles that can cause allergic responses.

REFERENCES AND FURTHER READING

Alexander, J. O. (1984). *Arthropods and human skin*. Berlin: Springer-Verlag.

Archibald, R. G., & King, H. H. (1919). A note on the occurrence of a coleopterous larva in the urinary tract of man in the Anglo-Egyptian Sudan. *Bulletin of Entomological Research, 9*, 255–256.

Arnett, R. H., Jr., & Thomas, M. C. (2001). *American beetles, Vol. 1 — Archostemata, Myxophaga, Adephaga, Polyphaga: Staphyliniformia*. Boca Raton: CRC Press.

Arnett, R. H., Jr., Thomas, M. C., Skelley, P. E., & Frank, J. H. (Eds.). (2002). *American beetles, Vol. 2 — Polyphaga: Scarabaeoidea through Curculionoidea*. Boca Raton: CRC Press.

Arnett, R. H., Jr. (1984). *The false blister beetles of Florida (Coleoptera: Oedemeridae)*. Florida Department of Agriculture & Consumer Services, Entomology Circular 259, 4 p.

Ashe, J. S., & Timm, R. M. (1987). Predation by and activity patterns of 'parasitic' beetles of the genus *Amblyopinus* (Coleoptera: Staphylinidae). *Journal of Zoology, London, 212*, 429–437.

Avancini, R. M. P., & Ueta, M. T. (1990). Manure breeding insects (Diptera and Coleoptera) responsible for cestoidosis in caged layer hens. *Journal of Applied Entomology, 110*, 307–312.

Axtell, R. C., & Arends, J. J. (1990). Ecology and management of arthropod pests of poultry. *Annual Review of Entomology, 35*, 101–126.

Bailey, W. S., Cabrera, D. J., & Diamond, D. L. (1963). Beetles of the family Scarabaeidae as intermediate hosts for *Spirocerca lupi*. *The Journal of Parasitology, 49*, 485–488.

Barr, A. C., Wigle, W. L., Flory, W., Alldredge, B. E., & Reagor, J. C. (1998). Cantharidin poisoning of emu chicks by ingestion of *Pyrota insulata*. *Journal of Veterinary Diagnostic Investigation, 10*, 77–79.

Barrera, A. (1969). Notes on the behaviour of *Loberopsyllus traubi*, a cucujoid beetle associated with the volcano mouse, *Neotomodon alstoni* in Mexico. *Proceedings of the Entomological Society of Washington, 71*, 481–486.

Bellas, T. E. (1989). *Insects as a cause of inhalational allergies: A bibliography 1900–1987*. Canberra: CSIRO Division of Entomology.

Berkov, A., Rodriguez, N., & Centeno, P. (2008). Convergent evolution in the antennae of a cerambycid beetle, *Onychocerus albitarsis*, and the sting of a scorpion. *Naturwissenschaften, 95*, 257–261.

Blodgett, S. L., & Higgins, R. A. (1990). Blister beetles (Coleoptera: Meloidae) in Kansas alfalfa: Influence of plant phenology and proximity to field edge. *Journal of Economic Entomology, 83*, 1042–1048.

Blodgett, S. L., Carrel, J. E., & Higgins, R. A. (1991). Cantharidin content of blister beetles (Coleoptera: Meloidae) collected from Kansas alfalfa and implications for inducing cantharidiasis. *Environmental Entomology, 20*, 776–780.

Blume, R. R. (1985). A checklist, distributional record, and annotated bibliography of the insects associated with bovine droppings on pastures in America north of Mexico. *Southwestern Entomologist Supplement, 9*, 1–55.

Bong, L.-J., Neoh, K.-B., Jaal, Z., & Lee, C.-Y. (2015). *Paederus* outbreaks in human settings: A review of current knowledge. *Journal of Medical Entomology, 52*, 517–526.

Bouchard, P. (Ed.). (2014). *The book of beetles: A life-size guide to six hundred of nature's gems*. The University of Chicago Press.

Bryant, J., & Maslan, A. M. (1994). Carpet beetle larval parts in Pap smears: Report of two cases. *Southern Medical Journal, 87*, 763–764.

Capinera, J. L., Gardner, D. R., & Stermitz, F. R. (1985). Cantharidin levels in blister beetles (Coleoptera: Meloidae) associated with alfalfa in Colorado. *Journal of Economic Entomology, 78*, 1052–1055.

Cheng, T. C. (1973). *General parasitology* (pp. 868–869). New York: Academic Press.

Christmas, T. I., Nicholls, D., & Greig, D. (1987). Blister beetle dermatosis in New Zealand. *New Zealand Medical Journal, 100*, 515–517.

Chu, G. S. T., Palmieri, J. R., & Sullivan, J. T. (1977). Beetle-eating: A Malaysian folk medical practice and its public health implications. *Tropical and Geographical Medicine, 29*, 422–427.

Clausen, C. P. (1972). *Entomophagous insects*. New York: Hafner Publ.

Crook, P. G., Novak, J. A., & Spilman, T. J. (1980). The lesser mealworm, *Alphitobius diaperinus*, in the scrotum of *Rattus norvegicus*, with notes on other vertebrate associations (Coleoptera, Tenebrionidae; Rodentia, Muridae). *The Coleopterists Bulletin, 34*, 393–396.

Crowson, R. A. (1981). *The biology of the Coleoptera*. London: Academic Press.

De las Casas, E., Harein, P. K., Deshmukh, D. R., & Pomeroy, B. S. (1976). Relationship between the lesser mealworm, fowl pox, and Newcastle disease virus in poultry. *Journal of Economic Entomology, 69*, 775–779.

Dumbacher, J. P., Wako, A., Derrickson, A., Samuelson, S. R., Spande, T. F., & Daly, J. W. (2004). Melyrid beetles (*Choresine*): A putative source for the batrachotoxin alkaloids found in poison-dart frogs and toxic passerine birds. *Proceedings of the National Academy of Sciences, 101*, 15857–15860.

Durden, L. A. (1987). Predator-prey interactions between ectoparasites. *Parasitology Today, 3*, 306–308.

Eisner, T., Conner, J., Carrel, J. E., McCormick, J. P., Slagle, A. J., Gans, C., et al. (1990). Systemic retention of ingested cantharidin in frogs. *Chemoecology, 1*(2), 57–62.

Eisner, T., Eisner, M., & Siegler, M. (2005). *Secret weapons — defenses of insects, spiders, scorpions, and other many-legged creatures*. Cambridge: Belknap Press of Harvard University.

Eschevarria, C. (2006). *Blister beetle poisoning: Cantharidin toxicosis in equines*. Animal Disease Diagnostic Laboratory. Purdue newsletter http://www.addl.purdue.edu/newsletters/2006/Fall/EquineCT.htm.

Essig, E. O. (1942). *College entomology*. New York: MacMillan Company.

Fincher, G. T. (1975). Effects of dung beetle activity on the number of nematode parasites acquired by grazing cattle. *The Journal of Parasitology, 61*, 759–766.

Fincher, G. T. (1994). Predation on the horn fly by three exotic species of *Philonthus*. *Journal of Agricultural Entomology, 11*, 45–48.

Fischer, O. A., Matiova, L., Dvorska, L., Svastova, P., Peral, D. L., Weston, R. T., et al. (2004). Beetles as possible vectors of infections caused by *Mycobacterium avium* species. *Veterinary Microbiology, 102*(3–4), 247–255.

Frank, J. H., & Kanamitsu, K. (1987). *Paederus,* sensu lato (Coleoptera: Staphylinidae): Natural history and medical importance. *Journal of Medical Entomology, 24*, 155–191.

Geden, C. J., Stinner, R. F., & Axtell, R. C. (1988). Predation by predators of the house fly in poultry manure: Effects of predator density, feeding history, interspecific interference and field conditions. *Environmental Entomology, 17*, 320–329.

Gibbs, L. M. (2015). Beware of the beetle: A case report of severe vesicating dermatitis. *Military Medicine, 180*, e1293–1295.

Gorham, J. R. (Ed.). (1991). *Insect and mite pests in food: An illustrated key*. U.S. Department of Agriculture, Agriculture Handbook No. 655.

Hald, B., Olsen, A., & Madsen, M. (1998). *Typhaea stercorea* (Coleoptera: Mycetophagidae), a carrier of *Salmonella enterica* serovar Infantis in a Danish broiler house. *Journal of Economic Entomology, 91*, 660–664.

Hall, M. C. (1929). Arthropods as intermediate hosts of helminths. *Smithsonsonian Miscellaneous Collections, 81*, 77 p.

Hanski, I., & Cambefort, Y. (Eds.). (1991). *Dung beetle ecology*. Princeton Univ. Press.

Harde, K. W. (1984). *A field guide in colour to beetles*. London: Octopus Books Ltd.

Hazeleger, W. C., Bolder, N. M., Beumer, R. R., & Jacobs-Reitsma, W. F. (2008). Darkling beetles (*Alphitobius diaperinus*) and their larvae as potential vectors for the transfer of *Campylobacter jejuni* and *Salmonella enterica* serovar *Paratyphi* B variant Java between successive broiler flocks. *Applied and Environmental Microbiology, 74*, 6887–6891.

Kellner, R. L. (2002). Molecular identification of an endosymbiotic bacterium associated with pederin biosynthesis in *Paederus sabaeus* (Coleoptera: Staphylinidae). *Insect Biochemistry and Molecular Biology, 32*, 389–395.

Lamson, G. H., Jr. (1922). The rose chafer as a cause of death of chickens. *Storrs Agricultural Experiment Station Bulletin, 110*, 118–135.

Lawrence, J. F., & Britton, E. B. (1994). *Australian beetles*. Carlton, Victoria: Melbourne Univ. Press.

Legner, E. F. (1995). Biological control of Diptera of medical and veterinary importance. *Journal of Vector Ecology, 20*, 59–120.

Ligett, H. (1931). Parasitic infestations of the nose. *Journal of the American Medical Association, 96*, 1571–1572.

Marshall, A. G. (1981). *The ecology of ectoparasitic insects*. London: Academic Press.

Mattuck, D. R., & Fehn, C. F. (1958). Human ear invasions by adult scarabaeid beetles. *Journal of Economic Entomology, 51*, 546–547.

McAllister, J. C., Steelman, C. D., & Skeeles, J. K. (1994). Reservoir competence of the lesser mealworm (Coleoptera: Tenebrionidae) for *Salmonella typhimurium* (Eubacteriales: Enterobacteriaceae). *Journal of Medical Entomology, 31*, 369–372.

McAllister, J. C., Steelman, C. D., Skeeles, J. K., Newberry, L. A., & Gbur, E. E. (1996). Reservoir competence of *Alphitobius diaperinus* (Coleoptera: Tenebrionidae) for *Escherichia coli* (Eubacteriales: Enterobacteriaceae). *Journal of Medical Entomology, 33*, 983–987.

Mokhtar, A. S., Sridhor, G. S., Mahmud, R., Jeffrey, J., Lau, Y. L., Wilson, J.-J., et al. (2016). First report of cantharisis in an infant caused by the larvae of *Lasioderma serricorne* (Coleoptera: Anobiidae). *Journal of Medical Entomology, 53*, 1234–1237.

Nicholls, D. S. H., Christmas, T. I., & Greig, D. E. (1990). Oedemerid blister beetle dermatosis: A review. *Journal of the American Academy of Dermatology, 22*, 815–819.

Okumura, G. T. (1967). A report of cantharisis and allergy caused by *Trogoderma* (Coleoptera: Dermestidae). *California Vector Views, 14*, 19–22.

Prado, G. P. D., Stefmi, L. M., Silva, A. S. D., Smaniotto, L. F., Garcia, F. R. M., & Moura, N. F. D. (2013). *Alphitobius diaperinus* (Coleoptera: Tenebrionidae) susceptibility to *Cunila angustifolia* essential oil. *Journal of Medical Entomology, 50*, 1040–1045.

Qadir, S. N. R., Raza, N., & Rahman, S. B. (2006). *Paederus* dermatitis in Sierra Leone. *Dermatology Online Journal, 12*(7), 9. http://dermatology.cdlib.org/127/case_reports/paederus/qadir.html.

Rajapakse, S. (1981). Letter from Sri Lanka: Beetle marasmus. *British Medical Journal, 283*, 1316–1317.

Ramsey, S., & Losey, J. E. (2012). Why is *Harmonia axyridis* the culprit in coccinellid biting incidents? An analysis of means, motive, and opportunity. *American Entomologist, 58*, 166–170.

Samish, M., Argaman, Q., & Perlman, D. (1992). The hide beetle, *Dermestes maculatus* DeGeer (Dermestidae), feeds on live turkeys. *Poultry Science, 71*, 388–390.

Samlaska, C. P., Samuelson, A., Faran, M. E., & Shparago, N. I. (2008). Blister beetle dermatosis in Hawaii caused by *Thelyphassa apicata* (Fairmaire). *Pediatric Dermatology, 9*, 246–250.

Schmitz, D. G. (1989). Cantharidin toxicosis in horses. *Journal of Veterinary Internal Medicine, 3*, 208–215.

Schroeckenstein, D. C., Meier-Davis, S., & Bush, R. K. (1990). Occupational sensitivity to *Tenebrio molitor* Linnaeus (yellow mealworm). *The Journal of Allergy and Clinical Immunology, 86*, 182–188.

Selander, R. B. (1988). An annotated catalog and summary of bionomics of blister beetles of the genus *Cylindrothorax* (Coleoptera: Meloidae). *Transactions of the American Entomological Society, 114*, 15–70.

Southcott, R. V. (1989). Injuries from Coleoptera. *Medical Journal of Australia, 151*, 654–659.

Strother, K. O., Steelman, C. D., & Gbur, E. E. (2005). Reservoir competence of lesser mealworm (Coleoptera: Tenebrionidae) for *Campylobacter jejuni* (Campylobacteriales: Campylobacteraceae). *Journal of Medical Entomology, 42*, 42–47.

Sweetman, H. L. (1965). *Recognition of structural pests and their damage*. Dubuque: Wm. C. Brown Co.

Théodoridès, J. (1950). The parasitological, medical and veterinary importance of Coleoptera. *Acta Tropica, 7*, 48–60.

Waterhouse, D. F. (1974). The biological control of dung. *Scientific American, 230*, 100–109.

Watson, D. W., Guy, J. S., & Stringham, S. M. (2000). Limited transmission of Turkey coronavirus in young turkeys by adult *Alphitobius diaperinus* (Coleoptera: Tenebrionidae). *Journal of Medical Entomology, 37*, 480–483.

Weatherston, J., & Percy, J. E. (1978). Venoms of Coleoptera. In S. Bettini (Ed.), *Arthropod venoms* (pp. 511–554). Berlin: Springer -Verlag.

White, R. E. (1983). *A field guide to the beetles of North America*. Boston: Houghton Mifflin Co.

Whitmore, R. W., & Pruess, K. P. (1982). Response of pheasant chicks to adult lady beetles (Coleoptera: Coccinellidae). *Journal of the Kansas Entomological Society, 55*, 474–476.

Woodruff, R. E. (1973). *Scarab beetles of Florida (Coleoptera: Scarabaeidae), part 1. Arthropods of Florida* (Vol. 8). Gainesville: Division of Plant Industry, Florida Department of Agriculture.

This page intentionally left blank

Chapter 10

Fleas (Siphonaptera)

Lance A. Durden[1] and Nancy C. Hinkle[2]

[1]Department of Biology, Georgia Southern University, Statesboro, GA, United States; [2]Department of Entomology, University of Georgia, Athens, GA, United States

Fleas are morphologically unique ectoparasites that are unlikely to be confused with any other arthropods. They are a monophyletic group that evolved from mecopteran (scorpion fly) winged ancestors during the early Cretaceous 120–130 million years ago in parallel with marsupial and insectivore hosts. Reports of giant fossil fleas from Jurassic and early Cretaceous China, possibly as ectoparasites of dinosaurs, are unsubstantiated (Dittmar et al., 2016). Whiting et al. (2008) and Zhu et al. (2015) provide molecular phylogenetic analyses for fleas. As a group, fleas have evolved principally as parasites of mammals on which ~94% of known species feed, representing 18 families and more than 200 genera of fleas. The remaining ~6%, representing five families and 25 genera, are ectoparasites of birds.

Cospeciation or coevolution has molded many host–flea associations, as reflected by the host specificity exhibited by large numbers of species and the morphological adaptations of some fleas that partially conform to the morphology of the host skin, fur, or feathers. Although many flea species do not cause significant harm to their hosts in nature, most species that feed on humans and their companion animals have medical or veterinary importance.

TAXONOMY

There are approximately 2,500 described species and subspecies of fleas that are currently placed in 18 families and 220 genera (Lewis, 1993a, 1998; Zhu et al., 2015). Many of these species have been catalogued by Hopkins and Rothschild (1953–1971), Mardon (1981), Traub et al. (1983), and Smit (1987). Except for the work by Traub et al. (1983), these publications are part of an eight-volume series published by the British Museum (Natural History), now the Natural History Museum, London. Another series of publications that addresses the geographical distribution, host preferences, and classification of the world flea fauna are those of Lewis (1972, 1973, 1974a, 1974b, 1974c, 1974d, 1993a) and Lewis and Lewis (1985). Lewis and Eckerlin (2018) prepared an identification guide to the fleas of North America. Holland (1985) produced a guide to the fleas of Canada, Alaska, and Greenland. An earlier work by Fox (1940) and a key by Benton (1983) can also be used to identify fleas from the eastern United States. Benton (1980) also provided an atlas outlining the geographical distributions of the fleas of this region. Lewis et al. (1988) provide a guide to the fleas of the US Pacific Northwest. Identification guides to the fleas of Britain (Whitaker, 2007), western Europe (Beaucournu and Launay, 1990), and several other regions are also available. Although flea larvae can be difficult to assign to genus or species, Elbel (1991) provides a useful guide for identifying the larvae of some flea taxa.

Most fleas of medical or veterinary importance are members of the family Pulicidae, with other important fleas belonging to the Tungidae, Ceratophyllidae, Leptopsyllidae, or Vermipsyllidae. Occasionally, members of other families, notably, the Hystrichopsyllidae and Rhopalopsyllidae, also feed on humans and domestic animals. Table 10.1 shows the family-level classification for flea species discussed in this chapter.

Flea classification is based almost exclusively on the chitinous morphology of cleared adult specimens, but molecular identification techniques are becoming available for some species. Male fleas may have the most complex genitalia in the animal kingdom, and the morphology of the sclerotized parts of these organs is important in most systems of flea classification. Although various classification schemes have been proposed for fleas, one that is widely used today is detailed by Lewis (1998). In this classification, the order Siphonaptera is divided into 15 families, the largest of which are the Ctenophthalmidae (744 species), Ceratophyllidae (540 species), Leptopsyllidae (346 species), Pulicidae (207 species), Pygiopsyllidae (185 species), Rhopalopsyllidae (145 species), and Ischnopsyllidae (135

Medical and Veterinary Entomology. https://doi.org/10.1016/B978-0-12-814043-7.00010-8

TABLE 10.1 Classification of Flea Species Mentioned in the Text

Family Pulicidae:
Cediopsylla simplex (rabbit flea)
Ctenocephalides canis (dog flea)
Ctenocephalides felis (cat flea)
Echidnophaga gallinacea (sticktight flea)
Echidnophaga larina
Echidnophaga myrmecobii
Euhoplopsyllus glacialis
Hoplopsyllus anomalus
Pulex irritans (human flea)
Pulex simulans
Spilopsyllus cuniculi (European rabbit flea)
Xenopsylla astia
Xenopsylla bantorum
Xenopsylla brasiliensis
Xenopsylla cheopis (Oriental rat flea)

Family Tungidae:
Tunga monositus
Tunga penetrans (chigoe)
Tunga trimamillata

Family Pygiopsyllidae:
Uropsylla tasmanica

Family Ctenophthalmidae:
Stenoponia tripectinata

Family Vermipsyllidae:
Dorcadia ioffi
Vermipsylla alakurt (alakurt flea)

Family Leptopsyllidae:
Leptopsylla segnis (European mouse flea)

Family Ischnopsyllidae:
Myodopsylla insignis

Family Ceratophyllidae:
Ceratophyllus gallinae (European chicken flea [hen flea in Britain])
Ceratophyllus niger (western chicken flea)
Nosopsyllus fasciatus (northern rat flea)
Orchopeas howardi (squirrel flea)
Oropsylla montana

Modified from Lewis (1993a) and Zhu et al. (2015).

species). Zhu et al. (2015) recognize 18 families within the Siphonaptera, notably splitting the Pygiopsyllidae into three families and elevating the Tunginae from a subfamily of the Pulicidae to full family status, the Tungidae.

MORPHOLOGY

Adult fleas are small (1−8 mm), wingless, typically bilaterally compressed, and heavily chitinized (Figs. 10.1 and 10.2). The hind legs are enlarged (Fig. 2.4B) and adapted for jumping. Many species bear one or more combs, or **ctenidia**, each appearing as a row of enlarged, sclerotized spines (Figs. 10.1 and 10.2). A comb on the ventral margin

FIGURE 10.1 Rabbit flea, *Cediopsylla simplex*; stacked image of cleared female. *Original image by Lorenza Beati and Lance A. Durden.*

of the head is called a **genal ctenidium**, whereas a comb on the posterior margin of the prothorax is called a **pronotal ctenidium**. Additional cephalic or abdominal ctenidia occur in some fleas. Smaller rows of specialized setae or bristles are present on various body regions of many fleas. The nature of the ctenidia and specialized setae often reflects the pelage or habits of the host, especially in host-specific fleas. They aid in preventing dislodgement of fleas from the hair or feathers of the host. It also has been suggested that ctenidia may protect flexible joints.

An important sensory feature of adult fleas is the **sensilium** (pygidium), present on abdominal tergum 9 or 10 (Figs. 10.2 and 10.3). This sensory organ aids fleas in detecting air movement, vibrations, and temperature gradients; in some species it also facilitates copulation. It has an important role in host detection and in initiating escape responses. Just anterior to the sensilium in most fleas are the stout, paired **antesensilial setae** (**antepygidial bristles**). Many adult fleas, especially those of diurnal hosts, possess well-developed noncompound eyes (Fig. 10.1), which are actually clusters of ocelli. Eyes are well-developed in most adult fleas of medical or veterinary importance. Short, clubbed, three-segmented antennae are held inside protective grooves called antennal fossae on the sides of the head, which prevent antennal damage as the flea moves through the pelage of its host.

The mouthparts of adult fleas are well-adapted for piercing and sucking (Fig. 2.2D and 10.2). After a suitable feeding site has been located by the sensory labial palps,

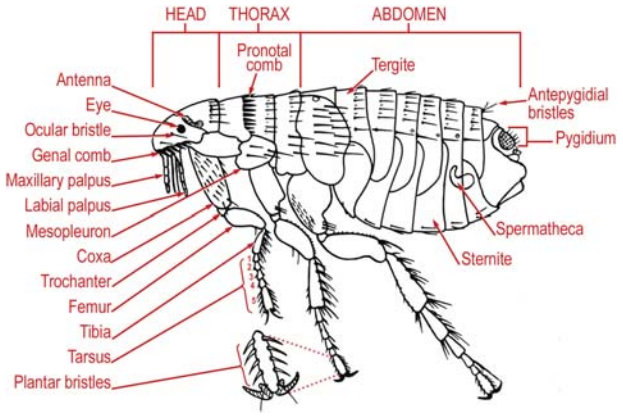


FIGURE 10.2 Morphology of a generalized adult female flea. *Courtesy, U.S. Public Health Service, Public Health Image Library.*

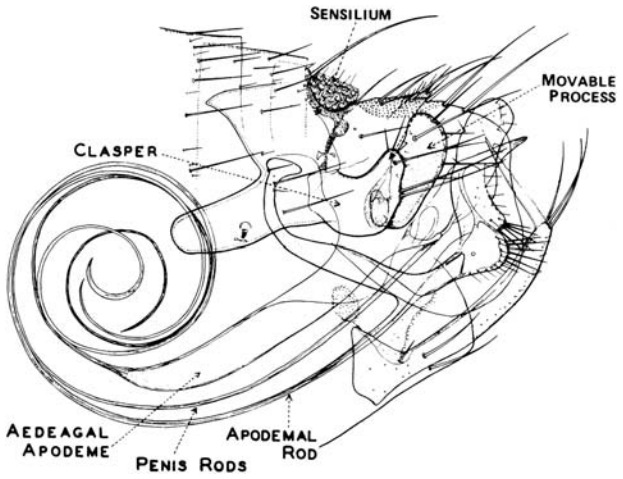


FIGURE 10.3 Genitalia of a male flea, the European chicken flea (hen flea) (*Ceratophyllus gallinae*). *Modified from Smit, 1957.*

three slender, elongate structures collectively called **stylets** or the fascicle are used to pierce the host skin. The three stylets consist of two lateral, blade-like maxillary laciniae and the central epipharynx (Fig. 2.2D). The laciniae penetrate the host skin and the tip of the epipharynx enters a host capillary. A salivary canal is formed by the closely appressed medial surfaces of the two laciniae. A food canal is formed at the confluence of the laciniae with the epipharynx (Fig. 2.2D). Anticoagulants including the antiplatelet enzyme **apyrase**, other salivary components, and sometimes allergens or pathogens are introduced into the bite wound via the salivary canal while host blood is imbibed through the food canal. In some sedentary fleas, such as sticktights (family Pulicidae) and alakurts (family Vermipsyllidae), which remain attached to the host for long periods, the mouthparts are elongate and barbed and also function as host-attachment devices.

Internally, the alimentary tract of fleas consists of an anterior pharynx that leads to the elongate esophagus and then to the proventriculus at the junction of the foregut and

FIGURE 10.4 Eggs (ova) and dried blood-rich feces ("flea dirt") excreted and provisioned as larval food by adults of the cat flea (*Ctenocephalides felis*). *Photograph by Nathan D. Burkett-Cadena.*

midgut. The **proventriculus** is armed with rows of spines that can be drawn together to prevent regurgitation of a blood meal from the midgut. The midgut expands to accommodate large blood meals but it lacks distensible diverticula or caeca. Many fleas imbibe larger volumes of blood than their midgut can accommodate and must void blood-rich feces during or soon after feeding. Some pathogens, such as *Rickettsia typhi*, the causative agent of murine typhus, are typically voided in these feces. Four excretory Malpighian tubules radiate from the junction of the midgut with the hindgut.

Male flea genitalia are morphologically complex (Fig. 10.3). The major structures are the paired claspers, which are used to secure the female during mating, the often highly specialized aedeagus, and the penis rods, which are partly inserted into the female genital opening during mating. The major components of the female genitalia are the vagina, the spermathecal duct, and the spermatheca; sperm are stored in the spermatheca after mating. During copulation, the male pushes his body beneath the female and grasps her from below with his claspers and with a series of sucker-like discs on his upraised antennae.

Flea eggs are small (0.1−0.5 mm), ovoid and pearly white (Fig. 10.4). Flea larvae are elongate, legless, and eyeless, with numerous stout body setae, especially on their abdominal segments. They possess a sclerotized head capsule armed with robust chewing mandibles (Fig. 10.5) and a pair of mandibular silk glands that produce silk for constructing a cocoon. Most flea larvae are small, worm-like, and highly active, with voracious appetites. Although few flea larvae can be identified to species, most can be assigned to the family level based on the arrangement of head papillae (small finger-like projections), setae, and sense organs.

Flea pupae are exarate (i.e., have externally projecting appendages) (Fig. 10.6) and are typically surrounded by a

FIGURE 10.5 Larvae of the cat flea (*Ctenocephalides felis*). *Photograph by Nathan D. Burkett-Cadena.*

FIGURE 10.6 Pupae of the cat flea (*Ctenocephalides felis*). *Photograph by Nathan D. Burkett-Cadena.*

loose silken cocoon that is secreted by the last larval instar. Because of the sticky nature of this silk, debris from the substrate often adheres to the cocoon and helps to camouflage it (Fig. 10.7). Many adult fleas possess an anterior frontal tubercle on the head that aids them in tearing free from the cocoon during emergence; after its use in this manner, the frontal tubercle breaks off in some species.

LIFE HISTORY

Fleas are holometabolous insects with an egg, larval (typically consisting of three instars), and pupal stage (Figs. 10.4–10.7). Gravid females of most flea species that have been studied can produce hundreds of eggs during their lifetime. Eggs typically hatch in about 5 days. Autogeny, or laying fertile eggs before ingestion of a blood meal, is not known to occur in fleas. The eggs are sticky and may adhere briefly to the host pelage; however, they usually drop into the host nest or bedding material where they hatch a few days later. Some fleas oviposit directly on leaves or debris in host nests such as females of *Stenoponia tripectinata*, a Palearctic rodent flea.

FIGURE 10.7 Cocoons of the cat flea (*Ctenocephalides felis*) covered with debris from substrate. *Photograph by Nathan D. Burkett-Cadena.*

Most flea larvae feed on organic matter in the nest or bedding materials of their hosts. Adult cat fleas, dog fleas, European rabbit fleas, and representatives of several other species void blood-rich fecal pellets (Fig. 10.4) often called **flea dirt** during feeding, which in turn provide a nutritious food source for the larvae. Larvae of the northern rat flea aggressively prod adult fleas until they excrete blood-rich feces, which the larvae then ingest. Some flea larvae supplement their diet by feeding on other small arthropods in the host nest; cannibalism among flea larvae appears to be common.

The pupal stage usually lasts 1–2 weeks but is influenced by ambient temperature and host availability. Eclosed adult fleas of several species can remain within the cocoon as preemergent adults until suitable host or environmental cues stimulate their emergence. Preemergent adult cat fleas can remain quiescent inside cocoons for four or five months to avoid desiccation or other environmental extremes that would kill free-living fleas.

Many fleas, including most of medical or veterinary importance, undergo continuous generations under favorable conditions. The cat flea is a good example. Indoors, the generation time for this flea is usually about one month but can be as short as 20 days. Other fleas, such as alakurts associated with migrating ungulates in Asia and species parasitic on migrating birds, are more likely to pass through just one generation per year in synchrony with host availability. Some fleas, especially in temperate regions, may undergo four or five generations each summer but less or none during the winter. Host availability clearly affects the number of generations in many fleas. Longevity of fleas in the absence of available hosts is greater at low temperatures and high humidity such as during winter in temperate regions. Under optimal conditions, adult fleas of certain species may survive away from the host for more than a year.

Specialized or unusual life cycles have evolved in several fleas, including some species of medical or

veterinary significance. Females of the genera *Tunga* and *Neotunga*, for example, burrow into host dermal tissue, where they undergo a dramatic size increase (up to 100-fold) accompanied by extensive morphological degeneration. This type of growth, called **neosomy**, involves major integumental chitin synthesis during the adult stage. The genital opening of the female protrudes through a pore in the host skin to facilitate mating with the free-living males. Fertilized eggs are flicked away from this opening by the embedded female. Because of the great size increase of neosomic females, they are able to produce many relatively large eggs. In some cases, this has led to a reduction in the number of larval instars from three to two; further modifications are exhibited by *Tunga monositus*, a parasite of New World rodents, in which neither larval instar feeds. The chigoe (*Tunga penetrans*) is an important parasite of humans and domestic animals that belongs to this group of fleas.

Adult females of a few fleas oviposit randomly into the environment, where the resulting larvae must search for organic matter to eat. Examples are vermipsyllid fleas in the genera *Vermipsylla* and *Dorcadia*, called **alakurts**, that feed on large ungulates and remain attached for several days. At the other end of the spectrum, females of *Uropsylla tasmanica* cement their eggs to the fur of their Australian hosts (dasyurid marsupials) and the larvae burrow into host skin, where they are subdermal parasites. Mature *U. tasmanica* larvae drop to the ground, where they spin a cocoon and pupate in a manner typical of most other fleas. Larvae of the North American hare-infesting *Euhoploplsyllus glacialis*, are ectoparasitic, and those of some other fleas feed on host carcasses or even on the superficial tissues of moribund hosts.

BEHAVIOR AND ECOLOGY

Fleas have evolved a variety of specialized behaviors and ecologies to locate and exploit their hosts. Host-finding behavior is extremely important for adult ectoparasites such as fleas, in which the immature stages typically occur off the host. Important stimuli used by fleas for host location include host body warmth, air movements, substrate vibrations, sudden changes in light intensity, and odors of potential hosts or their products (e.g., carbon dioxide, urine). The sensilium, antennae, and eyes are important organs used by fleas to detect potential hosts. In cases in which adult fleas emerge from their cocoons in close association with their host, locating a food source is not difficult. However, fleas of other groups of hosts such as ungulates or migrating birds typically must employ more elaborate strategies for this purpose. These include jumping toward dark or moving objects and moving toward warmth and CO_2 sources.

Some fleas are stimulated to emerge from their pupal case and cocoon by mechanical compression and vibrational stimuli, which often indicate the presence of a potential host. This response is especially noticeable in flea-infested human premises that have been vacated temporarily for weeks or months. When humans or pets return to the premises, these stimuli are largely responsible for synchronized emergences of adult cat and dog fleas from their cocoons.

Although some fleas spend much of their adult lives in the host pelage, most species visit the host principally to feed. In fact, some nidicolous (nest-associated) fleas (e.g., *Conorhinopsylla*, *Megarthroglossus*, and *Wenzella* spp.) spend little time on the active host, living instead in crevices in the nest and feeding when the host is asleep. Nidicolous habits have evolved in several families of fleas.

Once a flea locates a host, feeding is initiated by cues such as body warmth, skin secretions, and host odors. Sensory structures on the maxillary and labial palps aid in selecting a feeding site. The labium and labial palps then guide the stylet-like mouthparts into the host skin. Most fleas are capillary feeders; when the tip of the stylet bundle pierces a capillary, feeding is facilitated by contraction of powerful cibarial and pharyngeal muscles. Some fleas have mutualistic microorganisms in their midgut that aid in digestion of the blood meal.

Mating behavior in most fleas that have been studied follows a distinct sequence of events. When the male and female approach one another, the male touches the female with his maxillary palps and his antennae become erect. The male then moves behind the female, lowers his head, and pushes his body beneath hers while grasping her with his antennae using sucker-like discs along the inner antennal surfaces. Next, the male raises the apex of his abdomen, partially secures the female with his claspers, and extrudes his penis rods and/or aedeagus to initiate copulation. Sperm deposited into the female are stored in her spermatheca until her eggs are ready for fertilization.

Locomotory behavior in adult fleas usually involves walking or running on the substrate or through host pelage. However, jumping is the mode of locomotion for which fleas are best known; this provides both an important means of escape and a way to reach hosts. Fleas jump using a modification of the flight mechanism of their winged ancestors. In addition to using muscles derived from subalar and basalar flight muscles, they have retained the wing-hinge ligaments that have been displaced midlaterally owing to lateral compression of the flea body. The jump is not propelled by direct muscle action, but rather by the sudden expansion of discrete pads of a highly elastic protein called **resilin** in the pleural arch. This remarkable protein can store and release energy more efficiently than any synthetic rubber and more quickly than any muscle

tissue. The properties of resilin are unaffected by temperature, enabling fleas to jump even in subfreezing conditions.

Before jumping, the flea typically crouches, compresses its resilin pads, and keeps them compressed using one or more catch mechanisms. At "takeoff" the tergo-trochanteral depressor muscles relax to release the catch, allowing the resilin pads to expand and rapidly transfer energy to the hind legs (Fig. 2.4B). This results in an acceleration of about 200 G, catapulting fleas of some species more than 30 cm in about 0.02 s. While airborne, the flea somersaults, holding its middle or hind legs aloft to use as grappling hooks for snagging a host or the substrate. After landing, the muscles are rapidly readjusted in preparation for another jump. By repeating this action, the Oriental rat flea can make up to 600 jumps per hour for 72 h without rest.

Nest-associated fleas typically have reduced jumping abilities because they have less resilin in the pleural arch and have undergone secondary atrophy of jumping muscles. This appears to be adaptive in ensuring that these fleas do not leap out of a nest into an unfavorable environment.

Flea populations may be naturally regulated in several ways. Hosts are often efficient groomers and are able to reduce flea populations significantly on their bodies. Cats, for example, have been shown to remove up to 18% of their fleas within 24 h. Natural predators such as certain mesostigmatan mites, pseudoscorpions, beetles, ants, and other arthropods feed on fleas, especially the immature stages in host nests, thereby decreasing their numbers. Various parasites also contribute to flea mortality. These include the plague bacillus, *Yersinia pestis*, the protozoan *Nosema pulicis*, the fungus *Beauveria bassiana*, the nematode *Steinernema carpocapsae*, and parasitoids such as the pteromalid wasp *Baraimlia fuscipes*.

Environmental factors are often important in determining the abundance of fleas in different habitats or geographical regions. These factors are typically related to climate, weather, or soil conditions such as relative humidity, temperature, and soil moisture content. Favorable environmental conditions such as host abundance and availability, plentiful food for larvae, high relative humidity, and mild temperatures promote high populations of many flea species. Because the immature stages typically occupy niches different from those of adult fleas, the ecological requirements of one or more of the immature stages, rather than of the adult, may be limiting factors that do not permit a species to become established or abundant under certain conditions.

Host hormones can have an important role in synchronizing the development of fleas with that of their hosts. The life cycles of the rabbit fleas *Spilopsyllus cuniculi* and *Cediopsylla simplex*, for example, are mediated by host hormones imbibed with the host blood. These fleas can reproduce only after feeding on a pregnant doe. In this way, the emergence of adult fleas is synchronized with that of a litter of rabbits. Reproductive hormones (corticosteroids and estrogens) in the blood of the pregnant doe stimulate maturation of the ovaries and oocytes in feeding female fleas and testicular development in males. The adult fleas are ready to mate when the rabbit litter is born. Flea mating and oviposition occur after they have transferred onto the newborn young. The resulting flea larvae feed on organic matter in the nest debris. The next generation of adult fleas appears 15–45 days later, in time to infest the host littermates before they disperse from the burrow.

FLEAS OF MEDICAL–VETERINARY IMPORTANCE

Human Flea (*Pulex irritans*)

This flea (Fig. 10.8) will feed on humans and is capable of transmitting pathogens of medical importance. However, it more commonly parasitizes carnivores and sometimes pigs in most parts of the world. Although *Pulex irritans* is currently an infrequent parasite of humans in developed countries, this has not always been the case. *Pulex irritans* has a patchy but cosmopolitan distribution. Adults lack both genal and pronotal ctenidia (Fig. 10.8). *Pulex simulans* is a closely related species that parasitizes large mammals, including wild canids and domestic dogs, and sometimes people, in the Americas. Older records (before 1958) from this region are unreliable and could refer to either *P. irritans* or *P. simulans*.

FIGURE 10.8 Human flea, *Pulex irritans*; stacked image of cleared male. *Original image by Lorenza Beati and Lance A. Durden.*

Cat Flea (*Ctenocephalides felis*)

The cat flea (Fig. 10.9) occurs worldwide and is the most important flea pest of humans and many domestic animals. It is primarily a nuisance because it feeds not only on domestic and feral cats but also on humans, domestic dogs, and several livestock species. It also parasitizes wild mammals such as opossums and raccoons. This ectoparasite is the most common flea on dogs and cats in most parts of the world. Some strains of the cat flea appear to have adapted to ungulates such as horses or goats. Cases of severe anemia associated with huge numbers of cat flea bites have been recorded for these and other domestic animals.

Female cat fleas typically produce larger numbers of fertile eggs if they take their blood meals from cats rather than other host species. Under optimal conditions, a female cat flea can lay about 25 eggs per day for a month, contributing to very high densities of fleas in a relatively short time. Adult cat fleas have well-developed genal and pronotal ctenidia (Fig. 10.9) and can be distinguished from the dog flea (*Ctenocephalides canis*) by the longer head and longer first spine in the genal comb in *Ctenocephalides felis*.

Dog Flea (*Ctenocephalides canis*)

This flea (Fig. 10.10) is less common on domestic dogs in most parts of the world than it was in previous decades. Instead, the cat flea has become the most common flea on domestic dogs in most regions. No satisfactory explanation for this change has been documented; perhaps cat fleas can outcompete dog fleas under stress from modern pesticide applications. Nevertheless, dog fleas persist worldwide and remain the predominant fleas on dogs in some countries and regions. Dog fleas also parasitize wild canids such as foxes, coyotes, and wolves, on which they can be relatively common.

FIGURE 10.9 Cat flea, *Ctenocephalides felis*; stacked image of cleared male. *Original image by Lorenza Beati and Lance A. Durden.*

FIGURE 10.10 Dog flea, *Ctenocephalides canis*; stacked image of cleared male. *Original image by Lorenza Beati and Lance A. Durden.*

Oriental Rat Flea (*Xenopsylla cheopis*)

This flea (Fig. 10.11) is the principal vector of the causative agents of plague and murine typhus in many tropical and subtropical parts of the world. Some other species of *Xenopsylla* also are vectors of plague bacilli. Although it is most common on domestic rats, it also will feed on humans, dogs, cats, the house mouse, chickens, and other hosts, especially if rats become scarce. Like the human flea, adults of *Xenopsylla cheopis* lack both a pronotal and a genal ctenidium (Fig. 10.11), but the head and eyes are smaller in proportion to the rest of the body in *X. cheopis*.

FIGURE 10.11 Oriental rat flea, *Xenopsylla cheopis*; stacked image of cleared male. *Original image by Lorenza Beati and Lance A. Durden.*

European Rabbit Flea (*Spilopsyllus cuniculi*)

Originating in Europe, this flea has accompanied its host, the European rabbit, as it has been introduced to other parts of the world inadvertently, for food, or as a laboratory animal. It is an example of a sedentary flea; adults attach to the host for long periods using their elongate mouthparts to anchor themselves in host skin and feed. This flea typically attaches to the ears of rabbits, where a rich peripheral blood supply provides easily accessible blood meals. Adults have a genal ctenidium with a row of five blunt spines oriented almost vertically on the head and a well-developed pronotal ctenidium. *Cediopsylla simplex* (Fig. 10.1) is a morphologically similar flea that parasitizes North American rabbits.

Sticktight Flea (*Echidnophaga gallinacea*)

As indicated by its name, this is another sedentary flea. It is distributed globally wherever chickens have been introduced as domestic animals. This flea (Fig. 10.12) usually attaches semipermanently around the head and especially the wattle of chickens (Fig. 10.13). Many additional hosts are also parasitized by *E. gallinacea*, including other domestic birds (e.g., turkeys, quail), wild birds, peridomestic rats, dogs, cats, and occasionally humans. Adults of this small flea are easily recognized by their sharply angled squarish head and the absence of both pronotal and genal ctenidia (Fig. 10.12).

FIGURE 10.13 Sticktight fleas (*Echidnophaga gallinacea*) attached to the head of a chicken. *Photograph by Amy C. Murillo.*

Chigoe (*Tunga penetrans*)

This flea (Fig. 10.14), also called the **jigger** or **sand flea**, has major medical and veterinary significance because females burrow into tissues of humans and some domestic animals. In addition to being very small (c. 1 mm in length), free-living adult chigoes lack pronotal and genal ctenidia and have a sharply angled head (Fig. 10.14). This

FIGURE 10.12 Sticktight flea, *Echidnophaga gallinacea*; stacked image of cleared female. *Original image by Lorenza Beati and Lance A. Durden.*

FIGURE 10.14 Chigoe flea, *Tunga penetrans*; stacked image of cleared male. *Original image by Lorenza Beati and Lance A. Durden.*

flea is widely distributed in tropical and subtropical regions. Native to South America, it is now widespread in many tropical regions and is an emerging parasite in parts of sub-Saharan Africa, especially east Africa, where scores of female chigoes can embed in a single person, sometimes leading to secondary bacterial infections, gangrene, amputations (mainly of toes and feet), and (rarely) death. It is also an emerging parasite in parts of Brazil. The life cycle of the immature stages and the male of *T. penetrans* does not deviate significantly from that of most fleas. Initially, the adult female is free-living but later it invades host skin. Once embedded, she begins to swell by imbibing host fluids, often expanding about 80-fold to reach the size of a pea after 8−10 days (Figs. 10.15 and 10.16). She maintains an opening to the exterior through which she respires, mates with a free-living male, and expels her eggs. The male possesses one of the longest intromittent organs relative to body size in the animal kingdom. Eggs usually are flicked onto sandy soils, including coastal beaches (hence the term sand flea), frequented by potential hosts where the larvae complete their development. There are only two larval instars. Development from egg to adult usually takes 4−6 weeks, but sometimes only three weeks under optimal conditions.

An additional species, *Tunga trimamillata*, which causes tungiasis in humans and domestic animals in Ecuador and Peru, has been described (Pampiglione et al., 2009).

Northern Rat Flea (*Nosopsyllus fasciatus*)

This is a common flea of domestic rats, especially in temperate and northern regions of the world. Although it will bite humans and can transmit several zoonotic pathogens such as the agents of plague and murine typhus, it is far less important in this respect than the Oriental rat flea. Occasionally, it parasitizes other rodents or domestic mammals. Adults possess a well-developed pronotal comb but lack a genal comb (Fig. 10.17).

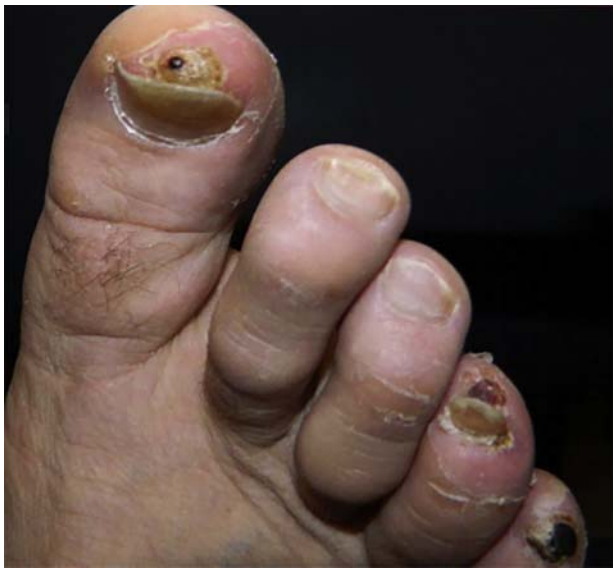


FIGURE 10.16 Tungiasis; female *Tunga penetrans* embedded in human big toe and lesions resulting from attempted surgical removal in two other toes. *From Richardson and Mangili, 2016.*

European Chicken Flea (Hen Flea in Europe) (*Ceratophyllus gallinae*)

This ectoparasite of feral and domestic birds, especially chickens, originated in Europe but has spread with poultry operations throughout much of the world, especially in cooler regions. Because *Ceratophyllus gallinae* can feed on

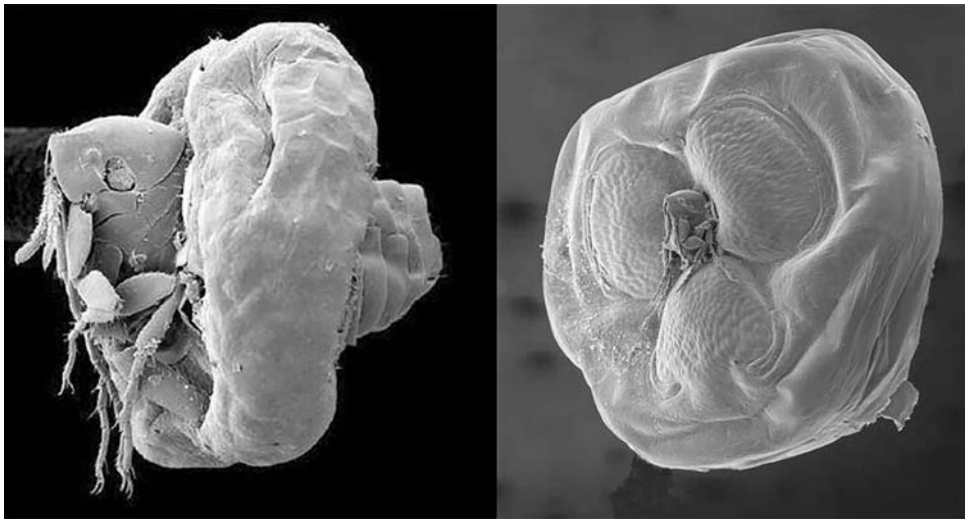


FIGURE 10.15 Chigoe (*Tunga penetrans*); neosomic adult females three and eight days, respectively, after penetration of host tissues. Images not to scale. *From Eisele et al., 2003.*

FIGURE 10.17 Northern rat flea, *Nosopsyllus fasciatus*; stacked image of cleared female. *Original image by Lorenza Beati and Lance A. Durden.*

FIGURE 10.18 European chicken flea (hen flea), *Ceratophyllus gallinae*; stacked image of cleared female. *Original image by Lorenza Beati and Lance A. Durden.*

so many bird species, it is often difficult to eradicate completely. In contrast to *E. gallinacea*, this flea is highly mobile on the host and can be especially common in host nesting material. Adult *Ceratophyllus* fleas have a distinct pronotal ctenidium but lack a genal ctenidium (Fig. 10.18).

European Mouse Flea (*Leptopsylla segnis*)

This cosmopolitan flea typically parasitizes the house mouse (*Mus musculus*), including mice in laboratory colonies. Rarely, large populations of *Leptopsylla segnis* cause host anemia or other problems in mouse rearing facilities. Adults of this flea possess a well-developed pronotal ctenidium and a vertical genal ctenidium consisting of four bluntly rounded spines (Fig. 10.19).

FIGURE 10.19 European mouse flea, *Leptopsylla segnis*; stacked image of cleared male. *Original image by Lorenza Beati and Lance A. Durden.*

PUBLIC HEALTH IMPORTANCE

Fleas can cause various public health threats. Many species are annoying biters that can cause considerable discomfort, sometimes leading to secondary infections of bite sites. The bites of some species can cause **flea allergy dermatitis** (FAD). Allergic responses also can result from contact with, or inhalation of, flea products (e.g., larval exuviae). Females of the chigoe invade human skin tissue, especially of the feet and toes, and cause painful lesions that are prone to secondary infections. Other fleas are intermediate hosts of tapeworms that can parasitize humans. Fleas also serve as vectors of the causative agents of several important zoonotic diseases such as murine typhus and plague (Table 10.2).

Flea bites (Fig. 10.20) can cause intense irritation for several days. Bites are characterized by a tiny purplish spot, or **purpura pulicosa**, surrounded by slightly swollen skin called **roseola pulicosa**. The vast majority of flea bites experienced by humans result from the cat flea. This flea is an unrelenting biter that generally attacks humans on the ankles (Fig. 10.20), although other parts of the body may be affected. Women tend to be more commonly bitten than men, suggesting an attraction to female hormones. In addition to the annoyance it causes, *C. felis* is a vector of the causative agents of murine typhus, cat flea rickettsiosis, and cat scratch disease. The cat flea is discussed in more detail with respect to its veterinary importance.

The human flea also is an annoying biter of people in various parts of the world. The closely related *P. simulans* sometimes infests households and has been recorded to cause dermatitis in humans in western North America. Several other species of fleas may bite humans to the point

TABLE 10.2 Pathogens and Parasites Transmitted by Fleas

Disease Agent	Disease	Vector(s)	Host(s)	Geographical Area
Viruses:				
Myxoma virus	Myxomatosis	*Spilopsyllus cuniculi*	Rabbits	Europe, Australia
Parapoxvirus	Squirrel pox	Squirrel fleas	Squirrels	Britain
Bacteria:				
Bartonella henselae	Cat scratch disease	*Ctenocephalides felis*	Cats, humans	Widespread
Coxiella burnetii	Q fever	Several fleas	Mammals	Global
Francisella tularensis	Tularemia	Several fleas	Mammals	Global
Rickettsia felis	Cat flea rickettsiosis	*Ctenocephalides felis*	Cats, humans	Widespread
Rickettsia typhi	Murine typhus	*Xenopsylla, Ctenocephalides*	Mammals	Global
Rickettsia prowazekii	Sylvatic epidemic typhus	*Orchopeas howardi*	Flying squirrels, humans	North America
Yersinia pestis	Plague	Mainly *Xenopsylla*	Humans, rodents, cats	Widespread
Protozoa:				
Trypanosoma lewisi	Murine trypanosomiasis	*Nosopsyllus, Xenopsylla*	Rats	Global
Trypanosoma nabiasi	Rabbit trypanosomiasis	*Spilopsyllus cuniculi*	Rabbits	Global
Nematoda:				
Acanthocheilonema reconditum[a]	Canine filariasis	*Ctenocephalides*	Carnivores	Global
Cestoda:				
Dipylidium caninum[a]	Double-pored tapeworm	*Ctenocephalides*	Dogs, cats, humans	Global
Hymenolepis diminuta[a]	Rodent tapeworm	*Nosopsyllus, Xenopsylla*	Rodents, humans	Global
Hymenolepis nana[a]	Dwarf tapeworm	*Nosopsyllus, Xenopsylla*	Rodents	Global

[a]*Fleas are not vectors for these parasites but instead serve as intermediate hosts.*

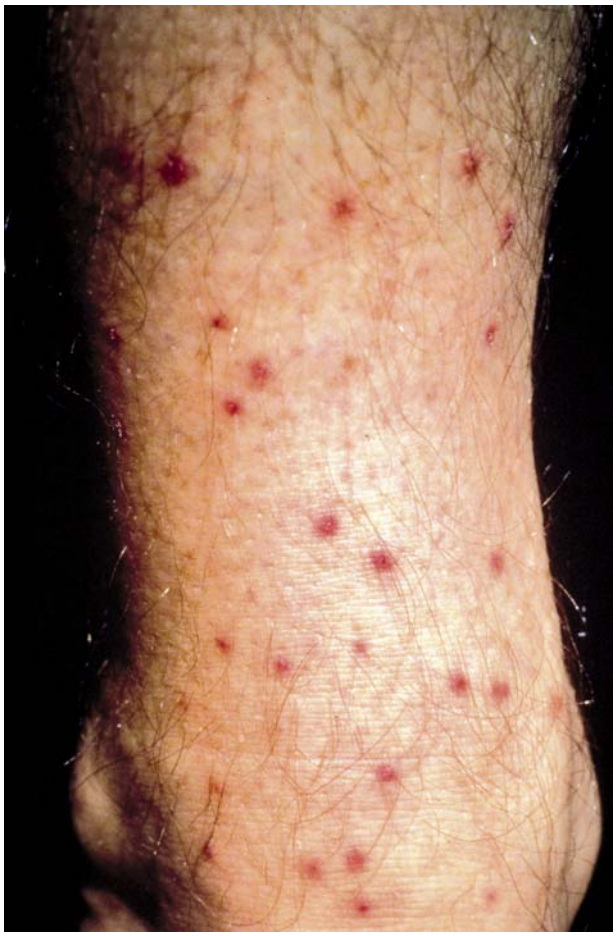


FIGURE 10.20 Multiple cat flea bites on a human ankle. *Photograph by Elton J. Hansens.*

of annoyance including the dog flea, the sticktight flea, the northern rat flea, and several species of *Xenopsylla*, including the Oriental rat flea (Table 10.1). Also, the squirrel flea, *Orchopeas howardi*, and some bird fleas belonging to the genus *Ceratophyllus*, including the European chicken flea (hen flea) (Fig. 10.18), occasionally bite humans.

Members of households and adjacent premises harboring pets or domestic rodents can be especially prone to flea bites. Cat fleas and dog fleas readily bite humans, especially if flea populations are large or if pets are removed temporarily. Rodents inhabiting households also can be a source of fleas that can bite humans. Fleas typically abandon dead hosts; if domestic rats die in wall voids, basements, or other poorly accessible structures, their fleas may seek human hosts. A similar situation can occur involving squirrel fleas when their hosts nest in attics or eaves of houses. Basements, garbage, and pet food supplies and feeding bowls also can attract scavenging mammals such as opossums, raccoons, and skunks in North America. These animals may leave behind cat fleas and other fleas that will bite humans. Flea bites represent occupational hazards on many farms, in barns, rabbit hutches, and poultry operations. The fleas in such cases may come directly from livestock or indirectly from domestic rodents attracted to food supplies.

Flea-Associated Allergies

FAD (flea allergy dermatitis, also sometimes called flea-bite dermatitis) typically occurs in persons who have become hypersensitive to flea saliva. In sensitized individuals, bite sites typically develop into papules, causing a form of papular urticaria, often with associated wheals, especially in children. In more serious cases, skin scaling, hardening, or discoloration can occur. In adults, usually the distal extremities (hands and feet) are involved, whereas in children the entire body may be affected. With time and repeated exposure to flea bites, hyposensitization may reduce the severity of the dermatitis without medical intervention. The administration of corticosteroids or desensitizing antigens can be helpful for some hypersensitive individuals.

People can also become sensitized to flea feces and particles of exoskeletons upon contacting or inhaling them in house dust. Adult fleas have been identified as the source of some of these allergens. Airborne larval exuviae also have been implicated as causes of asthmatic symptoms. Relief from these allergic responses may be achieved by administering a course of desensitizing antigens to the patient.

Plague

Plague is caused by infection with *Yersinia pestis*, a Gram-negative coccobacillus bacterium. The entire genome of *Y. pestis* was sequenced in 2001. The disease also is referred to as the **Black Death**, and in Francophone countries as *la peste*. The organism was first isolated in 1894, when Swiss bacteriologist **Alexandre Yersin** cultured it from patients at the Pasteur Institute in Hong Kong. Although plague originated in central Asian rodents such as gerbils, it is typically maintained in urban areas in peridomestic rodents, especially the black rat (*Rattus rattus*) and Norway (brown) rat (*Rattus norvegicus*). The pathogen is transmitted to these rodents by fleas, especially the Oriental rat flea and some other members of the genus *Xenopsylla*. The role of *X. cheopis* as a vector of *Y. pestis* was definitively demonstrated by French physician and bacteriologist **Paul-Louis Simond** during his plague studies in Pakistan (then part of India) in 1898.

Plague pandemics have had a significant impact on human civilization and have claimed more human lives than all wars ever fought. Three major pandemics have been documented. The first, sometimes called **Justinian's plague** (named for the Roman emperor Justinian), emerged

in AD 541 in north Africa and later spread throughout Mediterranean Europe, killing an estimated 40 million people during the 6th and 7th centuries.

The second pandemic, usually referred to as the **Black Death**, originated in central Asia in the 14th century and spread to Europe along developing trade routes between these two continents. In 1347, crew members of an Asian trading vessel that docked in Sicily, Italy were infected with a mysterious disease that was later identified as plague. Over the next five years, plague spread throughout most of Europe from this point of entry, with devastating consequences; by 1352, at least 25 million people had died in Europe alone. This pandemic endured for more than 200 years, with the disease appearing or reappearing in different regions of Europe. London experienced major epidemics in 1348 and 1665, with foci of the disease persisting in the city throughout this period. Ancient *Y. pestis* DNA has been detected in tooth pulp from human victims in several plague burial pits in Europe.

The third pandemic of plague, sometimes referred to as **modern plague**, spread across the globe in the late 1800s after emerging from a focus in China's Yunnan Province in 1855. From 1896 to 1948, this pandemic accounted for 12 million deaths in India alone. Several plague foci that initiated during the peak of this pandemic still persist. In the United States, for example, plague bacilli were introduced with infected ship-borne rats in 1898 in San Francisco and from there eventually spread to at least 14 western states and two Canadian provinces. Plague is still enzootic in native rodents and fleas in most of these areas of western North America. Worldwide, human plague cases have decreased dramatically since the widespread availability of antibiotics in the 1940s, combined with effective flea and reservoir host control programs. Accounts of plague include works by Duplaix (1988), Mee (1990), Poland et al. (1994), Madon et al. (1997), Dennis et al. (1999), Carniel and Hinnebusch (2004), Kelly (2004), Christakos et al. (2005), Gage and Kosoy (2005), Eisen et al. (2007, 2015), and Baegler et al. (2016).

Nucleotide sequencing of recombinant RNA in *Y. pestis* shows a correlation between the geographical distribution of genetic strains, or biovars, of *Y. pestis* and their spread during the three pandemics. There are three biovars of *Y. pestis*: biovar Antiqua occurs in Africa, biovar Medievalis mainly in central Asia, and biovar Orientalis in Europe, Asia, Africa, North America, and South America.

Today, plague occurs as fairly discrete foci in various parts of Asia, southern and northwestern Africa, South America, and western North America. Outbreaks have surfaced in Algeria, Brazil, the Democratic Republic of the Congo, Ecuador, India, Iran, Madagascar, Malawi, Mongolia, Peru, South Africa, Tanzania, Vietnam, and Zambia. Globally, nearly 19,000 human cases of plague were reported to the World Health Organization from a total of 20 countries in 1984−1994. Each year, 1,000−3,000 cases are reported, with most human plague deaths occurring in Africa. Since 2010, Madagascar and the Democratic Republic of Congo have reported more human plague cases than any other nations and multidrug resistant strains of *Y. pestis* have emerged in Madagascar. From 1970 to 1994, a total of 334 cases of indigenous plague were reported in the United States, whereas during 1988−2002, a total of 112 cases were reported. Peak years were 1983 and 1984, with 40 and 31 cases, respectively. About 80% of cases in the United States occurred in Arizona, New Mexico, or Colorado.

In addition to bites from infected fleas, plague infections can result from direct contact with moribund or dead mammals infected with *Y. pestis*, or rarely, from inanimate objects harboring the pathogen. In such cases, the pathogen typically enters the body through skin lesions. Inhalation infection also can occur from aerosolized *Y. pestis*.

Two ecological forms of plague are recognized: **urban plague** carried by domestic rats and their fleas in cities and towns, and **wild-rodent plague** (**sylvatic plague**, derived from the Latin *silva*, meaning trees, **campestral plague**, and **rural plague**) maintained enzootically in several species of mammals (mainly rodents) and their fleas in rural areas distant from human populations. Over 200 species of rodents and other mammals (e.g., certain carnivores) may serve as reservoir hosts of wild-rodent plague. In North America, ground squirrels, rock squirrels, chipmunks, and prairie dogs are particularly important, whereas in Asia, gerbils and susliks (ground squirrels) typically fill this role. Similarly, various gerbils and the peridomestic rat *Mastomys natalensis* are important reservoir hosts in parts of Africa. The short-tailed field mouse, *Zygodontomys brevicauda,* is important in some South American foci. Plague-infected tree squirrels have been found in some towns in the western United States.

In many plague-endemic regions, wild-rodent plague circulates enzootically in rodent and flea populations. Under certain conditions, the disease can become epizootic and spill over to peridomestic rats to trigger urban plague. Some populations of reservoir hosts are refractory to infection with *Y. pestis* and others are highly susceptible. This is reflected by large-scale die-offs in infected prairie dog (*Cynomys* spp.) towns in North America. Intermediate stages of susceptibility to plague exist between these two extremes in other reservoir populations. In most regions where plague exists, there are distinctly different species of enzootic and epizootic rodent reservoir hosts. Most carnivores, especially felids, are susceptible to infection with *Y. pestis*. The disease is often severe in domestic cats, which can serve as a source of infected fleas to households. Bites, scratches, or inhalation of infectious aerosols from infected cats also can disseminate *Y. pestis*. Human plague cases acquired from domestic cats have increased in the United States.

Although the Oriental rat flea is an important vector of *Y. pestis*, at least 125 species of fleas are capable of transmitting the pathogen, especially under laboratory conditions. In North America, several flea species can transmit the plague bacterium to native rodents; *Oropsylla montana* (Fig. 10.21), and to a lesser degree, *Hoplopsyllus anomalus*, are important among these. In Russia and northern Asia, fleas belonging to the genera *Citellophilus*, *Neopsylla*, and *Ctenophthalmus* are significant enzootic vectors within rodent communities. In addition, the widespread fleas *Pulex irritans* and *Nosopsyllus fasciatus* are capable of transmitting plague bacilli. Because *X. cheopis* survives poorly in cool climates, historical reevaluations suggest that, contrary to former dogma, *P. irritans* rather than *X. cheopis* may have been a principal flea vector of *Y. pestis* in the historical plague epidemics of northern Europe. The human body louse, *Pediculus humanus humanus*, has also been implicated as a likely historical vector of *Y. pestis*.

A susceptible flea typically becomes infected after imbibing plague bacilli in its blood meal from an infected host. The bacterium invades the flea midgut where under suitable conditions, it multiplies rapidly, often culminating in complete blockage of the gut anterior to the proventricular spines. This proventricular blockage (Fig. 10.22) results from clumping of the bacteria several days after ingestion. Although some gut blockages may clear spontaneously, persistent blockage of the flea gut is central to efficient transmission of *Y. pestis*. Blocked fleas are incapable of ingesting a blood meal from a host. Feeding attempts by these fleas result in the drawing of blood into the esophagus followed by regurgitation of the blood meal into the host. This regurgitation is caused by the elastic recoil action of the esophagus when resistance from the proventricular blockage is reached. Infection results when plague bacilli are regurgitated with the blood meal into the host.

FIGURE 10.21 *Oropsylla montana*; stacked image of cleared female. *Original image by Lorenza Beati and Lance A. Durden.*

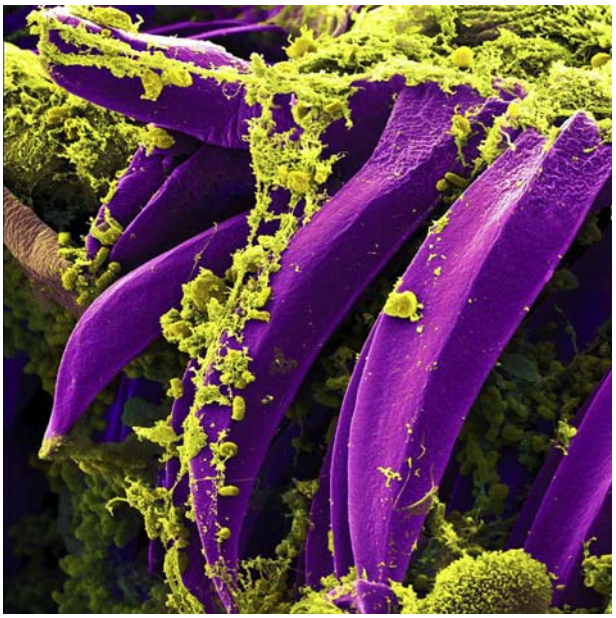


FIGURE 10.22 Blockage of proventricular spines in gut of Oriental rat flea (*Xenopsylla cheopis*) by a mass of *Yersinia pestis*, the causative agent of plague; colorized image. *Courtesy, U.S. Public Health Service, Public Health Image Library.*

Fleas with gut blockages are hungry and make repeated, aggressive feeding attempts. This can result in the infection of several different hosts and amplification of an epidemic. Unless the gut blockage clears, the infected flea ultimately succumbs to starvation, dehydration, or toxicity from bacterial metabolites, itself becoming a victim of plague. Although *Y. pestis* transmission by fleas is facilitated by proventricular blockage, some fleas can also transmit before or without blockage formation in a process called early-phase transmission (Eisen et al., 2015). If the ambient temperature exceeds 28°C, plague-infected fleas often can clear their guts of blockages and the disease typically does not develop to epidemic proportions. As with most other pathogens transmitted by fleas, plague bacilli do not pass through the gut wall of infected fleas to invade the hemocoel, salivary glands, or other organs.

There are three recognized clinical forms of plague infection: bubonic, septicemic, and pneumonic. The most common of these is **bubonic plague**, which usually results from the bites of infected fleas but can be caused by handling infectious mammal carcasses. A reddened lesion may develop at the site of an infectious flea bite. This type of plague is characterized by grossly enlarged, tender, peripheral lymph nodes called **buboes** (singular, bubo). They usually occur in the axillary or inguinal region (Fig. 10.23) and are typically teeming with plague bacilli.

In **septicemic plague**, the pathogen initially bypasses or overwhelms the peripheral lymph nodes and invades deeper recesses of the body. Although internal buboes may

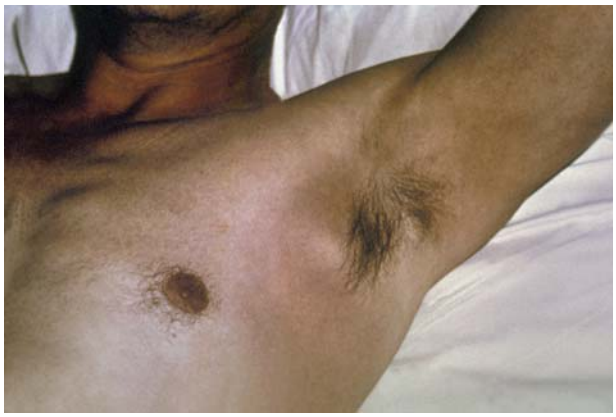


FIGURE 10.23 Plague patient with enlarged axillary lymph node, or bubo, characteristic of bubonic plague. *Courtesy, U.S. Public Health Service, Public Health Image Library.*

develop, they are not easily detected. Instead, the bloodstream is invaded rapidly by bacteria and capillary walls start to leak, often turning the skin black. The absence of external buboes to aid diagnosis, coupled with swift invasion of the blood, make this form of plague especially severe; many patients succumb to fatal septicemia.

The most life-threatening form of the disease is **pneumonic plague** (**plague pneumonia**), in which patients have a lung infection of *Y. pestis* and can cough or sneeze infectious, aerosolized bacteria into the environment. Inhalation of *Y. pestis* by susceptible individuals results in the pulmonary form of this disease. Also, bubonic or septicemic plague can progress to pneumonic plague. Without prompt and aggressive medical attention, pneumonic plague is invariably fatal; some untreated patients die within a day of inhaling the pathogen. The severe pathogenicity of *Y. pestis* is largely caused by endotoxins and exotoxins released by the dividing bacilli.

Flea-transmitted infection typically results in classic bubonic plague in which buboes develop after an incubation period of 2−6 days. Accompanying symptoms are severe headache, fever, and shaking chills. Without treatment, most patients deteriorate rapidly with a typical case fatality rate of 50%−60%. Septicemic plague is a particularly dangerous form of the disease because the incubation time is only 2−5 days and external buboes are often absent. Pneumonic plague has a very short incubation period (1−3 days) and may spread rapidly from one victim to another. Overwhelming pneumonia characterized by coughing, bloody sputum, chills, and fever usually results in death within three days unless specific medication is rapidly administered. Pneumonic plague spreads directly, and usually rapidly, from person to person without involving flea vectors. In some cases, humans have contracted pneumonic plague after inhaling aerosolized bacilli expelled by infected household cats.

Accurate diagnosis is important in identifying plague-infected patients, reservoir hosts, and vectors. Direct fluorescent antibody staining of tissues, testing cultures by specific bacteriophage lysis, and running serum samples on test strips for *Y. pestis* antibodies by passive hemagglutination and inhibition are currently used screening techniques for the bacterium. Pools of fleas can be tested by triturating samples in buffer and then inoculating the suspension into susceptible laboratory mice followed by euthanasia and necropsy of sick mice, culturing their liver and spleen tissues on sheep blood agar, and testing the resulting bacterial colonies for *Y. pestis* by specific bacteriophage lysis (Lowell et al., 2009).

Treatment of plague patients usually involves immediate hospitalization, isolation, and intravenous administration of broad-spectrum antibiotics. Formalin-inactivated plague vaccines are available. However, they are not totally effective. None are protective against pneumonic plague in humans and most must be administered in multiple doses, or at regular intervals, to ensure protection.

Efforts to control plague typically involve removing wild-rodent reservoir hosts and/or their fleas. In areas of potential plague activity, samples of rodent blood and tissues often are collected to monitor plague in reservoir host populations. Fleas also can be collected and screened for *Y. pestis*. If samples are positive, rodent and flea control measures should be considered.

Plague is an infectious disease of major concern as a potential bioweapon. Historically, plague was used as a bioweapon when conquering Mongol soldiers hurled plague cadavers into the besieged Crimean city of Caffa in 1346. Furthermore, fleeing infected inhabitants from Caffa may have had a role in introducing the Black Death into Europe in 1347 (Wheelis, 2002). In addition, "flea bombs" (cannisters containing plague-infected fleas and dropped from planes) were used as a bioweapon as recently as the 1930s and 1940s.

Murine Typhus

Murine typhus, also known as **endemic typhus**, Mexican typhus, shop typhus, rat typhus, urban typhus, and **flea-borne typhus**, is caused by the rickettsial organism *Rickettsia typhi* (formerly *Rickettsia mooseri*). Although this zoonosis is typically maintained in peridomestic rats by flea transmission, humans can also be infected. Murine typhus is one of the most prevalent rickettsial diseases of humans, but it is underdiagnosed and its importance is generally unappreciated. *Rickettsia typhi* is a small, obligate, intracellular bacterium that can cause a mild febrile infection in humans. It usually is transmitted via infected flea feces. When the bite site of an infected flea is scratched, rickettsiae from flea feces excreted next to the bite site gain access to the host through abraded skin. Under

experimental conditions, some fleas also can transmit this pathogen via bite. Reviews on the ecology and epidemiology of murine typhus were provided by Traub et al. (1978), Azad (1990), Rawlings and Clark (1994), Azad et al. (1997), Goddard (1998), Erickson et al. (2017), and Murray et al. (2017).

The geographical distribution of murine typhus is almost global. Although it occurs on all continents except Antarctica, its importance as a human pathogen has diminished. Significant foci persist, however, especially in Indonesia, China, Thailand, North Africa, and Central America. In the United States, the annual number of human cases has decreased from more than 5,000 in 1945 and 1946 to 20−80 per year from 1958 to the present. This zoonosis was formerly widespread throughout the southern and southwestern United States. Currently in the United States, it is principally recorded in Texas (Erickson et al., 2017; Murray et al., 2017); homeless persons in Houston, Texas, also have been recorded to be seropositive (Reeves et al., 2008). Several cases are usually reported annually from California and Hawaii, as well.

Murine typhus is maintained primarily in a cycle that involves commensal rats in the genus *Rattus* and their ectoparasites, especially fleas of the genus *Xenopsylla*. Humans are typically infected when feeding fleas void infectious feces on their skin. The black rat (*R. rattus*) and Norway rat (*R. norvegicus*) are the principal reservoir hosts of *R. typhi*. Infections also have been recorded in many other mammals including other peridomestic rats (*Rattus* spp.) worldwide, bandicoot rats (*Bandicota* spp.) on the Indian subcontinent, house mice (*M. musculus*) worldwide, the oldfield mouse (*Peromyscus polionotus*) in the southern United States, the giant pouched rat (*Cricetomys gambianus*) in Africa, the house shrew (*Suncus murinus*) in the Old World, domestic cats worldwide, and the Virginia opossum (*Didelphis virginiana*) in North America. New World strains of *R. typhi* are much less virulent (c. 2% case fatality rate in humans) than some Old World strains (up to 70% case fatality rate).

Peridomestic rats almost invariably are the most important reservoirs and amplifying hosts of *R. typhi*. Infection in rats is not fatal; instead, they display a persistent rickettsemia. This is important in extending the period during which ectoparasites, especially fleas, can feed on infective hosts and imbibe the pathogen. Virginia opossums can be reservoir hosts in some regions of the United States and have been associated with human cases.

At least 11 species of fleas belonging to nine different genera have been found to be infected with *R. typhi* in nature. *Xenopsylla cheopis* is the most important vector. Other vectors are *Xenopsylla astia*, *Xenopsylla bantorum*, *Xenopsylla brasiliensis*, *C. felis*, *P. irritans*, *L. segnis*, and *N. fasciatus*. Except for *C. felis* and *P. irritans*, all of these fleas are common ectoparasites of commensal rodents in various parts of the world. Human cases of murine typhus usually coincide with population peaks of *X. cheopis* on rats. Infection prevalences of field-collected *X. cheopis* in hyperendemic regions typically are 50%−70%.

Infection of a flea occurs when rickettsiae are ingested while the flea is feeding on a host with *R. typhi* circulating in its blood. The ingested rickettsiae then invade the midgut epithelial cells of the flea and start to replicate. The infection spreads rapidly until most or all of the midgut cells are infected after 7−10 days. Ultimately, infectious rickettsiae are released from these cells and liberated into the gut lumen, and then are excreted in the feces. *Xenopsylla cheopis* fleas are typically infective about 10 days after an infectious blood meal and infected fleas can transmit the pathogen for at least another 40 days. Infected fleas survive with a persistent *R. typhi* infection and demonstrate no obvious pathological effects. This contrasts with the related pathogen *Rickettsia prowazekii*, which causes a fatal infection in its body louse vector. Because *X. cheopis* can maintain and transmit *R. typhi* transovarially, this flea may be both a reservoir and a vector of murine typhus rickettsiae.

Although modes of *R. typhi* transmission other than via infected flea feces are known, their significance in nature remains unclear. Because *X. cheopis* has been shown to transmit *R. typhi* by bite in the laboratory, other fleas also may be capable of transmitting *R. typhi* by bite. The possibility of aerosol transmission from infective flea feces has been suggested.

Rickettsia typhi has been detected in some ectoparasites other than fleas. Because most of these arthropods do not bite humans, their presumed role is in transmitting *R. typhi* enzootically among commensal rats. Ectoparasites in this category include the sucking lice *Hoplopleura pacifica* and *Polyplax spinulosa*, the mesostigmatan mites *Laelaps echidnina* and *Ornithonyssus bacoti*, and the chigger *Ascoshoengastia indica*. Although the human body louse (*P. humanus humanus*) is an experimental vector of *R. typhi*, apparently it is not involved in natural transmission cycles.

Clinical symptoms of murine typhus appear after an incubation period of 6−14 days and include a rash, high fever, prostration, delirium, and coma, especially in severe cases. In milder cases, patients may have low-grade fever and remain partially mobile. Although case fatality rates are usually low (<5%) in untreated patients, severe debilitation may last 2−3 months and most patients often cannot work for extended periods.

The diagnosis of human infection usually involves the demonstration of antibodies against *Rickettsia typhi* or isolation of the bacterium in a blood or tissue sample. *Rickettsia typhi* detection in fleas includes using an enzyme-linked immunosorbent assay and polymerase chain reaction (PCR) techniques. These techniques are also useful for

patient diagnosis and in screening potential reservoir hosts and vectors.

Treatment of patients presenting with murine typhus is with antibiotics such as doxycycline and tetracycline. Surveillance and control techniques involve monitoring mammals (especially rats) and fleas for infection and then initiating reservoir or and/or vector control measures as needed.

Other Flea-Borne Rickettsial Agents

In addition to the causative agent of murine typhus, several other rickettsial agents may be transmitted by fleas. One of these, *Rickettsia felis,* has been detected in cat fleas (and some other blood-feeding arthropods) in many parts of the world (Pérez-Osorio et al., 2008; Reif and Macaluso, 2009; Brown and Macaluso, 2016). Human infection with this agent is sometimes referred to as **cat flea rickettsiosis** (also **flea-borne spotted fever** and cat flea typhus). It has been shown to have caused infection in humans who were serologically positive for infection with *R. typhi*, the etiologic agent of murine typhus. Definitive demonstration of infection by either *R. felis* or *R. typhi* involves PCR amplification of specific nucleotide primers. Therefore, it is likely that some human infections serologically attributed to *R. typhi* are actually caused by *R. felis*. *Rickettsia felis* is transmitted transovarially in fleas, and fleas, rather than mammals, appear to be reservoirs for the organism. However, *R. felis* has been detected in some mammals including cats, dogs, Virginia opossums, and humans. Infected humans can present with significant symptoms including fever, headache, and rash. An eschar (a lesion with a necrotic, darkened central area) often develops at the site of the infectious flea bite. Clinical treatment is typically with oral doxycycline. Reif and Macaluso (2009) and Brown and Macaluso, (2016) provide further information on *R. felis* infections.

Another rickettsial agent, *Coxiella burnetii,* the agent of **Q fever**, can be transmitted by fleas and also by some other blood-feeding arthropods, infected mammalian tissues, by infective fomites (inanimate objects), or by aerosol. **Sylvatic epidemic typhus** (**sporadic epidemic typhus**) is a potentially serious disease that occasionally is diagnosed in humans in the United States. The agent of this disease is *Rickettsia prowazekii*, which causes classic epidemic typhus transmitted to humans by the body louse, *P. humanus humanus* (see Chapter 7). However, flying squirrels (*Glaucomys volans*) rather than humans are the reservoir hosts of sylvatic epidemic typhus. Flying-squirrel fleas, especially the widespread squirrel flea *Orchopeas howardi*, and lice, also harbor the causative rickettsiae. The exact mode of transmission to humans is unknown, but because ectoparasites of flying squirrels rarely feed on humans, it has been hypothesized that under certain conditions, infective rickettsiae in the feces of fleas and lice become aerosolized and may be inhaled by humans. Infected flying squirrels are not adversely affected. These squirrels sometimes are closely associated with humans through their predilection for constructing nests in attics or eaves of houses. Chapman et al. (2009) provide additional discussion of this rickettsial zoonosis. In addition to these rickettsiae, some fleas are known to harbor symbiotic rickettsiae.

Other Flea-Borne Pathogens

Table 10.2 lists other pathogens known to be transmitted by fleas. Most of these microorganisms principally occur in the flea gut rather than the salivary glands or other organs. It has been suggested that this is why fleas are inefficient vectors of viruses. However, it is now known that murine typhus rickettsiae can escape the flea gut and multiply in other organs. Transmission of these gut-localized pathogens therefore occurs by either regurgitation (anterior station) or defecation (posterior station). The apparent ease with which fleas can acquire and harbor a wide variety of infectious agents indicates why these insects have a major role in the maintenance and epidemiology of enzootic infections among rodents and other mammals. Many of these pathogens can produce disease in humans and domestic animals if these fleas, or bridge vectors, feed on these hosts.

Bacteria

In addition to flea-borne rickettsial organisms, the following bacterial agents that can cause diseases in humans have been reported from various species of fleas: *Francisella tularensis* causing **tularemia**, *Salmonella enteritidis* causing **salmonellosis**, and *Staphylococcus aureus* causing **staphylococcal infection**. All of these agents can be transmitted by other means such as by other ectoparasites and contact or aerosol exposure to infective fomites and mammalian tissues. All three infections are widespread, and the importance of fleas in transmission varies regionally and can be difficult to quantify. Certain skin lesions including flea bites can result in methicillin-resistant *Staphylococcus aureus* (MRSA) infections that can prove difficult to treat with conventional antibiotics.

Other bacterial agents that can be transmitted by fleas are *Bartonella* (formerly *Rochalimaea*) *henselae*, the agent of **cat scratch disease** and *Bartonella elizabethae*, which can damage heart valves often causing inflammation (endocarditis). Infections by these zoonotic agents typically cause swollen regional lymph nodes. Long-term bacteremia caused by either of these agents can occur in inapparently infected cats, which are important reservoir hosts. About 25,000 cases of cat scratch disease occur annually in the United States, whereas endocarditis caused by *B. elizabethae* appears to be relatively uncommon. Infection with *B. henselae* also can cause fever, hepatitis (liver inflammation), endocarditis (which may eventually necessitate heart valve replacement surgery), bacillary

angiomatosis, and bacillary peliosis. The last two conditions manifest as vascular proliferations and are most commonly seen in immunocompromised persons such as HIV-positive individuals. Cat fleas can transmit *B. henselae* to cats under laboratory conditions. Although cat fleas may also be capable of transmitting this pathogen to humans, a scratch from an infected cat appears to be the more common mode of transmission. A related organism, *Bartonella clarridgeiae*, can also occur in cats and cause infection in humans, but the potential role of fleas as vectors of this agent has not been determined. Treatment of bartonellosis is with broad-spectrum antibiotics, but many patients recover without antibiotic treatment.

The following zoonotic bacterial agents also have been detected in fleas, but it is assumed that fleas are not vectors: *Borrelia burgdorferi*, the etiologic agent of Lyme disease; *Borrelia duttoni*, an agent of relapsing fever; *Listeria monocytogenes*, the agent of listeriosis; *Yersinia pseudotuberculosis* causing pseudotuberculosis (yersiniosis); *Erysipelothrix rhusiopathiae* causing erysipelas; and *Brucella abortus* causing brucellosis.

Viruses

Although several viral pathogens of humans have been isolated from, or detected in, fleas, the role of fleas in their transmission is either unknown or considered to be incidental. These viruses include those that cause lymphocytic choriomeningitis, tick-borne encephalitis, and the related Russian spring-summer encephalitis and Omsk hemorrhagic fever.

Demonstration of a pathogen within an arthropod does not necessarily imply that it is a vector of that agent. Controlled transmission experiments should be undertaken to further investigate the ability of fleas (or other blood-feeding arthropods) to transmit these pathogens.

Tungiasis

Tungiasis is the pathological condition resulting from infestation by fleas belonging to the genus *Tunga*. Although there are several species of *Tunga*, the **chigoe** (*T. penetrans*) (Fig. 10.14) is the principal species that attacks humans. *Tunga penetrans* occurs in many tropical and subtropical zones but is especially common in the New World tropics, the West Indies, tropical Africa, and southern India. It has become especially common in parts of east Africa, often infesting humans in large numbers. The first record of this flea was in 1492, from crewmen of Christopher Columbus stationed in Haiti. It apparently spread from the New World to other areas of the world by shipping commerce; it was first recorded on the African continent in 1732. Another species, *T. trimamillata*, has been recorded as infesting humans and domestic animals in Ecuador and Peru.

Females of *T. penetrans* usually invade a site between the toes, beneath the toenails, or on the soles of the feet. Other sites may include the hands, legs, arms, especially around the elbow, and genital region in heavy infestations. Skin invasion by this flea can cause painful, subcutaneous lesions that often lead to more serious medical complications. The embedded chigoes (Figs. 10.15 and 10.16) invariably cause intense irritation and can result in secondary infections that ooze pus. When several chigoes attack an individual host at the same time, ulcerations often develop as the resultant lesions coalesce. Some people may harbor many lesions on their feet and ankles, which can result in their being unable to walk. Multiple lesions on the hands, especially at the fingertips, can result in difficulty with gripping. Tetanus (in unvaccinated individuals), cellulitis (inflammation of cellular or connective tissue), regional lymphadenitis (swollen lymph nodes), deformation of digits, and loss of toenails and fingernails may occur. Impaired blood flow to the site can lead to gangrene and may necessitate amputation of toes, or sometimes an entire foot or lower leg. Therefore, chigoe lesions should receive prompt medical attention. Although the flea can be removed using a sterile needle or scalpel, it is important that lesions be thoroughly cleaned and dressed to avoid infection. This also applies to embedded dead fleas, which may rapidly cause affected tissues to fester and ulcerate if left untreated. Prudent defense against tungiasis includes not walking barefoot on beaches and other sandy soils where this flea develops in endemic regions. Eisele et al. (2003), Pampiglione et al. (2009), and Richardson and Mangili (2016) provide reviews of tungiasis.

Fleas as Intermediate Hosts of Helminths

Certain fleas are intermediate hosts for the cysticercoid stage of three species of tapeworms that occasionally infest humans. The most important of these is the **double-pored tapeworm** (*Dipylidium caninum*), the adults of which normally parasitize dogs. Gravid worm-like proglottids (Fig. 10.24) are released by *D. caninum* adults in the gut of the definitive host. These actively exit the anus, partially dry upon exposure to air, and fall to the ground where they resemble sesame seeds. The subsequently expelled eggs are ingested by flea larvae; the chewing mandibles of the larvae enable them to ingest the eggs whereas the sucking mouthparts of adult fleas do not. Fleas such as *C. felis*, *C. canis*, and *P. irritans* have a significant role as intermediate hosts for this tapeworm. The dog-chewing louse (*Trichodectes canis*) occasionally ingests *D. caninum* eggs and also can serve as an intermediate host.

The tapeworm develops slowly in flea larvae but rapidly in flea pupae. Cysticercoids can be seen in the body cavity of larvae and pupae, where they remain through development of the flea to the adult stage. Some flea mortality

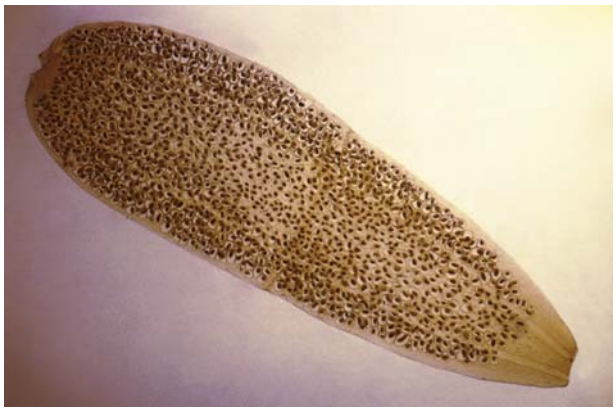


FIGURE 10.24 Proglottid of the double-pored tapeworm, *Dipylidium caninum*. *Courtesy, U.S. Public Health Service, Public Health Image Library.*

occurs in the pupal stage due to this helminth. Infestation of human (definitive) hosts occurs when a person incidentally ingests an infested flea. The cysticercoid is liberated from the flea by digestive enzymes, after which it everts and attaches to the gut of its new host. Children playing with pets are especially susceptible to infestation by this tapeworm.

Two other tapeworms that use fleas as intermediate hosts are the rodent tapeworm (*Hymenolepis diminuta*) and the dwarf tapeworm (*Hymenolepis nana*). Both infest rodents and occasionally parasitize humans, especially children. The development and transmission mechanisms of these two cestodes are similar to those for *D. caninum*. Both *H. diminuta* and *H. nana* form viable cysticercoids in several species of fleas, especially *C. canis*, *P. irritans*, *X. cheopis*, and *N. fasciatus*. They can also infest some other arthropods, notably coprophagous beetles.

The zoonotic nematode *Trichinella spiralis*, which causes trichinosis, has also been found in fleas, although this is assumed to represent an accidental association.

VETERINARY IMPORTANCE

Several species of fleas are important ectoparasites of domestic and wild animals. Emphasis here is given to those that infest pets and livestock. Many fleas associated with domestic animals are merely a nuisance through their biting activity. However, they also may cause FAD and anemia. Other fleas such as sticktights and chigoes embed their mouthparts or entire bodies in mammalian or avian tissues, causing local inflammation and other problems. Some fleas are intermediate hosts of helminths that parasitize domestic animals, whereas others transmit pathogens such as viruses and trypanosomes to their hosts.

The **cat flea** (*C. felis*) (Fig. 10.9) is an extremely important ectoparasite not only of cats and dogs but also of several other mammals including opossums, cattle, horses, sheep, goats, rabbits, and monkeys. Some populations of *C. felis* have adapted to certain hosts, such as dogs or cattle, and show a preference for feeding on these species. Occasionally, cat fleas infest goats, lambs, calves, or other ungulates in large numbers and can cause anemia or even death. Individual pets, especially cats and dogs, may support hundreds or thousands of cat fleas. Because the larvae thrive on blood-rich fecal pellets voided by adult fleas on the host, it is important to vacuum or treat areas where pets rest or sleep to reduce flea numbers. The dog flea (*C. canis*) (Fig. 10.10) is becoming a less frequent ectoparasite of dogs, with established populations on dogs persisting in only a few countries or regions. Almost invariably, fleas associated with dogs are *C. felis*. Further details on the biology of fleas associated with cats and dogs are provided by Dryden (1993) and Rust and Dryden (1997).

Several species of fleas are parasites of domestic and laboratory rats and mice. These include the Oriental rat flea (*X. cheopis*) (Fig. 10.11), the northern rat flea (*N. fasciatus*) (Fig. 10.17), and the European mouse flea (*L. segnis*) (Fig. 10.19). Flea infestations of these rodents are usually more important with respect to potential transmission of pathogens rather than their discomforting bites. The European rabbit flea (*S. cuniculi*) is a parasite of the European rabbit (*Oryctolagus cuniculus*) throughout much of the world, where it has been introduced as a game or small-livestock animal. Because this is the laboratory rabbit commonly used in scientific studies, the European rabbit flea occasionally is recorded in animal research facilities. This flea is commonly a pest in rabbit hutches and where European rabbits are raised commercially for food in many parts of the world. *Spilopsyllus cuniculi* usually attaches to the ears, where it embeds its mouthparts deeply and for long periods, causing host irritability and ear scabbing.

In central Asia, the **alakurt fleas** *Dorcadia ioffi* and *Vermipsylla alakurt* parasitize ungulates, especially horses, sheep, and yaks. These fleas often occur in large numbers on these hosts and can cause anemia, hair loss, retarded growth, unthriftiness, and occasionally death, especially in newborn lambs.

Other fleas that are annoying biters of domestic mammals include *P. simulans*, the human flea (*P. irritans*) (Fig. 10.8), and the sticktight flea (*E. gallinacea*) (Fig. 10.12), all of which may parasitize cats or dogs. *Pulex simulans* and the human flea can be important ectoparasites of dogs and swine, whereas the sticktight flea can infest domestic rats and several other mammals.

Several species of fleas feed on birds. At least three of these are important pests in the poultry industry. The sticktight flea is principally a poultry pest in the warm temperate, subtropical, and tropical regions of the New World. These small fleas typically attach to the non-feathered areas of birds such as the head (Fig. 10.13), comb, wattle, and anus. Large flea populations can cause

anemia. Feeding sites can become ulcerated; when this occurs around the eyes, blindness can result and the host is unable to feed. Secondary infections may develop. The European chicken flea (hen flea) (*C. gallinae*) (Fig. 10.18) is a nonsedentary ectoparasite of domestic fowl in several parts of the world, including Europe and eastern North America. In western North America, the western chicken flea (*Ceratophyllus niger*), another nonsedentary species, is a parasite of domestic fowl and several species of wild birds. All of these poultry fleas can cause host emaciation and reduced egg production when they occur in large numbers.

Flea Allergy Dermatitis

Allergic skin reactions (FAD) to flea bites are a common problem of domestic animals, especially dogs and cats. Hypersensitivity to saliva from feeding fleas is usually more apparent in pets than in humans because larger numbers of pets are bitten by fleas. A single flea bite can trigger an acute, sometimes chronic, dermatitis in hypersensitive dogs or cats. Frequent scratching and skin irritation, especially during the warmer months, often reflects this condition. In cats, flea-bite dermatitis usually manifests as purplish papules that are often covered with crusted skin; in dogs, crusts are typically absent. In both cats and dogs, lesions are usually concentrated on the rump and inner thighs, with accompanying fur loss from frequent scratching. Cats sometimes also have a ring of crusts around the neck. Diligent flea control is important in combating this condition. Administration of corticosteroids or a course of desensitizing antigens are other treatment options. Except in severe cases, the hypersensitivity often resolves after repeated flea bites as the host gradually becomes desensitized to antigens in flea saliva.

Tungiasis

Some domestic animals, especially hogs and occasionally dogs (Fig. 10.25), are parasitized by *T. penetrans* (Fig. 10.14) causing tungiasis. Infestations in hogs primarily affect the feet but also the snout, teats, legs, and scrotum. Infestations of the teats can result in restricted milk flow in nursing sows and starvation of piglets. Swine are reservoirs of tungiasis in some tropical climates and the fleas can be transferred to humans. Dogs are commonly infested by *T. penetrans* in some tropical regions including rural Brazil. Females often embed in the snout (Fig. 10.25) or the pads of dogs' feet. There are at least nine other species of fleas belonging to the genus *Tunga* that burrow into host tissues. Females of each of these species are subdermal parasites that mostly attack New World rodents. Hopkins and Rothschild (1953) illustrate and discuss other species of *Tunga*. A relatively recently discovered species, *T. trimamillata*, has been recorded as causing tungiasis in domestic animals and humans in Ecuador and Peru (Pampiglione et al., 2009).

FIGURE 10.25 Tungiasis; several female *Tunga penetrans* embedded in a dog's nose. *From Chiebao et al., 2005.*

Myxomatosis

Myxomatosis is primarily a disease of the European rabbit caused by infection with the Myxoma virus. The virus causes benign fibromas in its natural rabbit hosts in California (USA), Central America, and South America. However, in the European rabbit, a severe and usually fatal infection with enlarging skin lesions and generalized viremia occurs. Myxoma virus was purposely introduced into Australia in 1950 and into Europe in 1953. The aim of these introductions was to control burgeoning populations of European rabbits.

The virus is mechanically transmitted to rabbits by various blood-feeding arthropods, particularly mosquitoes. However, the European rabbit flea (*S. cuniculi*) is also a proven vector, at least in Britain where this flea occurs naturally, and in Australia, where it was introduced in 1966. Although it is an inefficient vector, an Australian sticktight flea (*Echidnophaga myrmecobii*) also can transmit the Myxoma virus to rabbits. Infection with this virus apparently does not adversely affect these flea vectors. As with most other flea-transmitted pathogens, Myxoma virus remains confined to the gut and mouthparts of *S. cuniculi*. Survival of the virus for 3–4 months in infected fleas has been demonstrated.

Because strains of Myxoma virus differ in virulence and rabbit populations differ in their susceptibility to this pathogen, the success of this virus in controlling rabbits has been variable. When the virus was first introduced to Australia and Europe, it was effective in culling wild rabbits; today, however, many rabbit

populations in both Australia and Europe have developed resistance to several strains of the virus.

Squirrel Pox

In Britain, squirrel fleas have been implicated as vectors of a *Parapoxvirus* to native red squirrels (*Sciurus vulgaris*) and invasive North American gray squirrels (*Sciurus carolinensis*). The virus probably first infected gray squirrels, which have developed immunity, but infected red squirrels are highly susceptible to the resulting pox lesions and associated pathology and often die. Partly because of this, invasive gray squirrels are currently much more common than native red squirrels in Britain.

Murine Trypanosomiasis

Trypanosoma lewisi is the causative agent of murine trypanosomiasis in domestic rats throughout much of the world. It is principally transmitted by the Northern rat flea (*N. fasciatus*) (Fig. 10.17) and the Oriental rat flea (*X. cheopis*) (Fig. 10.11). Fleas imbibe trypanosomes while feeding on infected rats; the pathogen remains in the flea midgut where development occurs. Within 6 h after ingestion, the trypanosomes invade midgut epithelial cells, transform into pear-shaped forms, and begin to divide. The parasitized gut cells rupture after 18 h to 5 days to release the trypanosomes; these then either invade new epithelial cells to repeat the process or move posteriorly to the rectum and anus. Trypanosomes in this "rectal phase" are voided in the flea feces. The trypanosomes enter rat hosts when the latter lick and scratch their fur during grooming, representing a classic example of posterior-station transmission. Murine trypanosomiasis is usually a benign infection in rats. However, the *T. lewisi*—flea—rat system has been used as a laboratory model to study more virulent trypanosome species that are pathogenic to humans and domestic animals.

At least 10 species of trypanosomes are transmitted to rodents by fleas. Other rodent trypanosomes with confirmed flea transmission cycles include *Trypanosoma musculi* (synonym: *Trypanosoma duttoni*) of house mice, *Trypanosoma rabinowitschi* of hamsters, *Trypanosoma neotomae* of wood rats, and *Trypanosoma grosi* of the European wood mouse (*Apodemus sylvaticus*). Also, *Trypanosoma nabiasi* is known to be transmitted to rabbits by fleas. Fleas are also suspected to be vectors of trypanosomes associated with some birds, shrews, voles, and other lagomorphs.

Other Flea-Borne Pathogens and Parasites

Many of the flea-borne pathogens listed in Table 10.2 cause diseases in humans, with wild or domestic animals serving as reservoirs. These include plague, tularemia, murine typhus, Q fever, cat scratch disease (bartonellosis), and sylvatic epidemic typhus. Infections of domestic animals with most of these pathogens can be inapparent, febrile, or fatal, depending on the host species, its health, and the species or strain of pathogen involved. Cats, for example, are typically susceptible to most strains of plague, whereas dogs usually are not.

Other pathogens of veterinary importance that have been isolated from, or detected in, fleas include lymphocytic choriomeningitis virus, which affects many mammals, especially rodents, feline leukopenia virus, and the following bacterial agents: *Borrelia burgdorferi* the causative agent of Lyme disease, *Listeria monocytogenes*, the agent of listeriosis mainly in ungulates; *Brucella abortus*, an agent of brucellosis mainly in bovines; *Burkholderia mallei*, the agent of glanders in equines; and *Burkholderia pseudomallei*, the agent of melioidosis in several mammals. However, the role of fleas as vectors of these pathogens is doubtful or undetermined.

Other microorganisms known to occur in fleas and that may be transmitted to vertebrates include hemogregarine sporozoans, various rickettsial organisms, and several symbionts. The protozoan *Hepatozoon erhardovae* is transmitted to European voles (*Clethrionomys* spp.) by at least five species of fleas. The parasite reproduces sexually in the hemocoel of fleas, where it develops to the sporocyst stage; transmission to voles occurs when they eat infected fleas during grooming. The related *Hepatozoon pitymysi* and *Hepatozoon sciuri*, which parasitize North American and Eurasian voles and North American squirrels, respectively, have also been detected in fleas and are thought to be transmitted in a similar way.

Fleas as Intermediate Hosts or Vectors of Helminths

The double-pored tapeworm (*D. caninum*) (Fig. 10.24) normally develops as an adult parasite in the intestines of dogs, cats, and some wild carnivores. The most important intermediate flea hosts are the cat flea and dog flea, although the human flea can also serve in this capacity. In tropical Africa, a warthog flea (*Echidnophaga larina*) is sometimes responsible for *D. caninum* infestations in domestic dogs. Infestations are usually initiated when animals consume parasitized fleas while grooming.

Two species of tapeworms that typically infest rats and mice as adults are the rodent tapeworm (*H. diminuta*) and the dwarf tapeworm (*H. nana*). Rat fleas, especially the Oriental rat flea and the northern rat flea, serve as intermediate hosts. Infestations are initiated when infested fleas are eaten by the definitive rodent hosts.

The onchocercid nematode, *Acanthocheilonema reconditum*, which causes a relatively benign form of canine

filariasis in many parts of the world, has been found in several species of fleas. The cat flea and dog flea are considered to be the principal vectors. Transmission of mature larvae probably occurs when carnivores ingest infected fleas during grooming (Napoli et al., 2014). Dogs, jackals, and hyenas are the principal definitive hosts of *A. reconditum*.

Several other species of helminths have been isolated from wild-caught fleas, and fleas have been found to serve as suitable intermediate hosts for some of these under laboratory conditions. However, the importance of fleas in maintaining these pathogens in nature is unknown. For example, the parasitic nematode, *Trichinella spiralis*, has been found to be encysted in the Oriental rat flea in India; this helminth normally encysts in the muscle tissue of rats and hogs, causing trichinosis.

PREVENTION AND CONTROL

Because the cat flea (*C. felis*) infests dogs and cats, it is the flea most commonly encountered as a household pest. Its broad host range means that wild animals serve as sources of reinfestation despite management efforts. Controlling cat fleas requires addressing them on their hosts, in buildings, and in outdoor host habitats.

Environmental flea control efforts should be focused on areas where pets spend the most time, especially where they sleep, because these are locations most likely to support the development of flea larvae. Indoors, daily vacuuming can remove flea eggs before they have a chance to hatch. Insect growth regulators are particularly effective when applied in these locations because they penetrate the flea egg shell and kill the developing embryo as well as prevent development of larvae that have already emerged. For immediate elimination of a flea infestation, steam cleaning of carpets can be effective.

Outdoor flea control can be challenging because feral mammals such as raccoons, opossums, dogs, and cats may continually reinfest premises. Crawl spaces should be sealed to prevent denning under the structure, and outbuildings should likewise be made inaccessible to wildlife. Flea larvae cannot survive the heat and drying conditions of full sun exposure, so control efforts should be directed toward shaded areas of host activity. Typically, this includes under shrubbery, against building foundations, in crawl spaces, or areas under porches or decks. **Pulicides** (flea control agents) used in these sites should have sustained residual efficacy and be photostable. Outdoor insecticide use typically is an ineffectual strategy against fleas. Instead, flea-infested habitats should be modified to expose them to sunlight and air movement, drying out the immature stages before they can develop into adult blood-sucking fleas.

Insect growth regulators, especially formulations of methoprene and pyriproxyfen, are popular flea-control weapons because they have low mammalian toxicity and good residual efficacy against flea larvae (Rust and Hemsworth, 2017). Although insect growth regulators and chitin synthesis inhibitors do not kill adult fleas, they can act as larvicides, preventing flea eggs from hatching and larvae from successfully molting. Lufenuron is a chitin synthesis inhibitor that is administered orally as a pill for dogs or as a liquid added to food or an injectable formulation for cats. Female fleas fed on lufenuron-laced blood yield eggs that do not hatch or that produce larvae incapable of molting, and quickly die. Pyriproxyfen and methoprene are juvenile hormone analogs that can be applied topically; they penetrate the chorion of flea eggs and prevent successful embryonic development.

Because adult fleas must feed on blood, host-targeted control uses the animal as the lure, ensuring that all adult fleas are exposed to the pulicide when they blood feed. Some insecticides may be given orally to the host so that the active ingredient passes into the bloodstream and is picked up as the fleas feed.

Other insecticides may be topically applied so that the flea acquires the toxicant via its cuticle. Topical products generally are lipophilic and maintained in the dermal lipids of the host, where they are available for cuticular absorption by fleas. Host-targeted ectoparasiticides include pyrethrins, pyrethroids, neonicotinoids, phenylpyrazoles, macrocyclic lactones, semicarbazones, oxadiazines, isoxazolines, juvenile hormone analogs, and chitin synthesis inhibitors. Development of host-targeted products requires chemistry that is efficacious against fleas and low in mammalian toxicity.

Some products, such as those containing permethrin, are toxic to felines and are not labeled for use on cats or young puppies. Furthermore, some products with the same name may include active ingredients in the canine version that are different from those in their feline counterparts.

On pets, flea infestation should be confirmed before control efforts are undertaken. Dogs and cats scratch for numerous reasons other than fleas, including dry skin. Bathing or the use of host-applied pulicides can further aggravate dry skin, increasing itching and scratching even in the absence of fleas. Flea combs can be used to sample the animal for fleas and also offer a mechanical control option for pet owners disinclined to use insecticides.

Two biological control agents, the parasitic nematode *Steinernema carpocapsae* and the entomopathogenic fungus *Beauveria bassiana*, reduce cat flea numbers under laboratory conditions, but commercialization requires specific formulation so that the product can function in microenvironments where fleas are found. The flagellate protozoan *Leptomonas ctenocephali* infects cat fleas and dog fleas but does not appear to cause significant mortality.

Some progress has been made in developing vaccines against fleas, mainly using midgut antigens of the cat flea to

induce an immune response in the host. In several trials, dogs, cats, and rabbits that were experimentally challenged with cat-flea antigens had a significantly higher number of dead or reproductively compromised fleas than did unvaccinated animals.

For humans, personal protectants such as those containing diethylmethylbenzamide, (formerly *N*,*N*-diethyl-m-toluamide) or permethrin can be helpful in reducing the number of flea bites. Permethrin should be applied only to clothing and not directly on the skin. Although banned for use in the United States, dichloro-diphenyl-trichloroethane (DDT) is still used to control outbreaks of flea-borne diseases such as plague or murine typhus in some parts of the world. As with the use of other insecticides, there is a constant risk that fleas may develop resistance to these chemicals.

Plague outbreaks are usually followed by public education and area-wide programs to remove rodent hosts and flea vectors. Control programs for murine typhus typically involve eliminating the flea vectors or rodent reservoirs by insecticide applications and trapping, respectively. Rodent harborages and access of these reservoir hosts to houses should be eliminated where feasible. Because fleas abandoning dead rodents present a health risk to humans, the reservoir hosts and their burrows should be treated with effective insecticides to eliminate the fleas before rodent eradication efforts. Dusting rodent burrows with insecticides or providing rodent bait stations spiked with flea-control agents can be effective in killing fleas. Afterward, rodent reservoir populations are sometimes eliminated to prevent subsequent disease outbreaks. Frequent surveillance of rodent and flea populations in plague-endemic regions often allows control measures to be implemented before human cases occur. Outbreaks of murine typhus may be handled in a similar manner, although there is greater emphasis on rodent control because the reservoir hosts are more likely to be commensal rats.

Ineffective approaches to flea control include ultrasonic repellent devices. No fleas that have been tested have shown responses to ultrasound or to devices incorporating it. Likewise, neither oral intake of garlic nor that of B-complex vitamins, including brewer's yeast has been proven to reduce flea populations on pets despite claims about their effectiveness. Flea control strategies are addressed by MacDonald (1995), Rust and Dryden (1997), Hinkle et al. (1997), Dryden (1999), Rust et al. (2003), and Rust (2017).

REFERENCES AND FURTHER READING

Azad, A. F. (1990). Epidemiology of murine typhus. *Annual Review of Entomology, 35*, 553–569.

Azad, A. F., Radulovic, S., Higgins, J. A., Noden, B. H., & Troyer, J. M. (1997). Flea-borne rickettsiosis: Ecologic considerations. *Emerging Infectious Diseases, 3*, 319–327.

Beaucournu, J.-C., & Launay, H. (1990). Les puces (Siphonaptera) de France et du Bassin Méditerraneén occidental. *Faune de France, 76*, 1–548.

Benton, A. H. (1980). *An atlas of the fleas of the eastern United States*. New York: Marginal Media, Fredonia, 177 p.

Benton, A. H. (1983). *An illustrated key to the fleas of the eastern United States*. New York: Marginal Media, Fredonia, 34 p.

Bertherat, E., Thullier, P., Shako, J. C., England, K., Koné, M.-L., Arntzen, L., et al. (2011). Lessons learned about pneumonic plague diagnosis from 2 outbreaks, Democratic Republic of the Congo. *Emerging Infectious Diseases, 17*, 778–784.

Bitam, I., Dittmar, K., Parola, P., Whiting, M. F., & Raoult, D. (2010). Fleas and flea-borne diseases. *International Journal of Infectious Diseases, 14*, e667–e676.

Brown, L. D., & Macaluso, K. R. (2016). *Rickettsia felis*, an emerging flea-borne rickettsiosis. *Current Tropical Medicine Reports, 3*, 27–39.

Carniel, E., & Hinnebusch, B. J. (Eds.). (2004). *Yersinia: Molecular and cellular biology*. Milton Park, UK: Horizon Bioscience, 420 p.

Chapman, A. S., Swerdlow, D. L., Dato, V. M., Anderson, A. D., Moodie, C. E., Marriott, C. E., et al. (2009). Cluster of sylvatic epidemic typhus cases associated with flying squirrels, 2004-2006. *Emerging Infectious Diseases, 15*, 1005–1011.

Chiebao, D. P., Rodrigues, A. R., Pinheiro, S. R., & Gennari, S. M. (2005). Ocorréncia de tungíase em cáes no município de São Paulo. *Clinica Veterinária, 10*(59), 50–54.

Christakos, G., Olea, R. A., Serre, M. L., Yu, H.-L., & Wang, L.-L. (2005). *Interdisciplinary public health reasoning and modelling: The case of the black death*. Berlin: Springer, 320 p.

Dennis, D. T., Gage, K. L., Gratz, N., Poland, J. D., & Tikhomirov, E. (1999). *Plague manual: Epidemiology, distribution, surveillance and control*. Geneva: World Health Organization. WHO/CDS/CSR/EDC/99.2. 171 pp. (also available as an electronic document on WHO website).

Dittmar, K., Zhu, Q., Hastriter, M. W., & Whiting, M. F. (2016). On the probability of dinosaur fleas. *BMC Evolutionary Biology, 16*(9).

Drancourt, M., Roux, V., Dang, L. V., Tran-Hung, L., Castex, D., Chenal-Francisque, V., et al. (2004). Genotyping, Orientalis-like *Yersinia pestis* and plague pandemics. *Emerging Infectious Diseases, 10*, 1585–1592.

Drancourt, M., Signoli, M., Dang, L. V., Bizot, B., Roux, V., Tzortzis, S., et al. (2007). *Yersinia pestis* Orientalis in remains of ancient plague patients. *Emerging Infectious Diseases, 13*, 332–333.

Dryden, M. W. (1993). Biology of fleas of cats and dogs. *Compendium on Continuing Education for the Practicing Veterinarian, 15*, 569–579.

Dryden, M. W. (1999). Highlights and horizons in flea control. *Compendium on Continuing Education for the Practicing Veterinarian, 21*, 296-298, 361–365.

Duplaix, N. (1988). Fleas. The lethal leapers. *National Geographic, 173*, 672–694.

Eisele, M., Heukelbach, J., van Marck, E., Melhorn, H., Meckes, O., Franck, S., et al. (2003). Investigations on the biology, epidemiology, pathology and control of *Tunga penetrans* in Brazil: I. Natural history of tungiasis in man. *Parasitology Research, 90*, 87–99.

Eisen, R. J., Dennis, D. T., & Gage, K. L. (2015). The role of early-phase transmission in the spread of *Yersinia pestis*. *Journal of Medical Entomology, 52*, 1183–1192.

Eisen, R. J., Eisen, L., & Gage, K. L. (2009). Studies of vector competency and efficiency of North American fleas for *Yersinia pestis*: State of the field and future research needs. *Journal of Medical Entomology, 46*, 737–744.

Eisen, R. J., Enscore, R. E., Biggerstaff, B. J., Reynolds, P. J., Ettestad, P., Brown, T., et al. (2007). Human plague in the southwestern United States, 1957-2004: Spatial models of elevated risk of human exposure to *Yersinia pestis*. *Journal of Medical Entomology, 44*, 530−537.

Eisen, R. J., & Gage, K. L. (2010). Transmission of flea-borne zoonotic agents. *Annual Review of Entomology, 57*, 61−82.

Elbel, R. E. (1991). Order Siphonaptera. In F. W. Stehr (Ed.), *Immature insects* (Vol. 2, pp. 674−689). Dubuque, Iowa: Kendall Hunt.

Erickson, T., da Silva, J., Nolan, M. S., Marquez, L., Munoz, F. M., & Murray, K. O. (2017). Newly recognized pediatric cases of typhus group rickettsiosis, Houston, Texas, USA. *Emerging Infectious Diseases, 23*, 2068−2071.

Fenner, F., & Ross, J. (1994). Myxomatosis. In H. V. Thompson, & C. M. King (Eds.), *The European rabbit. The history and biology of a successful coloniser* (pp. 205−239). Oxford: Oxford University Press.

Fox, I. (1940). *Fleas of eastern United States*. Ames: Iowa State College Press, 191 p.

Gage, K. L., & Kosoy, M. Y. (2005). Natural history of plague: Perspectives from more than a century of research. *Annual Review of Entomology, 50*, 505−528.

Goddard, J. (1998). Fleas and murine typhus. *Infections in Medicine, 15*, 438−440.

Hinkle, N. C. (2008). 5. Fleas. In X. Bonnefoy, H. Kampen, & K. Sweeney (Eds.), *Public health significance of urban pests* (pp. 155−173). Geneva: World Health Organization (Also available as an electronic document on the WHO web site).

Hinkle, N. C., Rust, M. K., & Reierson, D. A. (1997). Biorational approaches to flea (Siphonaptera, Pulicidae) suppression − present and future. *Journal of Agricultural Entomology, 14*, 309−321.

Holland, G. P. (1985). *The fleas of Canada, Alaska and Greenland (Siphonaptera). Memoirs of the Entomological Society of Canada*. no. 130. 631 p.

Hopkins, G. H. E., & Rothschild, M. (1953-1971). *An illustrated catalogue of the Rothschild collection of fleas (Siphonaptera) in the British Museum (Natural History). Vols. I-V*. London: British Museum (Natural History).

Hopla, C. E., & Hopla, A. K. (1994). Tularemia, pp. 113−123. In G. W. Beran (ed.-in-chief), *Handbook of Zoonoses. Section A. Bacterial, Rickettsial, Chlamydial, and Mycotic* (2nd ed.). Boca Raton: CRC Press.

Kelly, J. (2004). *The great mortality: An intimate history of the black death, the most devastating plague of all time*. New York: HarperCollins, 364 p.

Krasnov, B. R. (2008). *Functional and evolutionary ecology of fleas: A model for ecological parsitology*. Cambridge University Press.

Lewis, R. E. (1972). Notes on the geographic distribution and host preferences in the order Siphonaptera. Part 1. Pulicidae. *Journal of Medical Entomology, 9*, 511−520.

Lewis, R. E. (1973). Notes on the geographic distribution and host preferences in the order Siphonaptera. Part 2. Rhopalopsyllidae, Malacopsyllidae and Vermipsyllidae. *Journal of Medical Entomology, 10*, 255−260.

Lewis, R. E. (1974a). Notes on the geographic distribution and host preferences in the order Siphonaptera. Part 3. Hystrichopsyllidae. *Journal of Medical Entomology, 11*, 147−167.

Lewis, R. E. (1974b). Notes on the geographic distribution and host preferences in the order Siphonaptera. Part 4. Coptopsyllidae, Pygiopsyllidae, Stephanocercidae and Xiphiopsyllidae. *Journal of Medical Entomology, 11*, 403−413.

Lewis, R. E. (1974c). Notes on the geographic distribution and host preferences in the order Siphonaptera. Part 5. Ancistropsyllidae, Chimaeropsyllidae, Ischnopsyllidae, Leptopsllidae and Macropsyllidae. *Journal of Medical Entomology, 11*, 525−540.

Lewis, R. E. (1974d). Notes on the geographic distribution and host preferences in the order Siphonaptera. Part 6. Ceratophyllidae. *Journal of Medical Entomology, 11*, 658−676.

Lewis, R. E. (1993a). Notes on the geographic distribution and host preferences in the order Siphonaptera. Part 8. New taxa described between 1984 and 1990, with a current classification of the order. *Journal of Medical Entomology, 30*, 239−256.

Lewis, R. E. (1998). Résumé of the Siphonaptera (Insecta) of the world. *Journal of Medical Entomology, 35*, 377−389.

Lewis, R. E., & Eckerlin, R. P. (2018). *The Siphonaptera of North America*. Annals of Carnegie Museum.

Lewis, R. E., & Lewis, J. H. (1985). Notes on the geographic distribution and host preferences in the order Siphonaptera. Part 7. New taxa described between 1972 and 1983, with a supraspecfic classification of the order. *Journal of Medical Entomology, 22*, 134−152.

Lewis, R. E., Lewis, J. H., & Maser, C. (1988). *The fleas of the Pacific northwest*. Corvallis: Oregon State Univ. Press, 296 p.

Lowell, J. L., Eisen, R. J., Schotthoefer, A. M., Liang, X., Montenieri, J. A., Tanda, D., et al. (2009). Colorado-based plague surveillance systems: Relationships between targeted animal species and prediction efficacy of areas at risk for humans. *Journal of Vector Ecology, 34*, 22−31.

MacDonald, J. M. (1995). Flea control: An overview of treatment concepts for North America. *Veterinary Dermatology, 6*, 121−130.

Madon, M. B., Hitchcock, J. C., Davis, R. M., Myers, C. M., Smith, C. R., Fritz, C. L., et al. (1997). An overview of plague in the United States and a report of investigations of two human cases in kern County, California. *Journal of Vector Ecology, 22*, 77−82.

Marchiondo, A. A., Holdsworth, P. A., Green, P., Blagburn, B. L., & Jacobs, D. E. (2007). World Association for the Advancement of Veterinary Parasitology (W.A.A.V.P.) guidelines for evaluating the efficacy of parasiticides for the treatment, prevention and control of flea and tick infestations on dogs and cats. *Veterinary Parasitology, 145*, 332−344.

Mardon, D. K. (1981). *An illustrated catalogue of the Rothschild collection of fleas (Siphonaptera) in the British Museum (Natural History), London. Vol. VI. Pygiopsyllidae*. British Museum (Natural History), 298 p.

Marshall, A. G. (1981). *The ecology of ectoparasitic insects*. London: Academic Press, 459 p.

Mee, C. L., Jr. (1990). How a mysterious disease laid low Europe's masses. *Smithsonian, 20*(11), 66−79.

Murray, K. E., Evert, N., Mayes, B., Fonken, E., Erickson, T., Garcia, M. N., et al. (2017). Typhus group rickettsiosis, Texas, USA, 2003-2013. *Emerging Infectious Diseases, 23*, 645−648.

Napoli, E., Brianti, E., Falsone, L., Gaglio, G., Foit, S., Abramo, F., et al. (2014). Development of *Acanthocheilonema reconditum* (Spirurida, Onchocercidae) in the cat flea *Ctenocephlides felis* (Siphonaptera, Pulicidae). *Parasitology, 141*, 1718−1725.

Pampiglione, S., Fioravanti, M. L., Gustinelli, A., Onore, G., Mantovni, B., Luchetti, A., et al. (2009). Sand flea (*Tunga* spp.) infections in humans and domestic animals: State of the art. *Medical and Veterinary Entomology, 23*, 172−186.

Pérez-Osorio, C. E., Zavala-Velázquez, J. E., Arias-León, J. J., & Zavala-Castro, J. E. (2008). *Rickettsia felis* as an emergent global threat for humans. *Emerging Infectious Diseases, 14*, 1019−1023.

Perry, R. D., & Fetherston, J. D. (1997). *Yersinia pestis* − etiologic agent of plague. *Clinical and Microbiological Reviews, 10*, 35−66.

Poland, J. D., Quan, T. J., & Barnes, A. M. (1994). Plague, pp. 93−112. In: G. W. Beran (ed.-in-chief), *Handbook of Zoonoses. Sect. A: Bacterial, Rickettsial, Chlamydial and Mycotic* (2nd ed.). Boca Raton: CRC Press.

Rawlings, J. A., & Clark, K. A. (1994). Murine typhus, pp. 457−461. In G. W. Beran (ed.-in-chief), *Handbook of Zoonoses. Sect A: Bacterial, Rickettsial, Chlamydial, Mycotic* (2nd ed.). Boca Raton: CRC Press.

Reeves, W. K., Murray, K. O., Meyer, T. E., Bull, L. M., Pascua, R. F., Holmes, K. C., et al. (2008). Serological evidence of typhus group rickettsia in a homeless population in Houston, Texas. *Journal of Vector Ecology, 33*, 205−207.

Reif, K. E., & Macaluso, K. R. (2009). Ecology of *Rickettsia felis*: A review. *Journal of Medical Entomology, 46*, 723−736.

Richardson, D. J., & Mangili, A. M. (2016). Infection with the sand flea *Tunga penetrans* (tungiasis) in a traveler returning from Cameroon, Africa. *Journal of the Arkansas Academy of Science, 70*, 199−206.

Rust, M. K. (2017). The biology and ecology of cat fleas and advancements in their pest management: A review. *Insects, 8*(4), E118.

Rust, M. K., & Dryden, M. W. (1997). The biology, ecology, and management of the cat flea. *Annual Review of Entomology, 42*, 451−473.

Rust, M. K., & Hemsworth, W. L. H. (2017). Intrinsic activity of IGRs against larval cat fleas. *Journal of Medical Entomology, 54*, 418−421.

Smit, F. G. A. M. (1957). *Handbooks for the identification of British insects*. *Siphonaptera* (Vol. 1, Part 16). Royal Entomological Society of London.

Smit, F. G. A. M. (1987). *An illustrated catalogue of the Rothschild collection of fleas (Siphonaptera) in the British Museum (Natural History) Vol. VII. Malacopsylloidea*. London: Oxford University Press, Oxford and British Museum (Natural History), 380 p.

Traub, R. (1985). Coevolution of fleas and mammals. In K. C. Kim (Ed.), *Coevolution of parasitic arthropods and mammals* (pp. 295−437). New York: Wiley.

Traub, R., Rothschild, M., & Haddow, J. (1983). *The Rothschild collection of fleas. The Ceratophyllidae: Key to genera and host relationships with notes on their evolution, zoogeography and medical importance*. Cambridge: Cambridge University Press, 288 p.

Traub, R., & Starcke, H. (Eds.). (1980). *Fleas. Proceedings of the International Conference on Fleas, Ashton, England, June 1977*. Rotterdam: A. A. Balkema, 420 p.

Traub, R., Wisseman, C. L., Jr., & Azad, A. F. (1978). The ecology of murine typhus − A critical review. *Tropical Diseases Bulletin, 75*, 237−317.

Wheelis, M. (2002). Biological warfare at the 1346 siege of Caffa. *Emerging Infectious Diseases, 8*, 971−975.

Whitaker, A. P. (2007). Fleas (Siphonaptera). In *Handbooks for the identification of British insects* (2nd ed., Vol. 1, Part 16). St. Albans, UK: Royal Entomological Society, 178 p.

Whiting, M. F., Whiting, A. S., Hastriter, M. W., & Dittmar, K. (2008). A molecular phylogeny of fleas (Insecta: Siphonaptera): Origins and host associations. *Cladistics, 24*, 1−31.

Zhu, Q., Hastriter, M. W., Whiting, M. F., & Dittmar, K. (2015). Fleas (Siphonaptera) are Cretaceous, and evolved with Theria. *Molecular Phylogenetics and Evolution, 90*, 129−139.

This page intentionally left blank

Chapter 11

Flies (Diptera)

Reid R. Gerhardt[1] **and Lawrence J. Hribar**[2]

[1]*Department of Entomology and Plant Pathology, University of Tennessee, Knoxville, TN, United States;* [2]*Florida Keys Mosquito Control District, Marathon, FL, United States*

The Diptera, or "true flies," are one of the largest, most species-rich, anatomically varied, and ecologically exploitive orders of insects (Yeates et al., 2007). The order name means "two-winged" and refers to the fact that the hind pair of wings is greatly modified and reduced. Flies comprise no less than one-tenth of all species on earth (Wiegmann et al., 2011). The number of described species worldwide is estimated to be 152,000 or more, with many more as yet undescribed and unnamed. There are perhaps 20,000 species of Diptera in the Nearctic Region, a significant proportion of which is cataloged (Stone et al., 1965). Although flies with medical or veterinary significance constitute only a small fraction of these numbers, their diversity is impressive, ranging from mosquitoes to wingless ectoparasites, larvae that parasitize various animals, and species that help to decompose carrion or feces.

No other group of insects has as much impact on human and animal health as does the Diptera (Tables 11.1 and 11.2). Mosquitoes, blackflies, and biting midges annoy outdoor enthusiasts as well as livestock, pets, and other domestic and wild animals. Filth flies associated with cattle, hog, and poultry operations can annoy nearby residents and are frequently the focus of litigation. The ubiquitous housefly is an effective mechanical vector of many pathogens associated with enteric diseases. The depredation of bloodsucking and myiasis-producing flies has an adverse effect on the productivity and profitability of animal agriculture worldwide.

No other group of insects exhibits the number or diversity of vector relationships that have evolved among the Diptera (Table 11.1). The two-volume treatise *Flies and Disease* by Greenberg (1971, 1973) provides an exhaustive list of fly—pathogen associations. Mosquitoes stand as archetypical vectors; they are associated with such historically notorious diseases as malaria, encephalitis, yellow fever, and human filariasis. The story of the US Yellow Fever Commission in Cuba in 1900 and the names Carlos Finlay, Walter Reed (for whom the US Army Medical Center in Washington, DC, is named), and L. O. Howard are familiar to most students of medicine. Such is the importance of mosquitoes that some institutions offer a separate course in **culicidology**, the study of mosquitoes. Insect-vectored tropical diseases such as malaria, filariasis, leishmaniasis, and onchocerciasis currently affect almost half a billion humans worldwide, with about 3.5 billion rated at risk.

Flies are occasionally of direct use to humans. Knowledge of the taxonomy and biology of some necrophilous species makes them useful under certain circumstances in determining how long a body has been dead. This subspecialty of medical entomology, called **medicocriminal** or **forensic entomology**, is readily accepted in judicial circles.

Additional information regarding the medical and veterinary importance of Diptera is provided by Horsfall (1962), Smith (1973), Harwood and James (1979), Williams et al. (1985), and Lancaster (1986).

TAXONOMY

The order Diptera is divided by most authorities into two suborders, the Nematocera and the Brachycera (Table 11.3). However, modern systematic studies, including genetic, molecular, morphological, and paleontological datasets for all life stages, indicate that "Nematocera" is a paraphyletic group of superficially similar lineages (Oosterbrock and Courtney, 1995; Freiderich and Tautz, 1997; Amorim and Yeates, 2006). In fact, studies suggest that hematophagy arose 12 separate times within Diptera, larval endoparasitism 17 times; ectoparasitism 10 times, and loss of functional wings 18 times (Wiegmann et al., 2011). The most current, taxonomically broad, and taxonomically inclusive study of dipteran phylogeny indicates that two rare families, the Deuterophlebiidae and Nymphomyiidae, are the earliest extant fly lineages. The rest of the lower Diptera are

Medical and Veterinary Entomology. https://doi.org/10.1016/B978-0-12-814043-7.00011-X

TABLE 11.1 Major Fly-Borne Diseases and Related Problems Affecting Human Health

Family	Diseases and Other Health-Related Problems	Geographic Occurrence
Psychodidae	Bartonellosis	Andes Mountains of Columbia, Ecuador, and Peru
	Leishmaniasis	New World tropics; Old World tropics and temperate regions
	Sand fly fever	Mediterranean area to southern China and India
Culicidae	Dengue fever	Widespread between latitudes 40°N and 40°S
	Encephalitis	Widespread
	Filariasis	Tropics and Mediterranean area
	Malaria	Widespread in humid tropics
	Yellow fever	Widespread in humid tropics
	Chikungunya	Widespread in humid tropics
	Zika	Widespread in humid tropics
Simuliidae	Onchocerciasis	Tropical Africa and Americas
Tabanidae	Loiasis	Tropical Africa
	Tularemia	Widespread in Northern Hemisphere
Chloropidae	Conjunctivitis	United States (southern) and Mexico; Orient
Muscidae	Enteric diseases	Worldwide
Glossinidae	Trypanosomiasis	Tropical Africa
Calliphoridae	Enteric disease	Worldwide
	Myiasis	Worldwide
Sarcophagidae	Myiasis	Worldwide
Oestridae	Myiasis	Worldwide

composed of four infraorders: **Tipulomorpha** (crane flies), **Culicomorpha** (mosquitoes and related groups), **Psychodomorpha** (sand flies and allies), and **Bibionomorpha** (March flies and gall midges). The **Brachycera** are the sister group of the Bibionomorpha and include horseflies, deerflies, houseflies, and other flies with short antennae. The Brachycera are subdivided into **Tabanomorpha**, including the horseflies and deerflies; **Stratiomyomorpha**, including the soldier flies; **Asiloidea** (robber flies, bee flies, and relatives) and **Empidoidea** (dance flies, dagger flies, and relatives); and **Cyclorrhapha**. The Cyclorrhapha, in turn, is divided into the **Platypezoidea** (flat-footed flies, scuttle flies, and allies) and the **Schizophora**. The latter is divided into a number of acalyptrate superfamilies and **Calyptratae**. Calyptratae includes the medically important brachyceran families, including the superfamilies Oestridae (bot flies and blowflies), Muscoidea (houseflies, garbage flies, and allies), and Hippoboscoidea (tsetse, bat flies, and louse flies). This taxonomic scheme conflicts to some extent with that proposed by McAlpine et al. (1981) and followed by Borror et al. (1989). Studies on the phylogeny and systematics of Order Diptera are ongoing, and the taxonomy of the order is subject to change as new information comes to light. Table 11.3 reflects the most recent thinking but is by no means final. A catalog of the Diptera of America north of Mexico is provided by Stone et al. (1965).

Various keys are available for identifying adult flies. Keys to the families and genera of most Nearctic Diptera are presented in McAlpine (1981b). The flies of western North America are treated by Cole (1969). The key in Borror et al. (1989) is adequate for identifying most North American Diptera to the family level. The larvae of many Diptera can be identified to family with the aid of Teskey (1981b) and Foote (1991); those of synanthropic species are treated by Dusek (1971). Furman and Catts (1982) present a usable key to both adults and larvae of medically important flies, particularly in the United States, and James (1947) covers flies that cause myiasis in humans. For identifying taxa outside the Nearctic Region, students should refer to Lindner's (1949) series on Palearctic Diptera, and to Zumpt (1965) for Old World myiasis-causing flies.

TABLE 11.2 Major Fly-Borne Diseases and Related Problems Affecting Livestock, Poultry, and Other Domestic or Wild Animals

Family	Diseases and Other Health-Related Problems	Geographic Occurrence
Psychodidae	Leishmaniasis	New World tropics; Old World tropics and temperate regions
Ceratopogonidae	Bluetongue	Widespread
Culicidae	Malaria	Widespread in tropics
	Dirofilariasis	Widespread in tropics and temperate regions
	Encephalitis	Widespread
	Fowlpox	Widespread
	Yellow fever	Widespread in humid tropics
Simuliidae	Leucocytozoonosis	Widespread, especially North America
	Feeding damage	Worldwide
Tabanidae	Anaplasmosis	Widespread
	Tularemia	Widespread in Northern Hemisphere
	Exsanguination	Worldwide
Muscidae	Annoyance	Worldwide
	Bovine pinkeye	Northern Hemisphere (widespread)
	Exsanguination	Worldwide
Glossinidae	Nagana	Tropical Africa
Calliphoridae	Myiasis	Worldwide
Sarcophagidae	Myiasis	Worldwide
Oestridae	Myiasis	Worldwide

MORPHOLOGY

Diptera originated in wet environments, and their morphology and life histories reflect that origin. The dipteran pupa became more impervious to the surrounding environmental conditions and the larva became morphologically reduced and evolved to feed on nutrient-rich substrates; flies as a whole occupy a broad range of trophic niches (Wiegmann et al., 2011). Larvae of lower Diptera range in length from only a few millimeters to many centimeters, depending on the species, and are usually distinguished by having a conspicuous head capsule with opposable mandibles that move in a pincer-like horizontal plane (Fig. 11.1). The general body shape ranges from minute and eel-like in the Ceratopogonidae to large and fleshy in the Tipulidae. Some lower Diptera have thoracic prolegs (e.g., Chironomidae and Simuliidae) and others have caudal structures (e.g., Simuliidae) that assist in attachment to substrates. Although the early instars of many aquatic species depend on cuticular respiration, the later instars generally respire via gills or have various adaptations that permit them to obtain atmospheric air. Mosquito larvae, for example, are highly adapted, air-breathing lower Diptera that hang from the water's surface film by respiratory siphons or specialized abdominal setae.

Tabanomorpha larvae have fang-like mandibles that move in a vertical plane; the head capsule is frequently described as "incomplete posteriorly," meaning that only the anterior parts are sclerotized (Fig. 11.2). The latter character is best seen in specimens that have been cleared in potassium hydroxide or lactophenol. Horsefly larvae are good examples of this group. They often have posterior respiratory tubes.

Cyclorrhaphan larvae lack a sclerotized head capsule (Fig. 11.3A) and are commonly known as maggots. At the narrow, anterior end of the 12-segmented larva is the **cephalopharyngeal skeleton** (Fig. 11.3C) that usually bears one or two mouth hooks used for feeding and assisting the insect in movement. The caudal end of the

TABLE 11.3 Taxonomic Classification and Families of Diptera of Interest to Medical and Veterinary Entomologists

Higher Taxa	Family	Common Names
Suborder Nematocera		
Infraorder Tipulomorpha	Tipulidae	Crane flies
Infraorder Bibionomorpha	Bibionidae	March flies
	Mycetophilidae	Fungus gnats
	Sciaridae	Dark-winged fungus gnats
Infraorder Psychodomorpha	Psychodidae[a]	Moth flies, sand flies
Infraorder Culicomorpha	Chaoboridae	Phantom midges
	Corethrellidae	Frog-biting midges
	Culicidae[a]	Mosquitoes
	Simuliidae[a]	Blackflies
	Ceratopogonidae[a]	Biting midges
	Chironomidae	Chironomid midges
Suborder Brachycera		
Infraorder Tabanomorpha	Tabanidae[a]	Horseflies, deerflies
	Rhagionidae	Snipe flies
	Athericidae	Athericid flies
	Stratiomyidae	Soldier flies
Infraorder Xylophagomorpha	None	None
Infraorder Stratiomyomorpha	Stratiomyidae	Soldier flies
Cyclorrhapha	Syrphidae	Flower flies, hover flies
Platypezoidea	Phoridae	Humpbacked flies
Schizophora	Chloropidae	Chloropid flies, eye gnats
Tephritoidea	Piophilidae	Skipper flies
Ephydroidea	Drosophilidae	Small fruit flies, vinegar flies
Calyptratae	Muscidae[a]	Houseflies, stable flies, and allies
	Glossinidae[a]	Tsetse
	Hippoboscidae[a]	Louse flies
	Nycteribiidae[a]	Spider-like bat flies
	Streblidae[a]	Bat flies
Oestroidea	Oestridae[a] (including Cuterebrinae, Gasterophilinae, and Hypodermatinae)	Botflies, warble flies
	Calliphoridae[a]	Blowflies
	Sarcophagidae[a]	Flesh flies

[a]*Families are addressed in separate chapters.*

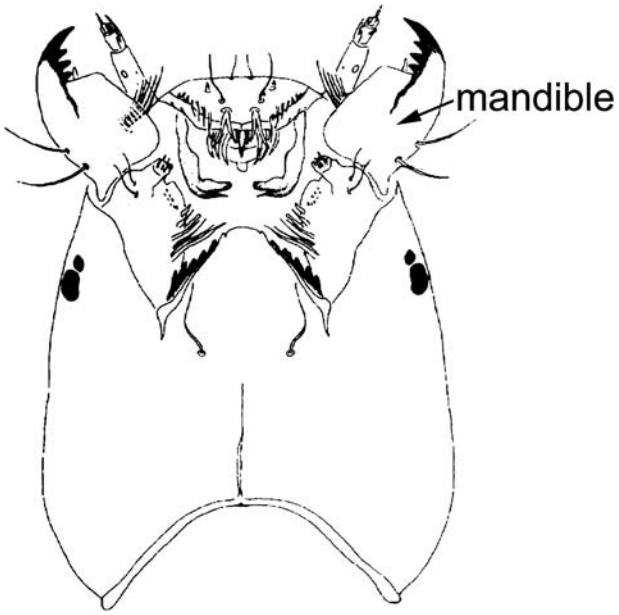


FIGURE 11.1 Representative lower dipteran head capsule with opposable mandibles; Chironomid midge (Chironomidae), ventral view. *Redrawn from Merritt and Cummins, 1996.*

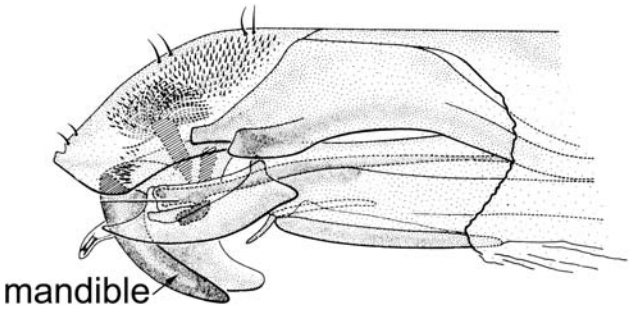


FIGURE 11.2 Lateral view of anterior part of *Tabanus marginalis* larva (Tabanidae) showing incomplete head capsule, and vertical, fang-like mouth hook. *From McAlpine et al., 1981b.*

maggot is broader and bears the posterior **spiracular plates** (Fig. 11.3E); like the cephalopharyngeal skeleton, they often are valuable for identification. The segments of the maggot typically bear spines in regular patterns (Fig. 11.3D) and the larvae of some species may possess structures that vary from simple setae to large protuberances. Others, such as cattle grubs and botflies, are rounded and robust, and their cuticle is frequently armed with stout spines. They range up to several centimeters in length.

Adults of the lower Diptera possess elongate, filamentous antennae composed of six or more segments

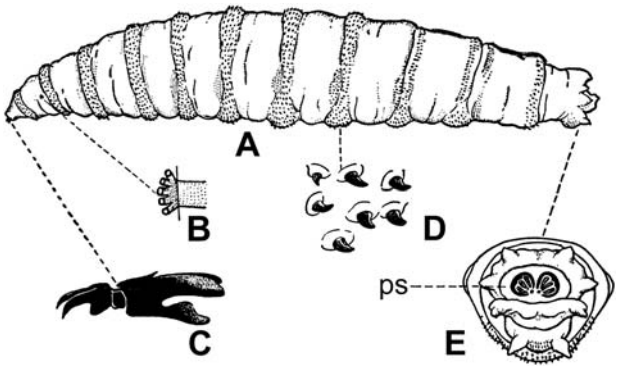


FIGURE 11.3 Blowfly larva, *Chrysomya bezziana* (Calliphoridae). (A) Complete larva; (B) anterior spiracle; (C) cephalopharyngeal skeleton; (D) spines; (E) caudal end with pair of spiracular plates (ps). *From James, 1947.*

(Fig. 11.4A and B). The antennae usually are longer than the length of the head and thorax combined. A notable exception is the family Simuliidae, in which the antennae are short and compact (Fig. 11.4B). In those groups that feed on blood, only the females display this behavior, doing so by means of piercing-sucking mouthparts as in mosquitoes.

Tabanomorpha adults are characterized by having relatively short antennae bearing a terminal **annulus**, or **stylus** (Fig. 11.4C). In general, these are large, robust flies. Like the lower Diptera, only the females feed on blood. Members of the Tabanidae are good examples; they are typically large, active flies whose females aggressively pursue blood meals. Their **mouthparts** are adapted for lacerating skin to feed on blood that pools at the wound site.

Cyclorrhapha adults have antennae that are **aristate**, bearing a large dorsal bristle (**arista**) on the apical antennal segment (Fig. 11.4D). Diptera in the **Schizophora** have a frontal suture or lunule (Fig. 11.5); this group includes a large number of species generally known as the **muscoid flies**. The Schizophora is perhaps the most taxonomically complex group of Diptera. Members of the **Acalyptratae**, the acalyptrate muscoid flies, lack a dorsolateral seam on the second antennal segment, whereas this seam is present in the **Calyptratae**, the calyptrate muscoid flies (Fig. 11.4D). Calyptrate muscoid flies possess posterobasal wing lobes called **calypters** (Fig. 11.6) that cover the halteres. Included in the Calyptratae are the hippoboscoid flies, which are sometimes secondarily wingless.

The mouthparts of blood-feeding cyclorrhaphan adults are of the piercing-sucking (stylate haustellate) type. In contrast to other Diptera, both male and female Calyptratae

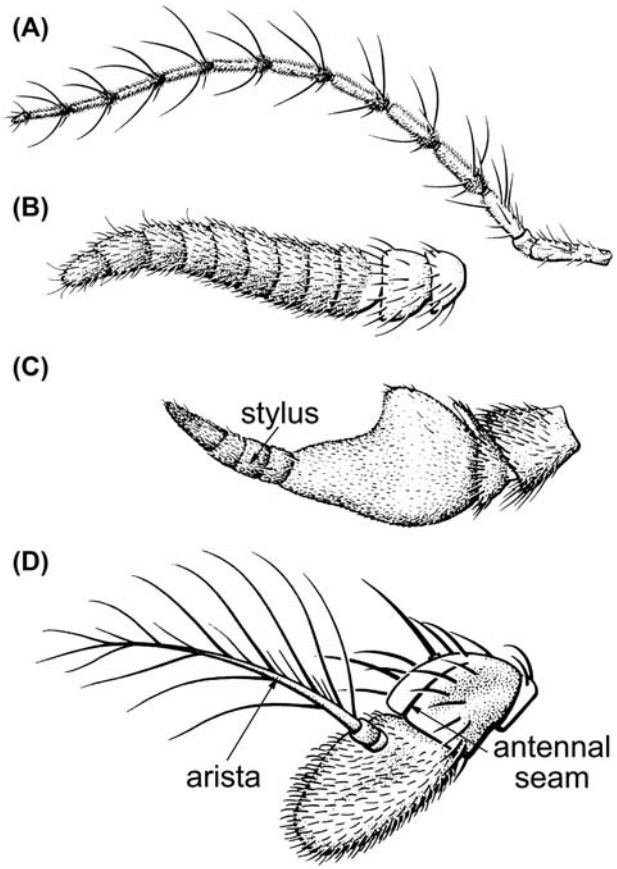


FIGURE 11.4 Antennae of adult flies. (A) Tipulidae (*Tipula*); (B) Simuliidae (*Cnephia*); (C) Tabanidae (*Tabanus*); (D) Drosophilidae (*Drosophila*). *From McAlpine et al., 1981b.*

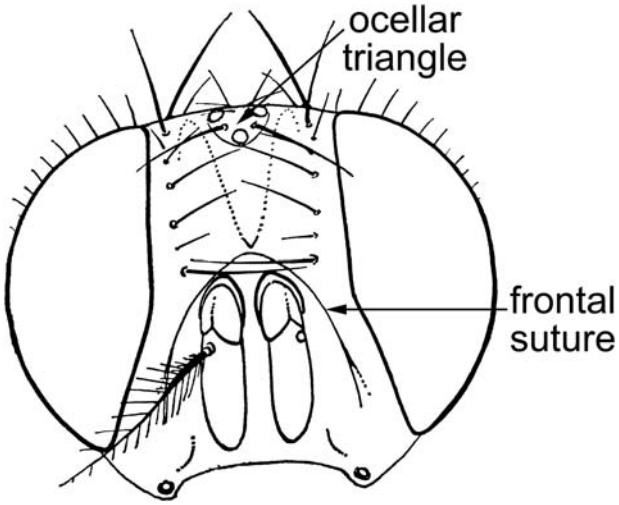


FIGURE 11.5 Frontal view of head of female fly showing frontal suture and ocellar triangle at vertex. *From Greenberg, 1971.*

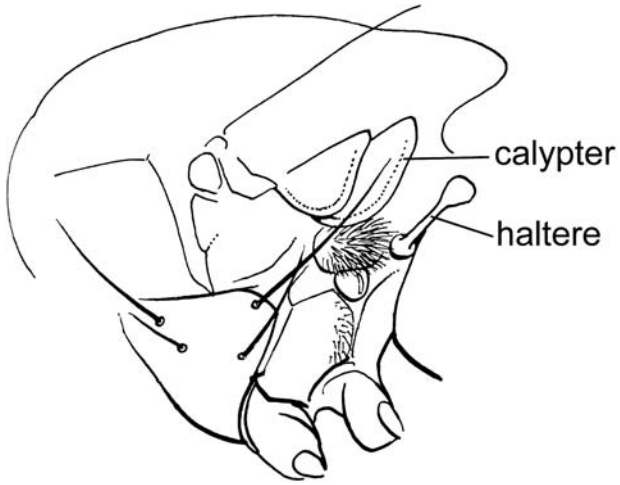


FIGURE 11.6 Calypterate fly showing haltere and calypters. *From Greenberg, 1971.*

suck blood in those species that exhibit this feeding style (e.g., horn flies and stable flies). Other species generally possess mouthparts that permit liquid food materials to be lapped or sponged. The latter type of mouthparts in some species have structures sclerotized enough to scarify tissue during feeding activities (e.g., face fly).

The functional pair of wings in the Diptera arises from the mesothorax. The metathoracic wings are modified to form a pair of knobbed balancing organs known as **halteres** (Fig. 11.6). The wing venation is highly variable between groups and provides valuable taxonomic characters for distinguishing the families. Many dipteran adults have characteristic wing patterns, including species of biting midges, deerflies, and horseflies.

The adults of most Diptera possess distinct compound eyes; ocelli are present in a triangle on the vertex of many species (Fig. 11.5). Adults are identified easily to sex, because most species exhibit some degree of sexual dimorphism. Males of families in the lower Diptera often possess densely plumose antennae and the females of bloodsucking species bear stylet-like mouthparts. The eyes of brachyceran males typically meet along the dorsal midline of the head **(holoptic)**, whereas the eyes of females are more widely separated **(dioptic)**. The female abdomen ends in an ovipositor (larvipositor in some species), whereas the male abdomen typically bears distinct genitalia at the terminus. In the males of some lower Diptera and Brachycera, the genital segments rotate one-half turn shortly after the adult fly emerges; thus, the genital capsule appears "upside down" in adults of those species. In the Schizophora, this rotation continues through a full circle, so that the genital capsule is in

its normal position. A morphological approach to identification that has proven useful in the Diptera, particularly with the Cyclorrhapha, is the characteristic appearance of male genitalia. In many species the aedeagus, claspers, and associated structures are unique. Dissection of male genitalia is a technique used by dipterists that permits detailed examination of the genital structures. Descriptions of species in some families such as the Sarcophagidae are based in large part on male specimens.

McAlpine (1981a) and Teskey (1981a) present comprehensive reviews of the morphology and related terminology of dipteran adults and larvae, respectively.

LIFE HISTORY

The Diptera are holometabolous. Most dipteran females lay eggs and are thus oviparous. Others are ovoviviparous, hatching their eggs internally and thus producing motile early-instar larvae. Such flies are called larviparous and are represented by flesh flies (Sarcophagidae). In a few dipteran groups, the developing larvae are retained within the female's body until they are ready to pupate. These flies are called pupiparous and include the louse flies (Hippoboscidae) and tsetse flies (Glossinidae). The number of offspring produced per female by larviparous and pupiparous species is low compared with oviparous and ovoviviparous species.

Many dipteran species inhabit aquatic or semiaquatic environments during their immature stages. Typical examples are mosquitoes, blackflies, and most horseflies and deerflies. The females of many of these hematophagous flies are capable of producing an initial batch of eggs before obtaining a blood meal, known as **autogeny**. In contrast, those species that must feed on blood before they produce eggs are referred to as **anautogenous**.

Whereas the number of larval instars varies within the Diptera, it remains generally constant for a given species. Mosquitoes and most other lower Diptera have four larval instars, whereas most cyclorrhaphan Diptera pass through three observable larval instars, with a fourth instar, the **prepupa**, occurring cryptically inside the pupal case. The cyclorrhaphan instars usually can be distinguished morphologically: the first instar lacks anterior spiracles and generally has only one slit in each caudal spiracular plate; second and third instars bear anterior spiracles and have two and three slits, respectively, in the caudal spiracular plate. These slits are lacking in some groups; instead, the spiracular plate has many small openings (e.g., cattle grubs).

The pupae of lower Diptera are **obtect**, with the appendages and other external body structures of the developing adult being discernible externally. Pupae are typically immobile; significant exceptions are the pupae of mosquitoes and a few other lower dipteran families that can move by means of caudal paddles. The Brachycera have **coarctate** pupae, in which the pupa is encased within the hardened exuviae of the penultimate larval instar. The latter structure, called a **puparium**, is most frequently brown and is often said to resemble a "pill." It retains many morphological features of the larval integument. Adult flies emerge from the pupal case by employing hydrostatic pressure from hemolymph to generate splits along predetermined lines. The head of most Schizophora has an eversible sac, the **ptilinum**, which facilitates the fly's escape from the puparium. After emergence, the ptilinum retracts through a fissure proximate to the lunule at the antennal bases.

BEHAVIOR AND ECOLOGY

An aspect of fly behavior of particular interest to medical and veterinary entomologists is the host-finding capabilities of blood-feeding species. Although various mechanisms have been described, they generally fall into two categories: olfactory and visual. A common olfactory cue used by blood-feeding insects, including many true flies, is the relative titer of carbon dioxide (CO_2) in the atmosphere surrounding or downwind from the host. If the goal of a mobile parasite is to locate a warm-blooded animal, exhaled CO_2 can serve as a cue for recognizing and locating potential hosts. A practical result is the widespread use of dry ice or bottled CO_2 to improve trapping success for common blood-feeding flies such as mosquitoes, blackflies, and no-see-ums. Other chemicals (e.g., mercaptans, octenol, and lactic acid) are used as olfactory cues by certain species. The principal means by which most insect repellents work is by inhibiting olfactory perception, thereby disrupting normal host-seeking behavior.

Visual host-finding cues are employed effectively by some flies, notably, the Tabanidae. Entomologists have not been able to prove conclusively what horseflies and deerflies actually "see," but there is little question that blood-seeking females are sensitive to blackbody radiation outside the spectrum visible to humans. It has been theorized in some cases that such females sense warmth against a cool background, in the manner that thermal-vision cameras are able to scan houses for heat leaks, crops for disease-induced stress, and nocturnal battlefields for invading personnel. The shape and size of hosts also may be important to some flies because they visually recognize

or orient to certain host animals. In many instances, olfactory and visual cues presumably complement each other.

Another important aspect of fly behavior is the female's ability to identify an environment suitable for development of her offspring. As with host-finding behavior, olfaction can have an important role. Necrophilous blowflies and flesh flies appear quickly after an animal dies; olfaction is almost certainly their major cue, even though the odor may not be detectable by humans. Similarly, face flies appear at cattle dung pats almost immediately after cattle defecate. Most flies, in common with many other types of insects, can perceive chemical cues at a level many orders of magnitude greater than that of humans. The females of other dipteran species similarly locate appropriate breeding sites. As examples, salt marsh mosquitoes and tree-hole mosquitoes must select aquatic habitats suitable for their eggs. The females of some blowfly and flesh fly species are highly attracted to human feces, and female screwworms are readily drawn to sores or wounds on living hosts.

Flies of medical and veterinary importance afford excellent examples of both *K*- and *r*-strategies in their life history. A few dipterans are known as ***K*-strategists**, typified best by tsetse, sheep keds, and other related families. The symbol *K* represents the carrying capacity of the environment. These flies have longer life cycles, produce fewer offspring, and are particularly influenced by density-dependent mortality factors. More common are pest fly ***r*-strategists**, in which large numbers of offspring are produced, each individual with a relatively small chance of survival. The symbol *r* denotes the instantaneous rate of increase for a population. These flies typically exhibit rapid growth, short life cycles, and high mortality attributable mainly to density-independent factors. Houseflies and other filth-breeding species, as well as mosquitoes, serve as good examples.

FAMILIES OF MINOR MEDICAL OR VETERINARY INTEREST

The major families of Diptera of medical–veterinary importance are treated in separate chapters of this book. The following discussion is provided for 13 other families of minor medical–veterinary importance, which include species that can cause problems for humans and other animals.

Tipulidae (Crane Flies)

Adults are slender-bodied flies 5–60 mm in length. They have long stilt-like legs, lack ocelli, and have a V-shaped mesonotal suture (Fig. 11.7A). Many species are attracted to light and readily enter houses, where they may be mistaken for large mosquitoes. Some are known to feed on nectar, but there is only one report of tipulids apparently attempting to bite a human (McCrae, 1967). Tipulid larvae have a distinct head capsule that can be retracted into the anterior thoracic segments (Fig. 11.7B). They are found in a wide range of aquatic and semiaquatic habitats and are commonly collected at the margins of streams and ponds and in moist leaf litter. A few species occur in dry soil, where the larvae may be pests of grain and turf crops by feeding on the roots. Most species in temperate areas have one or two generations a year, with four larval instars and a brief pupal stage. The length of the life cycle varies from six weeks to four years; the latter is typical of some Arctic species.

The Tipulidae are a large, cosmopolitan family of Diptera with over 60 genera and 1,500 species described in North America. Keys to both the adults and larvae of the Nearctic genera are provided by Alexander and Byers

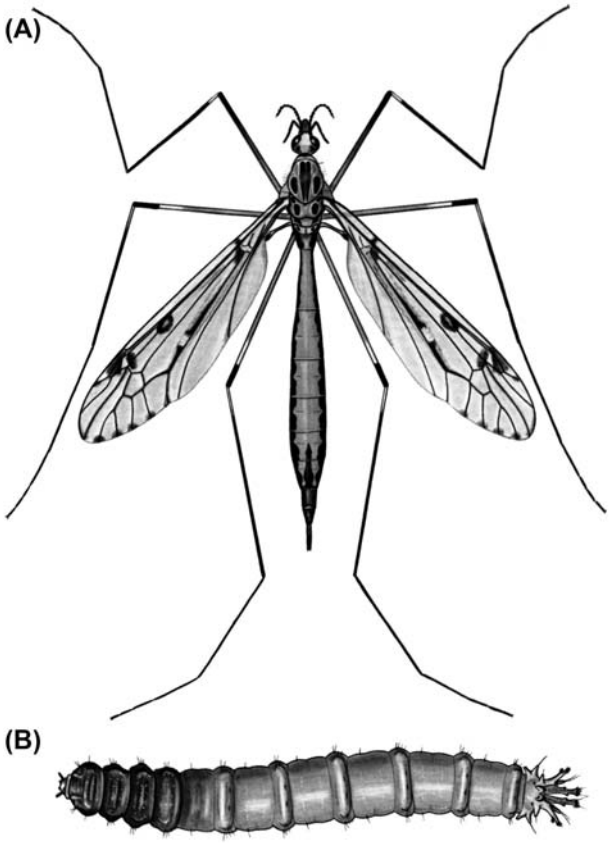


FIGURE 11.7 Tipulidae (*Tipula*). (A) Adult; (B), larva. *From McCafferty, 1981.*

(1981) and Byers (1984). Adult and larval ecology are presented in Knizesk and Sullivan (1984) and Freeman (1967), respectively.

Bibionidae (March Flies)

March flies are dark-colored flies varying in size from small to moderately large (4−10 mm). The adults (Fig. 11.8) generally can be distinguished from other lower Diptera by the lack of a V-shaped suture on the mesonotum, the presence of ocelli, antennae inserted below the eyes, and the presence of tibial spurs and pulvilli. Adults usually emerge in the spring and feed on flower nectar and pollen. The larvae are scavengers and are found mostly in decaying organic materials such as forest litter, manure, and soils rich in humus. Some species cause damage to the roots of cultivated plants, especially cereal and grass crops.

Adults of the **lovebug** (*Plecia nearctica*) (Fig. 11.8) often emerge in large swarms along the Gulf and South Atlantic coasts of the United States mainly during May and Sept. Larvae are found in aggregations under moist, decaying materials including leaves, grass clippings, Spanish moss, and manure. Adults are most often seen as copulating pairs and may remain *in copula* for several days, even while feeding together on flowers. When locally abundant, flying pairs can pose a hazard to automobile travelers by obscuring vision, clogging radiators, and occasionally damaging automobile finishes. Large flights occur in the United States along the Gulf Coast of Florida westward to Louisiana and eastern Texas and southward to Central America (Denmark and Mead, 1992). *Plecia nearctica* also occurs in large numbers along the Atlantic Coast of Georgia and southern South Carolina. Taxonomic keys for the larvae and adults of the six North American bibionid genera are provided in Hardy (1981). Denmark and Mead (1992) provide keys and review the biology and ecology of Nearctic *Plecia*.

Sciaridae (Dark-Winged Fungus Gnats)

Dark-winged fungus gnats are 1−11 mm in length and closely resemble the Mycetophilidae except that their eyes meet above the base of the antennae. The adults (Fig. 11.9A) are usually encountered in moist, shady habitats. The larvae (Fig. 11.9B) feed on a wide range of materials including fungi, decaying plants, manure, and, in some cases, the roots of greenhouse plants, soybeans, and clovers. *Lycoriella ingenua* is a major pest of commercial mushrooms, feeding on compost and all stages of mushrooms. *Bradysia* species are known to infest greenhouses, where they damage plant roots and consume fungi in potting soil; they also transmit spores of plant-parasitic fungi of the genus *Pythium*. There are four larval instars. The adult-to-adult life cycle lasts 15−49 days in some economically important species. Like the mycetophilids, the sciarids may emerge inside houses from ornamental plantings and potted plants.

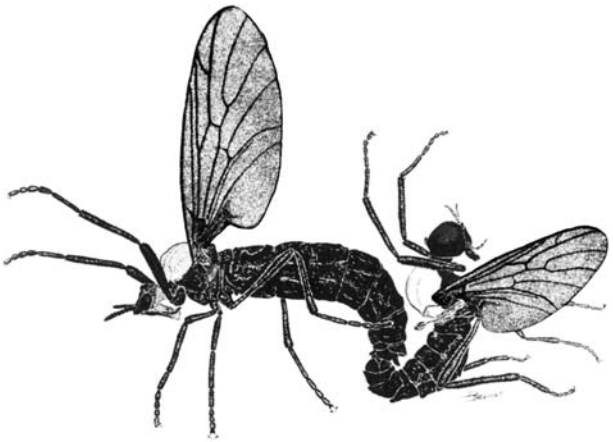


FIGURE 11.8 Bibionidae, lovebug (*Plecia nearctica*); pair of adults in copula, female at left. *From Leppla et al., 1975.*

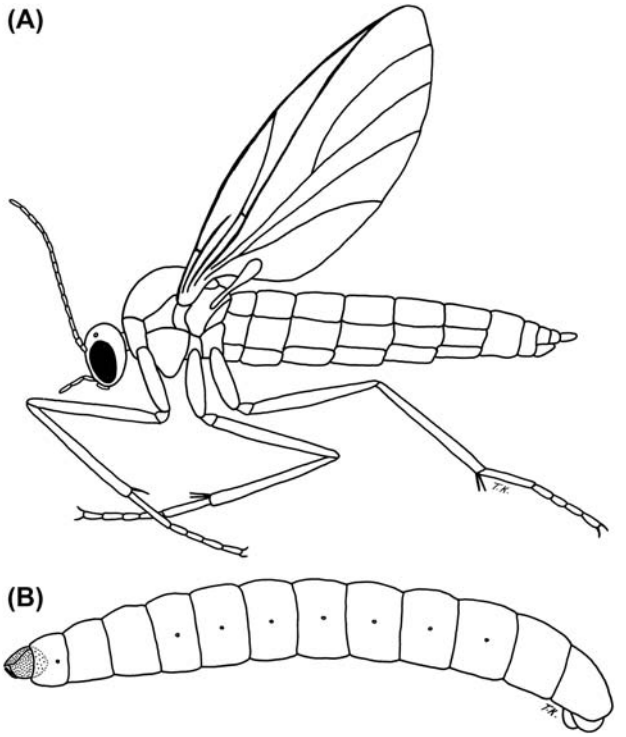


FIGURE 11.9 Sciaridae, dark-winged fungus gnat. (A) Adult female; (B) larva. *Original by Takumasa Kondo.*

Sciarids pose no medical problems except for rare reports of household pets becoming ill after eating adult flies. In one Florida case, a four-month-old dog died after ingesting large numbers of an unidentified sciarid species during an unusually large emergence in early May. The dog exhibited seizures and shock and was comatose by the time it was seen by a veterinarian. The dog died a short time later after experiencing extensive internal hemorrhaging and hepatic toxicosis. Examination of the stomach contents revealed several hundred sciarid adults (G.R. Mullen, personal communication).

There are at least 166 Nearctic species (Mohrig et al., 2012). Keys to some genera of adults and larvae are found in Steffan (1981). The ecology of some species is presented in Madwar (1937).

Chaoboridae (Phantom Midges)

Adults are small (1.4−10 mm in length), mosquito-like midges without the elongate proboscis and abundant wing scales characteristic of the Culicidae (Fig. 11.10A). There are 14 species in three genera in North America (Borkent, 2014). The transparent larvae (Fig. 11.10B) are aquatic and are found commonly in lentic habitats (e.g., large lakes, small pools, bogs, small ponds). The larvae of all North American species are predators that grasp their prey with prehensile antennae. Prey include small crustaceans and aquatic insect larvae.

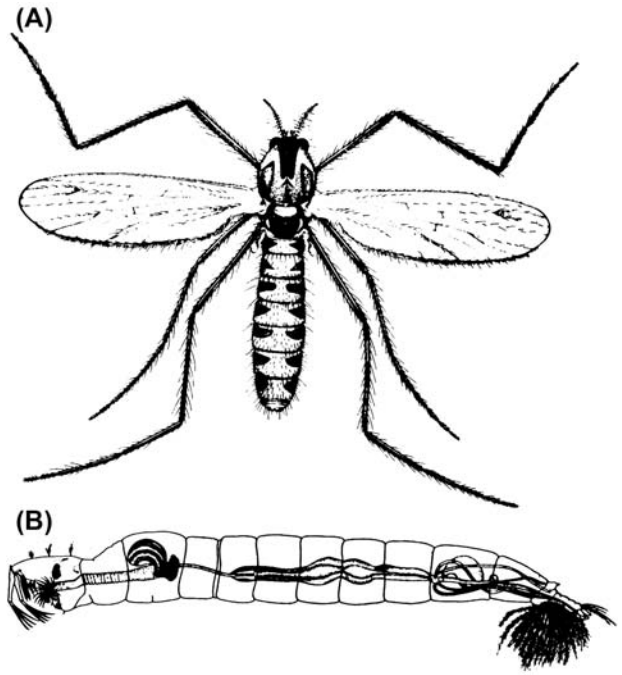


FIGURE 11.10 Chaoboridae, the Clear Lake gnat (*Chaoborus astictopus*). (A) Adult female; (B) larva (Herms, 1937). *Copyright: Regents of the University of California; used with Permission.*

The **Clear Lake gnat** (*Chaoborus astictopus*) is an inhabitant of large lakes and impoundments in the western United States. Large numbers emerge synchronously in the spring and are attracted to lights in residential and resort areas, where they can be annoying (Herms, 1937; Linquist and Deonier, 1942). In Clear Lake itself, numbers of gnats have declined over the years after the introduction of predatory fish to the lake (S.F. Cook, 1981). Generic keys for chaoborid larvae and adults are found in E.F. Cook (1981).

Corethrellidae (Frog-Biting Midges)

Adults are small (0.6−2.5 mm in length) midges superficially resembling mosquitoes but lacking the elongate proboscis and abundant wing scales (Fig. 11.11B). Eggs of the only genus, *Corethrella*, are laid on the surface of water and hatch within 2−4 days. The larval stage averages 15−32 days and the pupa is active and lasts 3−6 days. The transparent larvae (Fig. 11.11A) are aquatic and are found commonly in lentic habitats (e.g., large lakes, small pools, bogs, small ponds). The larvae are predators that grasp their prey with prehensile antennae. Prey include small crustaceans and aquatic insect larvae, including mosquitoes (Fig. 11.11A), which they sometimes eliminate from restricted habitats.

Females have toothed mandibles and have been found with avian and mammalian blood in their digestive tracts (Williams and Edman, 1968). *Corethrella brakeleyi* and *Corethrella wirthi* have been observed feeding on tree frogs (*Hyla* spp.) (McKeever, 1977). Together, these two species of corethrellids use at least seven species of frogs as hosts, including true frogs, tree frogs, and cricket frogs (Camp and Irby, 2017). *Corethrella* females are attracted to the calls of male tree frogs (McKeever and French, 1991), to which *C. wirthi* can transmit *Trypanosoma wirthi* (Johnson et al., 1993). Often, large numbers of *Corethrella* females will feed on the same frog (Fig. 11.11C). They do not feed on female frogs that do not call. Keys to United States species of *Corethrella* are found in Stone (1968) and Borkent (2008).

Chironomidae (Chironomid Midges)

Adult chironomid midges (Fig. 11.12A) are 1−10 mm long with slender legs, narrow scaleless wings, and plumose antennae in the adult males. They are often mistaken for adult mosquitoes but lack the long proboscis and are unable to feed on blood. Adults are short-lived, living only a few days to several weeks. Some imbibe honeydew and other

FIGURE 11.11 Corethrellidae. (A) Larva feeding on an early-instar mosquito larva; (B) Adult female *Corethrella brunnea*; (C) Adult females of *Corethrella mitra* feeding on a frog (*Limnonectes leporinus*), Sabah, Malaysia. *(A) Photograph by James Newman; (B and C) from Borkent and Grafe, 2012; (C) Photograph by Oliver Konopik.*

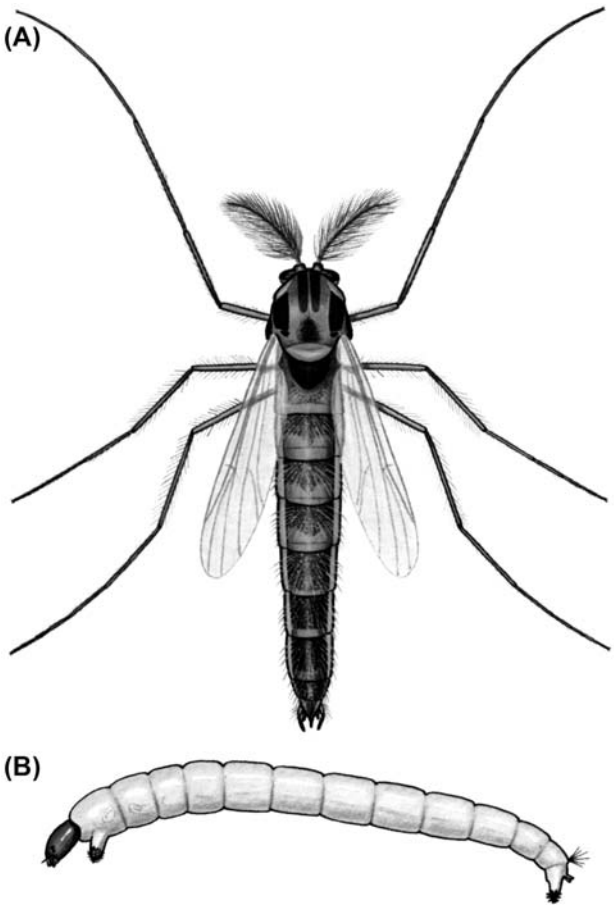


FIGURE 11.12 Chironomidae. (A) Adult male (*Chironomus* sp.); (B), larva (*Pseudodiamesa* sp.). *From McCafferty, 1981.*

natural sugars, but some take no food at all as adults. Most chironomid larvae are aquatic or semiaquatic and construct tubes in or attached to the substrate. They are often the most abundant benthic organisms and occur in all types of habitats including rivers, streams, lakes, ponds, water supplies, and sewage systems. Chironomid larvae are cylindrical and have paired prolegs on the prothoracic and last abdominal segments (Fig. 11.12B). The head is heavily sclerotized and nonretractile. They have no spiracles. Many species, however, have a hemoglobin-like substance in their hemolymph and are called **bloodworms** because of their pink or red color. Most species are detritus feeders that graze on aquatic substrates. Others filter drifting food particles from the water with strands of saliva or are predators on other chironomid larvae or oligochaete worms.

In addition to being mistaken for adult mosquitoes, chironomids can pose other medical and economic problems. Inhabitants of localities where large, synchronous emergences occur can develop allergies to the larval hemoglobin that is carried over from the larva to the adult and becomes airborne as the bodies of the adults decompose (Cranston, 1988). Larval hemoglobin also can induce allergies in workers who process bloodworms into fish food for aquaria. Large chironomid emergences from polluted bodies of water are common and may cause local annoyance to humans in addition to economic damage to machinery, paint finishes, automobiles, and airplanes (Ali, 1991). Several serogroups of *Vibrio cholerae*, the bacterium responsible for cholera, have been isolated from chironomid egg masses and from the cuticle of adults. This suggests that they may be involved in maintaining and moving *V. cholerae* in and between bodies of water (Broza et al., 2005, 2008). Large numbers of adult midges can discourage tourism and contaminate materials in food processing, pharmaceutical, and manufacturing plants. Larvae that occur in water-storage and water-distribution systems can pass through taps into homes (Bay, 1993).

The Chironomidae are a large family distributed worldwide with more than 130 genera and 700 species in North America (Oliver, 1981). Armitage et al. (1995) give an overall account of the biology and ecology of chironomids. Failla et al. (2015) review the ecological and public health impacts of chironomids.

Rhagionidae (Snipe Flies)

Adult snipe flies (Fig. 11.13A) are 4−15 mm in length with long legs, often spotted wings, and distal antennal flagellomeres forming a slender stylus (Fig. 11.13A). Most prey on other insects, except that females of *Symphoromyia* in western North America and *Spaniopsis* in Australia suck blood. Larvae (Fig. 11.13B) are predatory and are usually found near the surface of moist soil in meadows and steep, well-drained slopes usually associated with mosses, woodland grasses, willows, and/or alders.

In California (the United States), *Symphoromyia* adults are active from Apr. through mid Jul. They readily attack humans, deer, cattle, and horses, usually inflicting a painful bite around the head. Most of the species studied appear to be anautogenous and univoltine. Although they may be annoying to humans, livestock, and wildlife, they have not been implicated in the transmission of any disease organisms (Hoy and Anderson, 1978). In Yellowstone National Park (the United States), biting activity starts in early Jul. and continues until early Sept. Horses, mule deer, and humans are often attacked by swarms of females in localized areas along trails, with relatively fewer attacks outside these areas (Burger, 1995). Human responses to bites range from mildly annoying to very painful, with rare incidences of anaphylactic shock (Turner, 1979). Kerr (2010) provides keys to the genera of Rhagionidae. For further information on the taxonomy and biology of the genus *Symphoromyia*, see Turner (1974), James and Turner (1981), and Burger (1995).

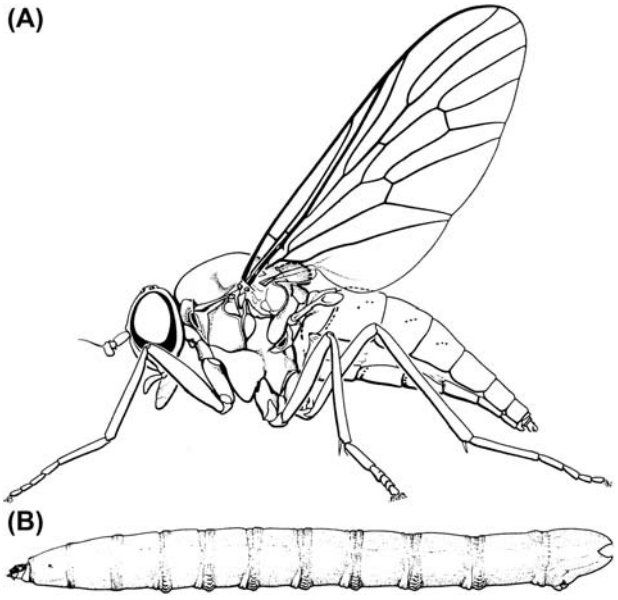


FIGURE 11.13 Rhagionidae. (A) Adult female (*Symphoromyia* sp.); (B) larva (*Rhagio* sp.). *From McAlpine et al., 1981b.*

Athericidae (Athericid Flies)

Adult athericids (Fig. 11.14A) are 7−8 mm long and resemble rhagionids. They differ from snipe flies by the presence of a strongly developed subscutellum, the R_1 cell being closed at the wing margin, and the absence of spurs on the foretibia. Larvae (Fig. 11.14B) inhabit flowing water, where they prey on other insect larvae. There is apparently one generation per year. Some adults prey on insects, but females of *Suragina* species are known to suck blood from humans, cattle, and some cold-blooded vertebrates (Hoy and Anderson, 1978). The family includes only six Nearctic species; all known species occurring in Texas (the United States) and Mexico. Of these, three belong to the blood-feeding genus *Suragina*. Keys to the North American species are provided in Webb (1977, 1981).

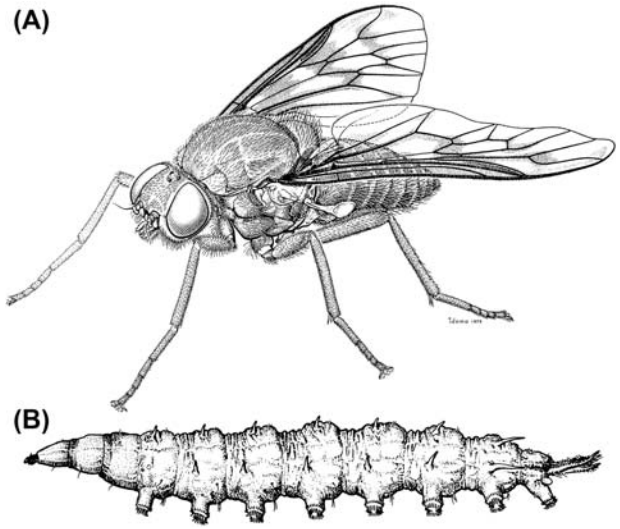


FIGURE 11.14 Athericidae (*Atherix*). (A) Adult female; (B) larva. *From McAlpine et al., 1981b.*

Stratiomyidae (Soldier Flies, Latrine Flies)

Adult soldier flies (Fig. 11.15A) vary from 2 to 20 mm in length. Their wings are distinctive in that they have all branches of the radius thickened and crowded toward the costal margin, ending before the apex of the wing. The body color may be yellow, green, blue, or black, and sometimes metallic. Many adults visit flowers, cattails, or other emergent aquatic vegetation. Larvae (Fig. 11.15B) are elongate and dorsoventrally flattened, and have a toughened or leathery integument with small, closely spaced calcareous tubercles. Many larvae are aquatic, living in a wide range of shallow, lentic habitats where they breathe at the surface through posterior spiracles. Others are terrestrial, breeding in animal wastes and decaying plants and animals, or in soil where they feed on roots of grasses.

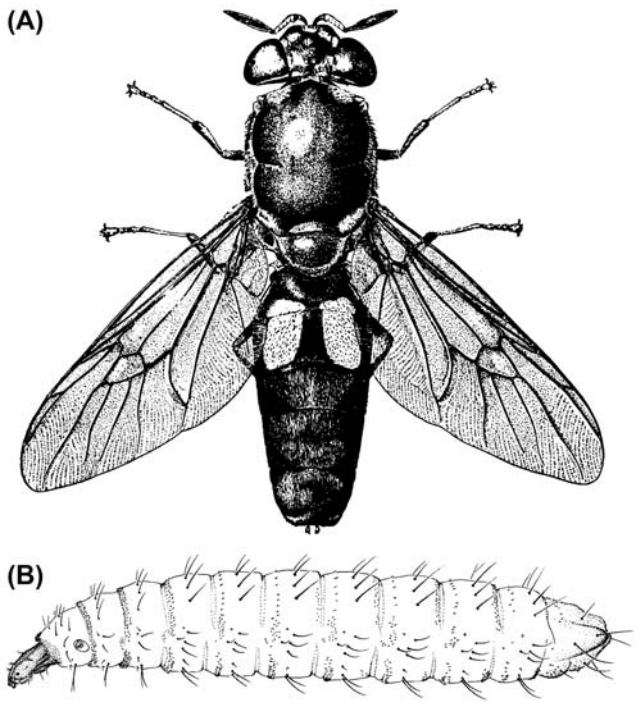


FIGURE 11.15 Stratiomyidae, black soldier fly (*Hermetia illucens*). (A) Adult female; (B) larva. *From Gagné, 1987.*

The **black soldier fly** (*Hermetia illucens*) (Fig. 11.15) is the stratiomyid best known to medical and veterinary entomologists and sanitary engineers. The adults are about 20 mm long, bluish-black, with yellowish white tarsi and two lateral, translucent spots on the second abdominal segment. Mature larvae are about 20 mm long, flattened dorsoventrally, and dull tan with a narrow head bearing eye spots. They develop in a broad spectrum of decaying materials including fruits, vegetables, human and animal wastes, and carrion. Eggs are laid in masses on the substrate and hatch in four days. The five larval instars last a total of about 14 days and pupation occurs inside the last larval integument, lasting about two weeks. Black solider fly larvae can become abundant in sewer processing plants with trickle filters, where they may be numerous enough to block the system. In caged-layer poultry manure, large populations of larvae can churn the manure and cause it to become liquefied and thus unsuitable for housefly larvae (Sheppard, 1983). In addition to helping to control houseflies, this process reduces the total volume of manure and populations of the pathogens *Escherichia coli* O157:H7 and *Salmonella enterica* serovar *enteritidis*. Mature larvae can also recycle food waste and are processed into animal food (Sheppard et al., 1994; Erickson et al., 2004; Nguyen et al., 2015). Larvae occasionally are eaten by humans in overripe fruit or undercooked meat, which can result in intestinal myiasis (James, 1947). James (1960, 1981) discusses the biology and provides keys to larvae and adults.

Phoridae (Humpbacked Flies, Scuttle Flies)

Adult phorids are 0.5−5.5 mm long with an enlarged thorax that gives them a characteristic humpbacked appearance (Fig. 11.16A and B). The hind femora are flattened and the major bristles of the head and legs are feathered. They run in short, quick bursts and are usually found in damp places near larval habitats. Larvae (Fig. 11.16C) are less than 10 mm long, lack an apparent head, and possess abdominal projections that range from being inconspicuous to large and plumose. Larval habitats are extremely varied. They include all kinds of decomposing plant and animal matter, fungi, bird nests, feces, dead insects, sewage treatment beds, and commercial mushrooms. Some larvae are internal parasitoids of other arthropods or live as commensals with social insects.

Megaselia scalaris (Fig. 11.16B) is the phorid of most medical importance. The female lays eggs in fruits and vegetables, feces, and decaying plant and animal matter. Sporadic cases of **facultative human myiasis** caused by *M. scalaris* have been documented in many areas of the world; they include cutaneous, pneumonic, nasal, gastrointestinal, urogenital, and ophthalmic myiasis (Carpenter and Chastain, 1992). Phorid larvae also are commonly associated with decomposing animal remains, where they tend to be late invaders after the calliphorid flies have pupated (Smith, 1986). This fly is often a problem around mausoleums and mortuaries, where the larvae develop in burial crypts, producing large numbers of adults

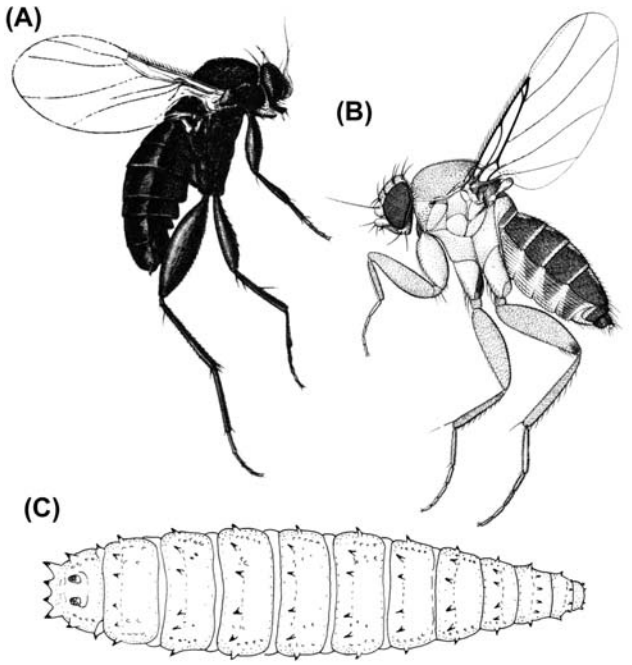


FIGURE 11.16 Phoridae. (A) Coffin fly (*Conicera tibialis*), adult female; (B) *Megaselia scalaris*, adult female; (C) larva (*Megaselia*). *From Smith, 1986.*

(Katz, 1987). A small, black European species called the **coffin fly** (*Conicera tibialis*) (Fig. 11.16A) is commonly associated with interred human remains that have been underground for up to a year (Smith, 1986).

There are about 350 species and 48 genera of phorid flies in North America. Keys to adults in the Nearctic region are provided in Peterson (1987). The biology, ecology, and keys for identification of Phoridae are included in Disney (1994).

Syrphidae (Flower Flies, Hover Flies)

Adults of this family vary in length from 4 to 25 mm and are distinguished by the presence of a spurious vein between the radius and media. Many are boldly marked with black and yellow transverse bands and are effective wasp mimics. Others, including *Eristalis* and *Eristalinus*, which are called **drone flies**, are covered with fine yellow hairs and resemble honeybees or bumblebees (Fig. 11.17A). Most adults are strong fliers and are often seen hovering near flowers where they feed on nectar. They neither bite nor are capable of stinging. Syrphid larvae are varied in form and feeding habits. Some are slug-like and live exclusively in nests of social insects. The most common larval forms are strongly flattened and are predaceous on aphids and other plant-feeding insects. The larvae of *Eristalis* and *Eristalinus* species are aquatic. They are known as **rat-tailed maggots** because of their long, retractable caudal segment bearing the posterior spiracles, which can be extended two to three times the length of the body (Fig. 11.17B). This extensible air tube allows the aquatic larvae to breathe air from the surface while inhabiting highly polluted water. Rat-tailed maggots, especially *Eristalis tenax*, are often found in manure-polluted water in and around confined livestock operations. They are common in wastewater treatment lagoons for livestock and human wastewater treatment facilities. Occasionally *E. tenax* larvae can cause **enteric pseudomyiasis**, **gastrointestinal**, or **urogenital myiasis** in humans. There are over 900 species and more than 90 genera of syrphids in the Nearctic Region (Vockeroth and Thompson, 1987).

Piophiliae (Skipper Flies)

Adult piophilids (Fig. 11.18A) are small (~5 mm in length), dark, acalypterate flies that are usually shiny black with strong black bristles. The vermiform larvae (Fig. 11.18B) live in a variety of dead plant and animal materials, including carrion, bones, hides, fungi, and stored food products of animal origin. The species most likely to come to the notice of medical or veterinary personnel is the cosmopolitan **cheese skipper**, *Piophila casei* (Fig. 11.18A). It is a pest of stored food, particularly

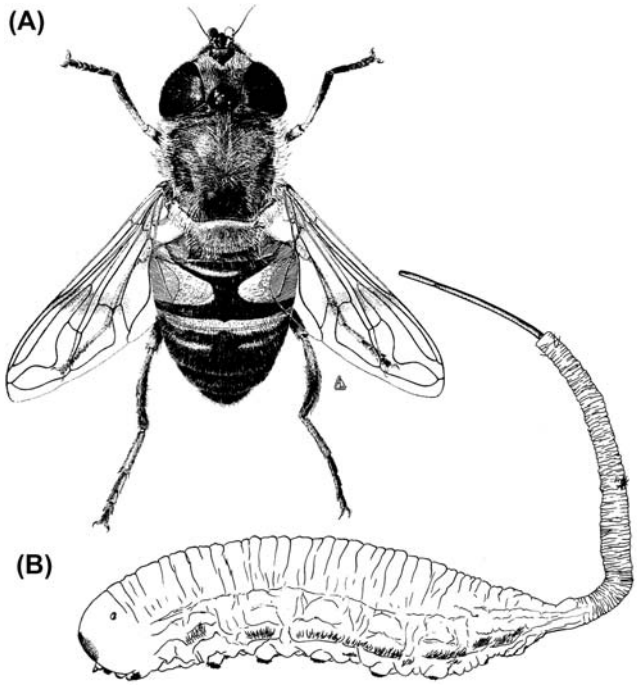


FIGURE 11.17 Syrphidae, drone fly (*Eristalis tenax*). (A) Adult female; (B) larva, rat-tailed maggot. *From Gagné, 1987.*

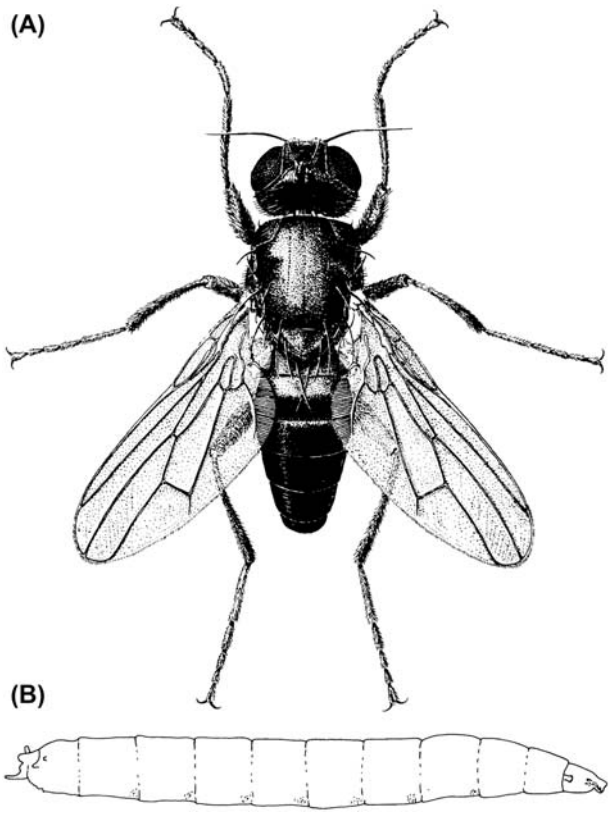


FIGURE 11.18 Piophilidae, cheese skipper (*Piophila casei*). (A) Adult female; (B) larva. *(A) From Gagné, 1991, (B) From Smith, 1986.*

cheeses and cured hams. The common name derives from the larva's ability to catapult itself into the air and assume an O-shape by seizing its anal papillae with its mandibles and abruptly releasing its hold. Cheese skippers that are consumed by humans in contaminated food have been responsible for numerous cases of **gastrointestinal myiasis** (James, 1947). Cheese skipper larvae (Fig. 11.18B) sometimes colonize corpses in situations in which the larger calliphorid and sarcophagid flies are denied access. There are 14 genera containing about 60 species in the Nearctic Region (McAlpine, 1987).

Drosophilidae (Small Fruit Flies)

Also commonly referred to as **vinegar flies**, these are generally small insects (1−6 mm) typically with red eyes. The adults (Fig. 11.19A) are found around the larval habitats of decaying vegetation, plant sap, fungi, and ripe fruit. Larvae are maggot-like with stalked posterior spiracles (Fig. 11.19B). Most feed on yeast and other microorganisms in the decaying substrate. Some are leaf miners whereas others are parasitoids or predators of Homoptera.

Drosophilids are familiar in most households, flying around or crawling on overripe fruit. *Drosophila melanogaster* is a common laboratory animal used extensively in genetic research. Although the flies are generally harmless, some species (especially *Drosophila repleta*) are a potential means for mechanical transmission of pathogens when they breed in animal feces (Greenberg, 1973; Harrington and Axtell, 1994). Males of *Phortica variegata* feed on ocular secretions and have been incriminated as vectors of *Thelazia callipaeda*, an eye worm, in Europe. This is the only known instance of transmission of a vertebrate pathogen strictly by male arthropods (Otranto et al., 2006). *Drosophila* species occasionally are found in the putrid effluents from corpses. *Drosophila funebris* has been reported to cause **intestinal myiasis** in humans (James, 1947). There are 17 genera and approximately 175 North American species (Wheeler, 1987).

Chloropidae (Grass Flies, Eye Gnats)

Adults (Fig. 11.20A) are small (1.5−5 mm in length) with few large bristles and a prominent break in the costal vein of the wing just mesad of the subcostal junction. Many adults are commonly found in grasses and other low vegetation, or visiting flowers. Larvae (Fig. 11.20B) lack an apparent head and have posterior spiracles and palmate anterior spiracles. Most larvae are phytophagous, feeding on stems, roots, and root hairs of grasses. The frit fly (*Oscinella frit*) and the wheat stem maggot (*Meromyza americana*) are important agronomic pests of grain crops. Other species are saprophytes, feeding mostly on decaying vegetable matter in soils, whereas a few are predators or gall formers.

Others are known as **eye gnats**, including *Liohippelates* species in North and South America and *Siphunculina* species in Asia. Eye gnats are attracted to humans and other mammals, where they hover about the face, body orifices,

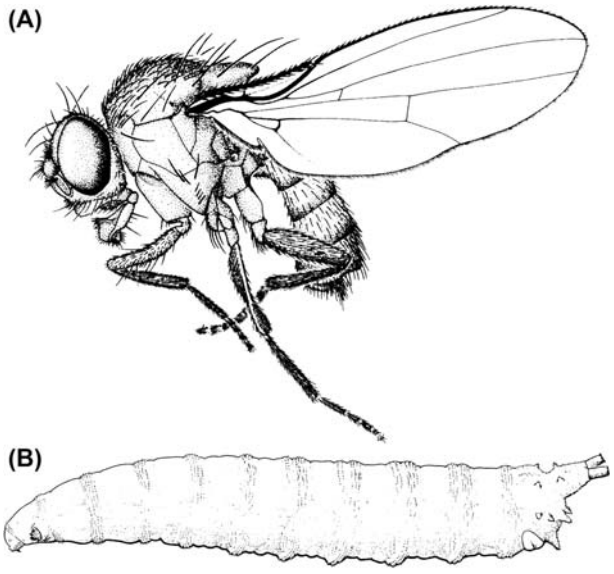


FIGURE 11.19 Drosophilidae, *Drosophila melanogaster*. (A) Adult female; (B) larva. *(A) From Gagné, 1991, (B) From Wheeler, 1987.*

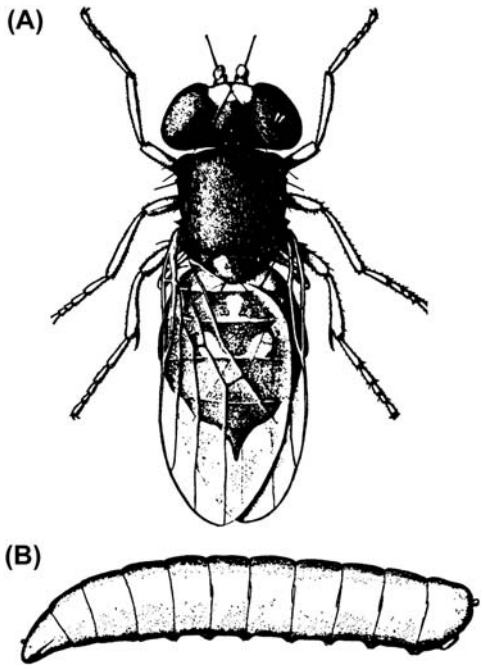


FIGURE 11.20 Chloropidae, eye gnat (*Liohippelates pusio*). (A) Adult female; (B) larva. *From Herms, 1939.*

and open wounds. Members of the genus *Liohippelates* (formerly included in the genus *Hippelates*) occur throughout much of North America (Sabrosky, 1980). Several species are particularly abundant in some of the sandy-soil regions of the southeastern United States (e.g., *Liohippelates pusio* and *Liohippelates pallipes*) and irrigated areas of southern California *(Liohippelates collusor*). The larvae feed on decaying organic matter in soil and can be particularly abundant in humus-enriched, cultivated soil or turf in sandy soils. The life cycle is about two weeks and there are multiple generations each year. The adults hover around the head of humans causing annoyance, especially when they fly into eyes, nostrils, or mouths. They also are commonly found on domestic animals, especially on areas soiled with urine or manure (Greenberg, 1971).

Liohippelates species have been implicated in the mechanical transmission of several organisms that cause diseases in humans and livestock. *Treponema pertenue*, the spirochete that causes **yaws**, has been shown to be transmitted by *Liohippelates flavipes* in Jamaica and other Caribbean and South American locales (Kumm, 1935). Human acute conjunctivitis (**pinkeye**) caused by several bacterial species, is noticeably more prevalent during outbreaks of *Liohippelates* in the United States and *Siphunculina* in the Orient (Dow and Hines, 1957; Greenberg, 1973). *Liohippelates* species also have been implicated in the spread of the causative organisms of **vesicular stomatitis** in livestock and streptococcal infections of human skin (Taplin et al., 1967; Francy et al., 1988). **Brazilian purpuric fever** is a fulminating, highly fatal bacterial disease of children caused by *Haemophilus influenzae* biotype *aegyptius*, which produces acute conjunctivitis in children (Harrison et al., 1989, 2008; The Brazilian Purpuric Fever Study Group, 1992). *Liohippelates puruanus* and *Hippelates neoproboscideus* have been implicated as mechanical vectors of *H. i.* biotype *aegyptius* (Tondella et al., 1994).

There are 55 genera and about 270 described species in the Nearctic Region (Sabrosky, 1987). Keys to *Liohippelates* species are provided by Sabrosky (1980). There are no effective area-wide methods for controlling *Liohippelates* species. Temporary relief from their annoyance is provided by protective head nets and insect repellents containing diethyltoluamide (DEET).

PUBLIC HEALTH IMPORTANCE

On a global scale, the Diptera are the most important order of insects affecting human health (Table 11.1). Mosquitoes are the foremost group because of their role as vectors of more pathogenic organisms than any other flies. The adverse impact of malaria, mosquito-borne arboviruses (e.g., yellow fever, dengue, and encephalitis), and metazoan infections such as filariasis on humans worldwide exceeds that of publicized ailments such as Lyme disease or AIDS. Other human-related vector−pathogen relationships involving the Diptera are exemplified by sand flies and sand-fly fever, bartonellosis, and leishmaniasis; by blackflies and onchocerciasis (river blindness); and by tsetse and African trypanosomiasis (sleeping sickness). On a global scale, about 270 million humans are infected with malaria, 90 million with lymphatic filariasis, 17 million with onchocerciasis, and 12 million with leishmaniasis. In total, almost 3.5 billion humans are rated at risk for fly-borne pathogens.

The Diptera also figure prominently in public health in regard to **filth flies**, which are associated with materials such as dung, carrion, and garbage. Houseflies, stable flies, and blowflies are a few examples. Beginning shortly after Pasteur's formulation of the germ theory in the late 1870s, the link between muscoid flies and human enteric problems was gradually understood. During the Spanish-American War (1898), this relationship became evident to U.S. troops in Cuba when "white-legged" muscoid flies roaming on food in mess tents were recognized as the same creatures that previously had been noted on lime-doused feces or corpses. In what was to become one of his major entomological contributions, Leland O. Howard directed public attention to such filth-breeding flies and almost succeeded in his quest to rename the housefly the "typhoid fly" (Howard, 1911). Many other microorganisms causing enteric disease, such as *Shigella* and *Entamoeba*, can be transmitted in a similar manner. The impact of filth flies on public health was particularly severe in the days before sanitary plumbing, effective pest management, and the ready availability of vaccines. Only the advent of the Salk vaccine for poliomyelitis in the 1950s halted long-term research on possible filth-fly involvement with the etiology of that disease.

Accounts of the enormous numbers of flies associated with corpses on battlefields are virtually as old as warfare itself; those from World War I in France and Belgium and from World War II in the Pacific region are particularly compelling. The beneficial use of certain necrophilous blowflies as **surgical maggots** stems almost directly from battlefield observations. Although **allantoin**, a natural antibiotic secreted by blowfly maggots, has been supplanted by more effective synthetic drugs, the use of surgical maggots remains an effective option for treating wounds, especially when they involve bone infections or drug-resistant bacteria that are refractory to blood-borne chemotherapy.

VETERINARY IMPORTANCE

The well-being of wild and domestic animals is directly affected by the Diptera in many ways (Table 11.2). As vectors of pathogens, flies are responsible for spreading

viruses such as those that cause bluetongue disease of sheep and cattle and hemorrhagic disease of deer; rickettsial infections such as anaplasmosis; protozoans such as those causing avian malaria; and metazoan infections such as canine heartworm. *Trypanosoma*-caused nagana vectored by tsetse has eliminated most animal agriculture throughout large areas of Africa.

In many parts of the world, infestations of living tissue with fly larvae, called **myiasis**, can be a problem for livestock and other domestic or wild animals. At one time, myiasis caused by screwworms constituted a major impediment to cattle, hog, and sheep production in the southern United States, Mexico, and Central America and was a major mortality factor among wildlife, especially deer. With the success of sterile-male releases and other antiscrewworm measures, this species is no longer a significant problem in North America. Other myiasis-causing flies in North America include cattle and reindeer grubs, sheep nose bots, deer and reindeer nose bots, rabbit and rodent bots, and stomach bots of horses and other equids.

In most localities, species of blowflies that cause myiasis have a direct summertime impact on most types of livestock and pets. These flies can invade wounds, sores, or body orifices such as unhealed navels of newborns, and vaginal tissues of postpartum females. The condition is perhaps best recognized by the sheep industry as **fly-strike** or **sheep strike** and has had an extensive impact, particularly in Australia and New Zealand.

Blood-feeding flies can affect the productivity and profitability of livestock operations by causing exsanguination and, in extreme cases, anemia. Although the biting rates of diurnal species are obvious to livestock producers (e.g., horseflies), those of crepuscular or nocturnal species are largely unappreciated. Livestock on pasture, or in production systems where large numbers of animals are artificially confined, may be subject to intense biting rates from flies. Bunching, kicking, and other avoidance behavior by animals under attack by biting flies can interfere with grazing time, feed consumption, and efficiency of energy conversion.

The enormous numbers of nonbiting flies associated with livestock and livestock facilities can constitute an annoyance factor. The feeding spots and fecal spots made by flies can create sanitary and aesthetic problems. In the poultry industry, **fly specks** on chicken eggs are a major economic problem. Flies produced in one location may affect animals in another. For example, stable flies emanating from cattle feedlots may emigrate to proximate farmsteads where they feed on pets and companion animals. Flies such as stable flies and eye gnats breeding adjacent to dog kennels may annoy dogs and other animals even at considerable distances from the breeding sites.

PREVENTION AND CONTROL

In addition to insecticidal applications against immature or adult flies, strategies for suppression of fly-caused problems typically include personal protection. Limiting access of flies to humans or other animals by window screening, mosquito netting, or other physical barriers remains one of the cheapest and most effective forms of control. The invention and ready availability of standard 16-mesh window screening, although considered mundane by most, should actually rank as a genuine marvel of the 20th century. Retreating behind such screening affords humans the opportunity to eat without fly-caused contamination and incessant biting by several species of blood-feeding flies. Similarly, chemical barriers to fly bites afforded by repellents are an important part of arthropod-borne pathogen management. Repellents are at the core of preventive medicine with respect to many fly-borne pathogens, especially in protecting military personnel. Typified best by mosquito repellents, these personal protectants include various formulations of DEET or picaridin. The insecticide permethrin has repellent activity when used as a clothing treatment. Repellents also are often applied to livestock in an effort to reduce biting rates of flies and the annoyance of other, nonbiting species.

A major emphasis of both civil and military contingency planning is preparation for the management of filth-fly populations in the event of natural or human-caused disasters. Proceeding from the axiom that, all other factors being equal, elimination of larval habitats will eliminate fly populations, a major goal of vector ecologists and sanitary engineers is to reduce the amount of substrate capable of supporting nuisance species. Examples of such environmental hygiene range from virtually replacing outdoor human privies and pit latrines with sanitary sewage systems in urban and suburban areas to instituting large-scale programs designed to facilitate effective manure management in areas where livestock are aggregated in large numbers.

Feces from cattle, hogs, and poultry are excellent larval habitats for many species of noxious flies, most importantly the housefly. Before the advent of automobiles, dung from horses was an important source of houseflies and stable flies. Dung mixed with other materials such as hay, straw, mud, and wood shavings is called manure. Like dung, manure is often an excellent larval habitat. A biologically inescapable by-product of livestock production is the generation of significant amounts of dung and manure. Houseflies in particular tend to disperse from their larval habitats, frequently invading surrounding neighborhoods where they stain and speck paint, cause annoyance, and may start local

fly populations. Modern systems of livestock production therefore depend on the effective removal of dung and manure; when these fail, the problem of "rural flies in the urban environment" often winds up in today's courts.

REFERENCES AND FURTHER READING

Alexander, C. P., & Byers, G. W. (1981). Tipulidae. In J. F. McAlpine, B. V. Peterson, G. E. Shewell, H. J. Teskey, J. R. Vockeroth, & D. M. Wood (Eds.), *Manual of Nearctic Diptera* (Vol. 1, pp. 153–190). Canada: Res. Branch, Agric. Monogr. No. 27.

Ali, A. (1991). Perspectives on management of pestiferous Chironomidae: (Diptera), an emerging global problem. *Journal of the American Mosquito Control Association, 7*, 260–281.

Amorim, D. S., & Yeates, D. (2006). Pesky gnats: Ridding dipteran classification of the Nematocera. *Studia Dipterologica, 13*, 1–6.

Armitage, P. D., Cranston, P. S., & Pinder, L. C. V. (1995). *The Chironomidae: Biology and ecology of non-biting midges*. New York: Chapman & Hall, 572 p.

Bay, E. C. (1993). Chironomid (Diptera: Chironomidae) larval occurrence and transport in a municipal water system. *Journal of the American Mosquito Control Association, 9*, 275–284.

Belkin, J. N., & McDonald, W. A. (1955). A population of *Corethrella laneana* from Death Valley, with descriptions of all stages and discussion of the Corethrellini (Diptera: Culicidae). *Bulletin of the Southern California Academy of Sciences, 54*, 82–96.

Borkent, A. (2008). The frog-biting midges of the world (Corethrellidae: Diptera). *Zootaxa, 1804*, 1–456.

Borkent, A. (2014). World catalog of extant and fossil Chaoboridae (Diptera). *Zootaxa, 3796*, 469–493.

Borkent, A., & Grafe, T. U. (2012). The frog-biting midges of Borneo – from two to eleven species (Corethrellidae: Diptera). *Zootaxa, 3279*, 1–45.

Borror, D. J., Triplehorn, C. A., & Johnson, J. F. (1989). *An introduction to the study of insects* (6th ed.). Philadelphia: Saunders, 875 p.

Broza, M., Gancz, H., Halpen, M., & Kashi, Y. (2005). Adult non-biting midges: Possible windborne carriers of *Vibrio cholerae* non-O1 non-O139. *Environmental Microbiology, 7*, 576–585.

Broza, M., Gancz, H., & Kashi, Y. (2008). The association between non-biting midges and *Vibrio cholerae*. *Environmental Microbiology, 10*, 3193–3200.

Burger, J. (1995). Yellowstone's snipe fly summer. *Yellowstone Science, 3*, 2–5.

Byers, G. W. (1984). Tipulidae. In R. W. Merritt, & K. W. Cummins (Eds.), *An introduction to the aquatic insects of North America* (4th ed., pp. 773–800). Dubuque, IA: Kendall/Hunt.

Camp, J. V., & Irby, W. S. (2017). Molecular confirmation of frogs (Anura) as hosts of Corethrellidae (Diptera) in the southeastern United States. *Journal of Insect Science, 17*(5), 1–3.

Carpenter, T. L., & Chastain, D. O. (1992). Faculative myiasis by *Megaselia* sp. (Diptera: Phoridae) in Texas: A case report. *Journal of Medical Entomology, 29*, 561–563.

Cole, F. R. (1969). *The flies of western North America*. Berkeley: Univ. of California Press, 693 p.

Cook, E. F. (1981). Chaoboridae. pp. 335–339. In J. F. McAlpine, B. V. Peterson, G. E. Shewell, H. J. Teskey, J. R. Vockeroth, & D. M. Wood (Eds.). *Manual of Nearctic Diptera* (Vol. 1, pp. 217–222). Canada: Res. Branch, Agric. Monogr. No. 27.

Cook, S. F. (1981). The Clear Lake example: An ecological approach to pest management. *Environment: Science and Policy for Sustainable Development, 23*(10), 25–30.

Cranston, P. S. (1988). Allergens of non-biting midges (Diptera: Chironomidae): A systematic survey of chironomid haemoglobins. *Medical and Veterinary Entomology, 2*, 117–127.

Denmark, H. A., & Mead, F. W. (1992). *Lovebug, Plecia nearctica Hardy (Diptera: Bibionidae). Entomology. Circular No. 350*. Fla. Dept. Agric. & Consumer Serv, 8 p.

Disney, R. H. L. (1994). *Scuttle Flies: The Phoridae*. London: Chapman & Hall, 467 p.

Dow, R. P., & Hines, J. D. (1957). Conjunctivitis in southwest Georgia. *Public Health Reports, 72*, 441–448.

Dusek, J. (1971). Key to larvae. In B. Greenberg (Ed.), *Flies and disease ecology, classification, and biotic Associations* (Vol. 1, pp. 163–199). Princeton, NJ: Princeton Univ. Press.

Erickson, M. C., Islam, M., Sheppard, C., Liao, J., & Doyle, M. P. (2004). Reduction of *Escherichia coli* O157:H7 and *Salmonella enterica* serovar *enteritidis* in chicken manure by larvae of the black soldier fly. *Journal of Food Protection, 67*, 685–690.

Failla, A. J., Vasquez, A. A., Fujimoto, M., & Ram, J. L. (2015). The ecological, economic and public health impacts of nuisance chironomids and their potential as aquatic invaders. *Aquatic Invasions, 10*, 1–15.

Foote, B. A. (1991). Order Diptera. In F. W. Stehr (Ed.), *Immature insects* (Vol. 2, pp. 690–699). Dubuque, IA: Kendall/Hunt.

Francy, D. B., Moore, C. G., Smith, G. C., Jakob, W. L., Taylor, S. A., & Calisher, C. H. (1988). Epizootic vesicular stomatitis in Colorado, 1982: Isolation of virus from insects collected along the northern Colorado Rocky Mountain front range. *Journal of Medical Entomology, 25*, 343–347.

Freeman, B. E. (1967). Studies on the ecology of larval Tipulinae (Diptera, Tipulidae). *Journal of Animal Ecology, 36*, 123–146.

Friedrich, M., & Tautz, D. (1997). Evolution and phylogeny of the Diptera: A molecular phylogenetic analysis using 28S rDNA sequences. *Systematic Biology, 46*, 674–698.

Furman, D. P., & Catts, E. P. (1982). *Manual of medical entomology*. Cambridge: Cambridge Univ. Press, 207 p.

Greenberg, B. (1971). *Flies and disease. Vol. 1. Ecology, classification and biotic associations*. Princeton, NJ: Princeton Univ. Press, 856 p.

Greenberg, B. (1973). *Flies and disease. Vol. 2. Biology and disease transmission*. Princeton, NJ: Princeton Univ. Press, 447 p.

Hardy, D. E. (1981). Bibionidae. pp. 217–222. In J. F. McAlpine, B. V. Peterson, G. E. Shewell, H. J. Teskey, J. R. Vockeroth, & D. M. Wood (Eds.). *Manual of Nearctic Diptera* (Vol. 1). Canada: Res. Branch, Agric. Monogr. No. 27.

Harrington, L. C., & Axtell, R. C. (1994). Comparisons of sampling methods and seasonal abundance of *Drosophila repleta* in caged-layer poultry houses. *Medical and Veterinary Entomology, 8*, 331–339.

Harrison, L. H., Da Silva, G. A., Pitmann, M., Fleming, D. W., Vranjac, A., Broome, C. V., et al. (1989). Epidemiology and clinical spectrum of Brazilian purpuric fever. *Journal of Clinical Microbiology, 27*, 599–604.

Harrison, L. H., Simonsen, V., & Waldman, E. A. (2008). Emergence and disappearance of a virulent clone of *Haemophilus influenzae* Serogroup *aegyptus*, cause of Brazilian purpuric fever. *Clinical Microbiology Reviews, 21*, 594–605.

Harwood, R. F., & James, M. T. (1979). *Entomology in human and animal health*. New York: Macmillan, 548 p.

Herms, W. B. (1937). *The clear lake gnat*. California Agricultural Expt. Stn., Bulletin 607.

Horsfall, W. R. (1962). *Medical entomology. Arthropods and human disease*. New York: Ronald Press, 467 p.

Howard, L. O. (1911). *The house fly, disease carrier. An account of its dangerous activities and the means of destroying it*. New York: Stokes, 312 p.

Hoy, J. B., & Anderson, J. R. (1978). Behavior and reproductive physiology of the blood-sucking snipe flies (Diptera: Rhagionidae: *Symphoromyia*) attacking deer in Northern California. *Hilgardia, 46*, 113–168.

James, M. T. (1947). *The flies that cause myiasis in man*. U.S. Dept. Agric., Misc. Publ. No. 631, 175 p.

James, M. T. (1960). The soldier flies or Stratiomyidae of California. *Bulletin of the California Insect Survey, 6*, 79–122.

James, M. T. (1981). Stratiomyidae. In J. F. McAlpine, B. V. Peterson, G. E. Shewell, H. J. Teskey, J. R. Vockeroth, & D. M. Wood (Eds.), *Manual of Nearctic Diptera* (Vol. 1, pp. 497–511). Canada: Res. Branch, Agric. Monogr. No. 27.

James, M. T., & Turner, W. J. (1981). Rhagionidae. In J. F. McAlpine, B. V. Peterson, G. E. Shewell, H. J. Teskey, J. R. Vockeroth, & D. M. Wood (Eds.), *Manual of Nearctic Diptera* (Vol. 1, pp. 483–488). Canada: Res. Branch, Agric. Monogr. No. 27.

Johnson, R. N., Young, D. G., & Butler, J. F. (1993). Trypanosome transmission by *Corethrella wirthi* (Diptera: Chaoboridae) to the green treefrog, *Hyla cinerea* (Anura: Hylidae). *Journal of Medical Entomology, 30*, 918–921.

Katz, H. (1987). Managing mausoleum pests. *Pest Control Technology, 15*, 72–74.

Kerr, P. H. (2010). Phylogeny and classification of Rhagionidae, with implications for Tabanomorpha (Diptera: Brachycera). *Zootaxa, 25*, 1–133.

Knizesk, H. M., & Sullivan, D. J. (1984). Temporal distribution of crane flies (Diptera: Tipulidae) in a southern New York woodland. *The Canadian Entomologist, 116*, 1137–1144.

Kumm, H. W. (1935). The natural infection of *Hippelates pallipes* Loew with the spirochaetes of yaws. *Transactions of the Royal Society of Tropical Medicine and Hygiene, 29*, 265–272.

Lancaster, J. L., & Meisch, M. V. (1986). *Arthropods in livestock and poultry production*. New York: Halsted, 402 p.

Lindner, E. (1949). *Die Fliegen der Palaerktischen Region, Handbuch*. Stuttgart: E. Schweizerbartische Verlagsbuchhandlung, 422 p.

Lindquist, A. W., & Deonier, C. C. (1942). Flight and oviposition habits of the clear lake gnat. *Journal of Economic Entomology, 35*, 411–415.

Madwar, S. (1937). Biology and morphology of the immature stages of Mycetophilidae (Diptera, Nematocera). *Philosophical Transactions of the Royal Society of London, Series B, 227*, 1–110.

McAlpine, J. F. (1981a). Morphology and terminology–adults. In J. F. McAlpine, B. V. Peterson, G. E. Shewell, H. J. Teskey, J. R. Vockeroth, & D. M. Wood (Eds.), *Manual of Nearctic Diptera* (Vol. 1, pp. 9–63). Canada: Res. Branch, Agric. Monogr. No. 27.

McAlpine, J. F. (1981b). Key to families–adults. In J. F. McAlpine, B. V. Peterson, G. E. Shewell, H. J. Teskey, J. R. Vockeroth, & D. M. Wood (Eds.), *Manual of Nearctic Diptera* (Vol. 1, pp. 89–124). Canada: Res. Branch, Agric. Monogr. No. 27.

McAlpine, J. F. (1987). Piophilidae. In J. F. McAlpine, B. V. Peterson, G. E. Shewell, H. J. Teskey, J. R. Vockeroth, & D. M. Wood (Eds.), *Manual of Nearctic Diptera* (Vol. 2, pp. 845–852). Canada: Res. Branch, Agric. Monogr. No. 28.

McAlpine, J. F., Peterson, B. V., Shewell, G. E., Teskey, H. J., Vockeroth, J. R., & Wood, D. M. (Eds.). (1981). *Manual of Nearctic Diptera* (Vol. 1). Canada: Res. Branch, Agric, 674 p.

McAlpine, J. F., Peterson, B. V., Shewell, G. E., Teskey, H. J., Vockeroth, J. R., & Wood, D. M. (Eds.). (1987). *Manual of Nearctic Diptera* (Vol. 2). Canada: Res. Branch, Agric, 1332 p.

McCrae, A. W. R. (1967). Unique record of crane flies biting man. *Uganda Journal, 31*, 128.

McKeever, S. (1977). Observations of *Corethrella* feeding on tree frogs (*Hyla*). *Mosquito News, 37*, 522–523.

McKeever, S., & French, F. E. (1991). *Corethrella* (Diptera: Corethrellidae) of eastern North America: Laboratory life history and field responses to anuran calls. *Annals of the Entomological Society of America, 84*, 493–497.

Mohrig, W., Heller, K., Hippa, H., Vilkamaa, P., & Menzel, F. (2012). Revision of the black fungus gnats (Diptera: Sciaridae) of North America. *Studia Dipterologica, 19*, 141–286.

Nguyen, T. T. X., Tomberlin, J. K., & Vanlaerhoven, S. (2015). Ability of black soldier fly (Diptera: Stratiomyidae) larvae to recycle food waste. *Environmental Entomology, 44*, 406–410.

Oliver, D. R. (1981). Chironomidae. In J. F. McAlpine, B. V. Peterson, G. E. Shewell, H. J. Teskey, J. R. Vockeroth, & D. M. Wood (Eds.), *Manual of Nearctic Diptera* (Vol. 1, pp. 423–458). Canada: Res. Branch, Agric. Monogr. No. 27.

Oosterbroek, P., & Courtney, G. (1995). Phylogeny of the nematocerous families of Diptera (Insecta). *Zoological Journal of the Linnean Society, 115*, 267–311.

Otranto, D., Cantacessi, C., Testini, G., & Lia, R. P. (2006). *Phortica variegata* as an intermediate host of *Thelazia callipaeda* under natural conditions: Evidence for pathogen transmission by a male arthropod vector. *International Journal for Parasitology, 36*, 1167–1173.

Peterson, B. V. (1987). Phoridae. In J. F. McAlpine, B. V. Peterson, G. E. Shewell, H. J. Teskey, J. R. Vockeroth, & D. M. Wood (Eds.), *Manual of Nearctic Diptera* (Vol. 2, pp. 689–712). Canada: Res. Branch, Agric. Monogr. No. 28.

Sabrosky, C. W. (1980). New genera and new combinations in Nearctic Chloropidae (Diptera). *Proceedings of the Entomological Society of Washington, 82*, 412–429.

Sabrosky, C. W. (1987). Chloropidae. In J. F. McAlpine, B. V. Peterson, G. E. Shewell, H. J. Teskey, J. R. Vockeroth, & D. M. Wood (Eds.), *Manual of Nearctic Diptera* (Vol. 2, pp. 1049–1067). Canada: Res. Branch, Agric. Monogr. No. 28.

Sheppard, D. C. (1983). House fly and lesser house fly control utilizing the black soldier fly in manure management systems for caged laying hens. *Environmental Entomology, 12*, 1439–1442.

Sheppard, D. C., Newton, G. L., Thompson, S. A., & Savage, S. (1994). A value added manure management system using the black soldier fly. *Bioresource Technology, 50*, 275–279.

Smith, K. G. V. (Ed.). (1973). *Insects and other arthropods of medical significance*. Publication 720. London: British Museum (Natural History), 561 p.

Smith, K. G. V. (1986). *A manual of forensic entomology*. Ithaca, NY: Cornell Univ. Press, 205 p.

Steffan, W. A. (1981). Sciaridae. In J. F. McAlpine, B. V. Peterson, G. E. Shewell, H. J. Teskey, J. R. Vockeroth, & D. M. Wood (Eds.), *Manual of Nearctic Diptera* (Vol. 1, pp. 247−255). Canada: Res. Branch, Agric. Monogr. No. 27.

Stone, A. (1968). The genus *Corethrella* in the United States (Diptera: Chaoboridae). *Florida Entomologist, 51*, 183−186.

Stone, A., Sabrosky, C. W., Wirth, W. W., Foote, R. H., & Coulson, J. R. (1965). *A catalog of the Diptera of America north of Mexico* (Vol. 276). U.S. Dept. Agric. Handb, 1696 p.

Taplin, D., Zaias, N., & Rebell, G. (1967). Infection by *Hippelates* flies. *Lancet, 2*, 472.

Teskey, H. J. (1981a). Morphology and terminology − larvae. In J. F. McAlpine, B. V. Peterson, G. E. Shewell, H. J. Teskey, J. R. Vockeroth, & D. M. Wood (Eds.), *Manual of Nearctic Diptera* (Vol. 1, pp. 65−88). Canada: Res. Branch, Agric. Monogr. No. 27.

Teskey, H. J. (1981b). Key to families−larvae. In J. F. McAlpine, B. V. Peterson, G. E. Shewell, H. J. Teskey, J. R. Vockeroth, & D. M. Wood (Eds.), *Manual of Nearctic Diptera* (Vol. 1, pp. 125−147). Canada: Res. Branch, Agric. Monogr. No. 27.

The Brazilian Purpuric Fever Study Group. (1992). Brazilian purpuric fever identified in a new region of Brazil. *Journal of Infectious Diseases, 165*(Suppl. 1), S16−S19.

Tondella, M. L. C., Paganelli, C. H., Bortolotto, I. M., Takano, O. A., Trino, K., Brandileone, M. C. C., et al. (1994). Isolamento de *Haemophilus aegyptius* associado a febre purpurica Brasileira, de cloropideos (Diptera) dos generos *Hippelates* e *Liohippelates*. *Revista do Instituto de Medicina Tropical de São Paulo, 36*, 105−109.

Turner, W. J. (1974). A revision of the genus *Symphoromyia* Frauenfeld (Diptera: Rhagionidae). I. Introduction. Subgenera and species-groups. Review of biology. *The Canadian Entomologist, 106*, 851−868.

Turner, W. J. (1979). A case of severe human allergic reaction to bites of *Symphoromyia* (Diptera: Rhagionidae). *Journal of Medical Entomology, 15*, 138−139.

Vockeroth, J. R. (1981). Mycetophilidae. In J. F. McAlpine, B. V. Peterson, G. E. Shewell, H. J. Teskey, J. R. Vockeroth, & D. M. Wood (Eds.), *Manual of Nearctic Diptera* (Vol. 1, pp. 223−246). Canada: Res. Branch, Agric. Monogr. No. 27.

Webb, D. W. (1977). The nearctic Athericidae (Insecta: Diptera). *Journal of the Kansas Entomological Society, 50*, 473−495.

Webb, D. W. (1981). Athericidae. In J. F. McAlpine, B. V. Peterson, G. E. Shewell, H. J. Teskey, J. R. Vockeroth, & D. M. Wood (Eds.), *Manual of Nearctic Diptera* (Vol. 1, pp. 479−482). Canada: Res. Branch, Agric. Monogr. No. 27.

Wheeler, M. R. (1987). Drosophilidae. In J. F. McAlpine, B. V. Peterson, G. E. Shewell, H. J. Teskey, J. R. Vockeroth, & D. M. Wood (Eds.), *Manual of Nearctic Diptera* (Vol. 2, pp. 1011−1018). Canada: Res. Branch, Agric. Monogr. No. 28.

Wiegmann, B. M., Trautwein, M. D., Winkler, I. S., Barr, N. B., Kim, J.-W., Lambkin, C., et al. (2011). Episodic radiations in the fly tree of life. *Proceedings of the National Academy of Sciences, U.S.A, 108*, 5690−5695.

Williams, J. A., & Edman, J. D. (1968). Occurrence of blood meals in two species of *Corethrella* in Florida. *Annals of the Entomological Society of America, 61*, 1336.

Williams, R. E., Hall, R. D., Broce, A. B., & Scholl, P. J. (Eds.). (1985). *Livestock entomology*. New York: John Wiley & Sons, 335 p.

Yeates, D. K., Wiegmann, B. M., Courtney, G. W., Meier, R., Lambkin, C., & Pape, T. (2007). Phylogeny and systematics of Diptera: Two decades of progress and prospects. *Zootaxa, 1668*, 565−590.

Zumpt, F. (1965). *Myiasis in man and animals in the old world*. London: Butterworths, 267 p.

Chapter 12

Phlebotomine Sand Flies and Moth Flies (Psychodidae)

Leonard E. Munstermann
Yale University School of Public Health, New Haven, CT, United States

The Psychodidae are considered to be the most ancient of dipteran families, with fossils dating back to the late Jurassic or possibly early Triassic period, approximately 200 million years ago. Several genera with modern psychodid morphology have been recovered from Burmese amber of the late Cretaceous. These flies are among the most widely distributed Diptera. They are found in the desert and rain forest and inhabit all zoogeographic regions. Of the six subfamilies, the Psychodinae and Phlebotominae are by far the most common; they are of serious economic and medical importance, especially the latter subfamily.

Members of the large subfamily Psychodinae are commonly known as **moth flies** or **owl flies** and have no role in disease transmission; these flies do not have bloodsucking mouthparts and often take little nutrition as adults. They are small, short-legged flies, with abundant scale-like setae on the body and wings giving them a moth-like appearance (Fig. 12.1); hence the common name for this subfamily is "moth flies." Moth flies have little economic importance, although a few species can be pests in sewage treatment facilities.

The Phlebotominae are biting flies (Fig. 12.2) and are known throughout the world for their role in transmitting two protozoan diseases, visceral and cutaneous leishmaniasis**.** They also are known vectors of several viral diseases such as sand fly fever, vesicular stomatitis, Changuinola and Chandipura viruses. One bacterial disease of humans, bartonellosis, is transmitted by several phlebotomines of the high Andes. Two genera encompass almost all known disease vectors: *Phlebotomus* in the Old World and

FIGURE 12.1 Moth fly, *Clogmia albipunctata* (Psychodinae), Spain; note the "hairy" wings and moth-like appearance. *Photograph by Katja Schulz.*

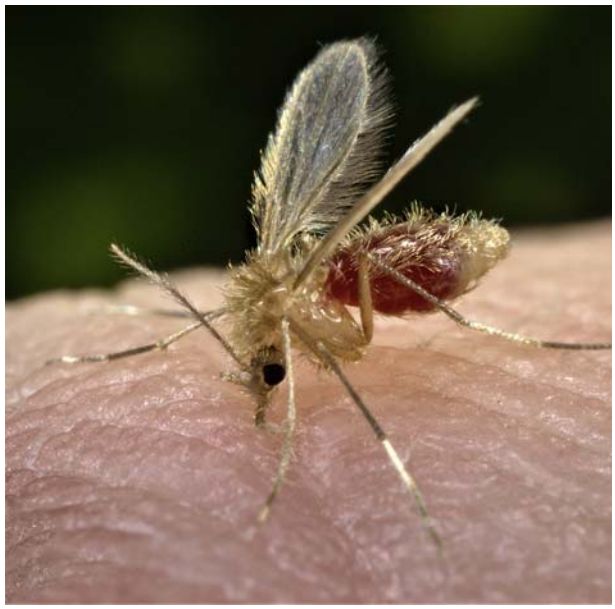


FIGURE 12.2 Sand fly, *Phlebotomus papatasi*, female feeding on a human. *Photograph by James Gathany. Public Health Image Library, Centers for Disease Control and Prevention, USA; PHIL #10275.*

Medical and Veterinary Entomology. https://doi.org/10.1016/B978-0-12-814043-7.00012-1

Lutzomyia in the Americas. Although the common name **sand fly** derives from studies of phlebotomines in the more arid regions of the Old World, equally diverse species numbers occur in the moist tropical regions of the Americas. The term "sand fly" has been a source of misunderstanding because the term is also applied commonly to biting midges (Ceratopogonidae) and occasionally to blackflies (Simuliidae).

TAXONOMY

A current listing of valid names for Psychodidae is composed of 3,190 described species (Global Biodiversity Information Facility). Of the six, only two feed on blood, the Sycoracinae and Phlebotominae. The other four, Bruchomyiinae, Trichomyiinae, Horaellinae, and Psychodinae, do not (Table 12.1). The first three are relatively small but widely distributed groups. The Bruchomyiinae consist of 60 species in seven genera and are considered the oldest lineage within Psychodidae, sharing several features with the primitive fly family, Tanyderidae. Although Bruchomyiinae are found on all continents, they are restricted to moist tropical or subtropical environments. In appearance, the group is characterized by long and dense scales, long legs, and long antennae. The only US representative, *Nemapalpus nearctica*, is confined to Florida and has endangered species status.

Trichomyiinae includes 85 species, all in the genus *Trichomyia*. Several additional fossil genera are described from Cretaceous amber. The flies are characterized by compact bodies with densely packed erect scales and short legs, with a typical moth fly appearance. They are cosmopolitan but are found only in moist woodlands or tree-hole environments (Azar et al., 2014). The half-dozen species of the subgenus Horaellinae are in the genus *Horaiella*, described from Thailand and India. They are closely related to and are formerly treated as Trichomyiinae.

Psychodinae

The subfamily Psychodinae forms the largest component of the Psychodidae and consists of about 2,000 species and 160 genera. In the United States and Canada, approximately 100 species in 18 genera have been reported (Quate and Vickeroth, 1981). The greatest species diversity of Psychodinae occurs in the tropics. Most adults are not commonly seen because of their cryptic resting behavior and nocturnal flight activity. The following physical features readily distinguish members of this subfamily from the Phlebotominae: short legs, short antennal segments, and compact bodies, with a dense layer of scales on all body parts. The wings are held as a V-shaped tent over the abdomen. For identification keys to the North American species, see Quate (1955); for species descriptions and revisions of New World taxa, see additional works by Quate and Ibáñez-Bernal.

Species of the cosmopolitan genera *Clogmia* and *Psychoda* can occur in pestiferous numbers near waste-processing plants or sewage lagoons in which the larvae thrive. In human dwellings, they easily survive in drain pipes or poorly maintained latrines. Although relatively rare, myiasis by moth fly larvae has been reported in human urogenital, intestinal, and nasopharyngeal tracts.

Relatively few genetic studies have been done with the Psychodinae. Chromosome numbers have been obtained in the genera *Clogmia* ($2N = 6$) and *Pericoma* ($2N = 14$). *Clogmia albipunctata* is an excellent laboratory species for study, is easily reared, and has large larval salivary chromosomes. The gap-gene system in *C. albipunctata* has been a model for comparing segmentation embryogenesis of these nematocerous flies with the more highly evolved Diptera.

Sycoracinae

The subfamily Sycoracinae consists of 45 species in three genera. All have bloodsucking mouthparts, have characteristic long legs, and relatively few, short scales. Field observations and identification of blood meals indicate that at least three species feed on amphibians. One species, *Sycorax silacea*, is a vector for the filarial nematode *Icosiella neglecta*, which parasitizes the French edible frog *Rana esculenta*. *Sycorax wampukrum* feeds on the stubfoot

TABLE 12.1 Subfamilies of the Psychodidae: Taxonomic, Geographic, and Behavioral Aspects

Subfamily	No. of Genera	No. of Species	Geographic Distribution	Blood-Feeding Behavior
Bruchomyiinae	7	60	Pantropical, subtropics	None
Trichomyiinae	1	85	Moist woodlands, tree holes	None
Horellinae	1	6	Tropical Southeast Asia	None
Psychodinae	160	c. 2,000	Cosmopolitan	None
Sycoracinae	3	45	New World tropics	Amphibians
Phlebotominae	23	960	Cosmopolitan	Vertebrates

toad (*Atelopus* sp.) in Ecuador; and *Aposycorax chilensis* feeds on a wood frog (*Batrachyla* sp.) in Patagonia, Argentina.

Phlebotominae

Until the 1990s, nearly all the approximately 960 species of Phlebotominae were represented by three genera: *Phlebotomus* (Afrotropical, Palearctic, Indo-Malay), *Lutzomyia* (Neotropical), and *Sergentomyia* (Afrotropical, Asia). More recently, many of the subgenera have been elevated to generic status along with descriptions of new genera. Based on traditional morphological criteria, Artemiev (1991) proposed for Psychodinae a system of seven subtribes and 24 genera. Later, in a cladistic analysis of Artemiev's subtribe Brumptomyiina (phlebotomines of the Americas), Galati proposed new genera and raised many of the *Lutzomyia* subgenera to generic status, totaling 23 genera (Rangel and Lainson, 2003). The designations are based on cladistical analyses of morphological characters as well as more current molecular data and are gradually coming into broader use. Akhoundi et al. (2016) provide a detailed compendium of sand fly taxonomic history, current status, and coevolution with their leishmanial parasites.

The classic phlebotomine identification handbook for Central and South America is that of Young and Duncan (1994). For the American fauna north of Mexico, see the review by Young and Perkins (1984). For detailed biological information, species descriptions, and keys to the Palearctic sand flies, see Perfil'ev (1966); and for the Mediterranean, West African, and Indo-Malayan areas, see Lewis (1982). Although sand flies of sub-Saharan Africa are the least known, Davidson (1990) summarizes the current knowledge of the African *Sergentomyia*. In general, many of the phlebotomine species descriptions are based on a few specimens collected at a single location, whereas most of the biology and epidemiological relationships are based on a few very widespread species. For this reason, generalizations about the life history, behavior, and ecology are based on a relatively few easily collected or colonized species and may not be representative of other species.

Genetic analyses have an important role in identifying cryptic species, providing a perspective on the phylogenetic relationships among genera and species (see Beati et al., 2004). Cryptic species have been identified in *Lutzomyia longipalpis* and members of the *Lutzomyia verrucarum* group based on karyotypes, isoenzymes, and mitochondrial and nuclear DNA gene sequences. The systematic rearrangements in *Lutzomyia* proposed by Galati have been supported by the sequencing of ribosomal genes of several genera and subgenera in *Phlebotomus* and *Lutzomyia*. As cytochrome c oxidase subunit I (COI) bar-coding sequences are accumulated in the Phlebotominae, they are likely to become a standard screening method (see Gutiérez et al., 2015, for *Lutzomyia* of Colombia).

Two medically important species, *Lutzomyia longipalpis* and *Phlebotomus papatasi*, have undergone whole-genome sequencing. The *Lu. longipalpis* genome consists of 150 megabases (Mb), 40% of which is currently sequenced. That of *P. papatasi* is estimated at 350 Mb with about 23% coverage. Details of gene annotation and functional classes can be found at the Vectorbase website (http//www.vectorbase.org). The two genomes are derived from laboratory strains with lowered genetic variability and serve as a base of comparison with field populations of these species, as well as related outgroups such as *Phlebotomus dubosqi*, *P. bergeroti*, *Lutzomyia intermedia*, and *Lu. migonei*.

MORPHOLOGY

Psychodinae

The eggs are less than 0.5 mm long and are deposited individually or in clusters of 10–100 or more. Newly deposited eggs are nearly white and gradually melanize to a tan or dark brown color.

The mature larvae are elongate, legless, and up to 6 mm long. The larvae of several genera have long spines or feathery processes along the body (e.g., *Pericoma*) (Fig. 12.3A), which are less pronounced in *Psychoda* and *Clogmia.* In the latter genera, the body is fusiform or subcylindrical, whereas in other species it may be dorsoventrally flattened. The larva has three thoracic and nine abdominal segments. The segments are secondarily divided into annuli, with two annuli comprising each of the thoracic

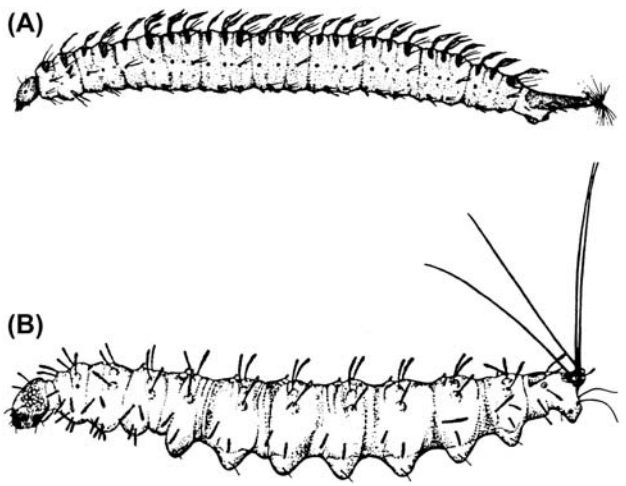


FIGURE 12.3 Psychodid larvae: (A) *Pericoma* (Psychodinae); (B) *Phlebotomus* (Phlebotominae). *(A) From Johansen, 1934; (B) Modified from Patton and Evans, 1929.*

and first abdominal segments and three comprising abdominal segments 2–7. The dorsal cuticle has minute spines and a narrow, transverse, sclerotized plate on each segmental annulus. The head is well-developed with short antennae, lateral eyespots, and strong mandibles. An anterior pair of spiracles is located on the prothorax and a posterior pair at the tip of a rigid siphon is at the tip of the abdomen. The posterior spiracles are surrounded by four lobes with water-repellent hairs. The larval cuticle is flexible and grayish, with a darker heavily sclerotized head and segmental plates, terminating with a dark brown or black syphon.

The pupae of moth flies may be free-floating or attached to the substrate. Attached forms are erect, with exuviae of the last larval instar adhering to the caudal end. The pupa is obtect, i.e., with visible appendages of the head and thorax held closely to the body. The thorax has a dorsal pair of tubelike respiratory organs, and the abdomen often bears numerous setae and spines.

Adult moth flies typically vary in length from 2 to 4 mm, but the extremes range from the European *Psychoda phalaenoides* with a wingspan of less than 2 mm to the Australian *Pericoma funebris* with a wingspan of 10 mm. The head and body are densely covered with hairlike scales of gray, brown, black, or yellowish color (Fig. 12.1). The elongate, 12- to 16-segmented antennae are similar in males and females. The antennal segments are beaded and covered with short setae; each segment also bears a characteristic whorl of longer setae. The palpi are long, recurved, and four-segmented, with scattered setae. Mandibles are rudimentary or absent. Ocelli are absent. The distinctive wings are large, broadly ovate to elliptic or pointed, and densely scaled; no cross-veins appear beyond the basal area. All longitudinal veins separate near the base, except for the branching of R_2 and R_3 and that of M_1 and M_2 (Fig. 12.4). The abdomen has six to eight apparent segments. Males are distinguishable by the slight abdominal extensions provided by the coxites of the genitalia.

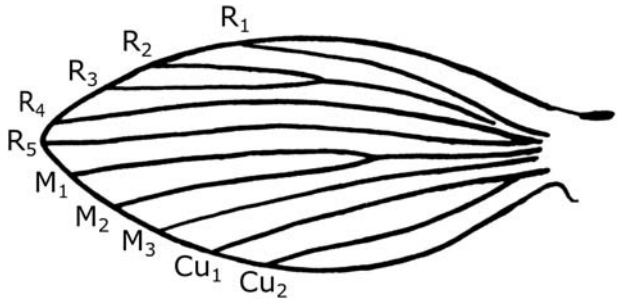


FIGURE 12.4 Typical wing venation of psychodid adults, with names of the veins abbreviated: *C*, cubital; *M*, medial; *R*, radial.

Phlebotominae

Eggs of the Phlebotominae are similar in structure to the Psychodinae; they are elongate with rounded ends, 150 × 400 μm. Within 12 h after oviposition, the egg chorion melanizes to brown or dark brown, with a somewhat glossy surface and a variety of fine surface markings. Flies may be taxonomically grouped based on the similarity of egg markings that include parallel ridges, irregular patterns, polygons, pits, and mountain- or volcano-like structures (De Almeidae, 2004). After oviposition, the larva develops inside the egg and emerges as a first-stage larva in 4–10 days.

The larvae grow through four instars to a length of up to 10 mm, with a light grayish body, a dark head, and long caudal setae (e.g., *Phlebotomus*) (Fig. 12.3B). The head, thorax, and abdomen bear numerous, prominent, clavate setae that can be useful for identification. The head is complete and prognathous, with lateral eyespots, short antennae, and heavy, toothed mandibles that oppose a heavy platelike, serrate mentum. As in the Psychodinae, the body consists of three thoracic and seven abdominal segments, but unlike them, they are not clearly differentiated into separate tagmata, nor do they show segmental annuli or dorsal sclerites. Abdominal segments 1–8 each bear a medioventral proleg, or pseudopodium. Two pairs of spiracles are present: an anterior pair on the prothorax and a posterior pair on the greatly reduced abdominal segment 9. This segment also carries two (first-instar larva) or four (instars 2–4) long, conspicuous caudal setae adjacent to the spiracles. Larvae of the New World genus *Brumptomyia* and the Old World species *Phlebotomus tobbi* have two caudal setae in all instars. Time of larval development can vary greatly from 4 to 8 weeks.

Pupae of sand flies attach in an erect position to substrates of the larval medium, with the exuviae of the last larval instar attached at the caudal end. The pupae can be distinguished from those of moth flies by the clubbed setae on the body and the retained long caudal setae of the larval exuviae. The pupa is obtect, with antennae, legs, and wings closely appressed and visible through the pupal casing. Initially, the pupa is a pale yellowish color, but it darkens as the cuticle tans and hardens. The prothorax bears a pair of short tubelike respiratory organs and the abdomen has numerous setae and spines. The sex can be determined by removing the attached larval exuviae to reveal the enclosed terminalia.

Sand fly adults (Fig. 12.2) are usually less than 5 mm long, with a more slender and elongate thorax and abdomen, in contrast to the moth flies (Fig. 12.1). The scales are erect but sparser and can be grayish, brownish, or yellowish. The head is small and hypognathous, with dark,

conspicuous eyes and no ocelli. The long, slender, 12- to 16-segmented antennae are similar in males and females. The segments are closely covered with short setae and each segment has a whorl of long setae**.** Closely associated with the mouthparts are the five-segmented palps bearing an array of sensory setae. The thorax is strongly humped. The wings are large, broadly ovate to elliptical or pointed, with long narrow scales lining the veins (Fig. 12.2). The abdomen is six- to eight-segmented. The male genitalia are conspicuous and are essential for identifying most species.

The female mouthparts include six broad, knifelike stylets (labrum, paired mandibles and maxillae, and hypopharynx) that are held within the fleshy labium when not in use. The mandibles and maxillae are toothed distally. When blood feeding, the mandibles cut the skin with scissors-like and sawing movements while the maxillary teeth engage the sides of the wound and hold the mouthparts in place. Blood is taken from a subcutaneous pool produced by injury to the blood vessels. The food canal is formed by apposition of the labrum above and the hypopharynx below, which contains the salivary duct. Components of the saliva that facilitate the blood-feeding process include anticlotting and vasodilatory factors. Compared with female mouthparts, those of males are generally much reduced, with few or no teeth. Although males have been observed to ingest blood from wounds made by the females, they normally feed on plant sugars as energy sources, as do females.

LIFE HISTORY

The life history, behaviors, and genetics of the Psychodidae are inferred largely from a relatively few common species and model systems based on laboratory colonies. The larval forms and habitats of most species remain largely unknown.

Psychodinae

The larvae of common species of *Psychoda* and *Clogmia* occur in aquatic and semiaquatic habitats that include rock pools, tree holes, and margins of ponds, streams, and ditches. They also are associated with highly organic, aquatic environments produced by disposal of human sewage, such as water-treatment plants, septic tanks, and indoor pipes and drains.

The eggs are deposited individually or in gelatinous masses of 20−100, which hatch in about 2 days. Parthenogenesis has been observed in the Old World species *Psychoda severini*. Larvae develop in floating vegetation, mud, manure, and similar wet or moist organic media, where they feed on decaying organic matter, bacteria, fungi, algae, and other microorganisms. The larval period is 9−15 days, during which they undergo four molts before pupation. The pupal period is 1−2 days. Larvae of *Psychoda alternata* are highly tolerant of pollution, low dissolved oxygen, low pH, and high temperatures. The adults normally remain in proximity to the larval habitat, where they mate and oviposit shortly thereafter. Nutrients necessary for egg production are ingested during the larval stages.

Phlebotominae

In contrast to the semiaquatic moth fly larvae, phlebotomine sand fly larval habitats are terrestrial, occurring chiefly in highly organic soils or accumulations of decomposed manure. Although larvae are difficult to find, adults are usually collected near sites where larvae develop. Natural adult environments include tree hollows, buttress roots, rock piles, animal burrows, and even termite mounds. Species of medical importance are often associated with domestic animal enclosures such as poultry houses or pigsties (Figs. 12.5 and 12.6) and can be found resting in dark, damp enclosures such as basements, wells, and privies. Emergence traps set over presumed larval sites can be used to capture adults and identify larval development sites (Fig. 12.5). Larvae of several important neotropical species have been found in the litter of the forest floor; these include *Lutzomyia gomezi*, *Lu. panamensis*, *Lu. pessoana*, and *Lu. trapidoi*. *Lutzomyia longipalpus* has been recovered from soil−manure mixtures at barnyard margins. Two common Eurasian species, *Phlebotomus papatasi* and *P. argentipes*, occur in organic soil near stables, barns, and houses. In Italy, *P. perfiliewi* larvae develop in farmyard manure, whereas the central Asian *P. caucasicus* is common in rodent burrows.

FIGURE 12.5 Breeding site of the sand fly *Lutzomyia longipalpis*, along the margin of a pigsty (pigpen) where larvae were collected. *Arrows* denote netted-tube emergence traps for collecting adults. *Photograph by Leonard E. Munstermann.*

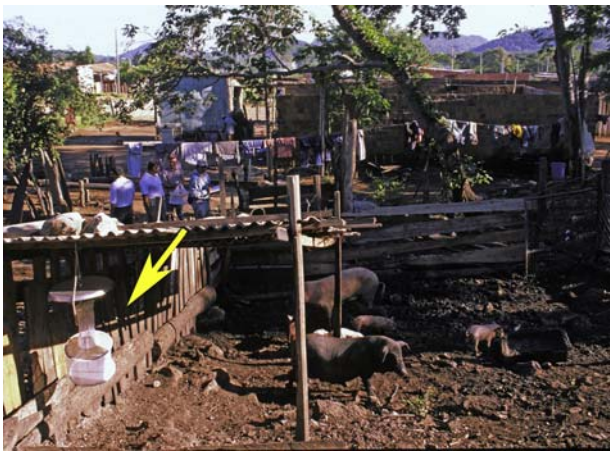


FIGURE 12.6 Pigsty near Corumbá, Brazil, where *Lutzomyia cruzi* was being collected in a Centers for Disease Control light trap (*arrow*). *Photograph by Leonard E. Munstermann.*

Sand flies may be autogenous or anautogenous. Females of autogenous species complete the first cycle of egg development without taking blood but require one or more blood meals to complete each subsequent cycle. Females of anautogenous species require one or more blood meals to complete each cycle, including the first. Autogeny has been observed under laboratory conditions in only a few species (e.g., *Lutzomyia lichyi*, *Phlebotomus bergeroti*, and *P. kazeruni*). In one study, 8% of female *P. papatasi* produced eggs autogenously with a mean clutch size of 12, compared with a clutch size of 60 to 70 from anautogenous females. Multiple blood meals in a single egg cycle have been demonstrated in several species. Females of most species complete multiple cycles; *P. argentipes* may complete as many as four cycles under laboratory conditions.

Phlebotomus papatasi illustrates a typical pattern of egg maturation after a blood meal. Ingested blood cells begin to break down 6–18 h after feeding. The peritrophic envelope surrounding the blood meal matures at 24 h. The processes of digestion, absorption, and assimilation of the blood and maturation of the eggs within the ovarian follicles are usually completed in 5–8 days. Approximately 30–60 eggs are produced in each gonotrophic cycle.

The eggs hatch in 4–20 days. Larvae feed on decaying organic matter, fungi, and associated microorganisms. Four larval instars develop over a period varying from 30 to 60 days, depending on temperature and food quality. In climates with a cold winter or a long hot or dry season, diapause or estivation may occur in the egg stage or the fourth larval instar and can survive as long as a year. In *P. papatasi*, the proportion of larvae in diapause increases from summer to fall and is independent of temperature.

The pupal period is approximately 1 week. The adult life span can vary from 2 to 6 weeks. Kasap and Alten (2005) found the thermal requirement for development of *P. papatasi* to be 440 days above 20°C. Detailed life-table studies have been produced for *P. papatasi* (Kasap and Alten, 2005), *Lutzomyia shannoni* (Cardenas et al., 1999), and *Lu. serrana* (Santamaria et al., 2002).

BEHAVIOR AND ECOLOGY

Psychodinae

Adult moth flies are common at lights and near larval habitats where suitable resting sites are available. Resting sites are commonly in dark, humid, protected areas. For human-associated species, resting areas include buildings, drains, and sewage treatment containments (e.g., cesspools and treatment plants). Adults feed on nectar and septic fluids. They walk with a characteristic hesitating motion and fly noiselessly in short, discrete hops of a few centimeters. They have been known to invade buildings more than 90 m from the nearest larval site and may be carried as far as 1.5 km by prevailing winds. Many of the feral species of *Psychoda* and *Brunnetia* are collected by light traps set in scrub brush or forests; however, most other genera are found amid vegetation, on tree trunks, or in rock crevices where sweep nets or aspirators are necessary.

Phlebotominae

Phlebotomine adults are found resting near larval sites and commonly near hosts that provide a blood source. In natural settings, they rest in forest litter, on tree trunks or tree hollows, in caves and rock crevices, and in animal burrows or nests. Domestic sites include livestock pens, buildings, masonry crevices, and other dark, humid locations. Collecting phlebotomine adults is typically by light traps (Fig. 12.6), whereas many species that are not attracted to light must be collected by handheld aspirators (Fig. 12.7) or other mechanical means. Neotropical forest species may be found in forest litter (*Lutzomyia trapidoi*), on understory plants (*Lu. pessoana*), on the trunks of trees (*Lu. trinidadensis*), or in the forest canopy (*Lu. rorotaensis*). Certain species are more peridomestic, including *Lu. longipalpis*, *Lu. verrucarum*, *Phlebotomus argentipes*, and *P. papatasi*. In the Americas, a common species, *Lu. longipalpis*, can be readily collected near pigsties, from the undersides of chicken coops, or feeding on penned ungulates. In southeastern Asia, *P. argentipes* is more domestic and is collected in the dark corners of houses, such as closets or bathrooms.

Mark-recapture studies with *Lu. longipalpus* have demonstrated that a flight range of 1.5 km is possible. Because they are not strong fliers, they are inhibited by wind and rain, although broader dispersal is probably facilitated by strong winds; e.g., *Phlebotomus orientalis*

FIGURE 12.7 The author aspirating *Lutzomyia shannoni* adults from tree buttresses in a tropical forest; Canal Zone, Panama.

does not fly at wind speeds above 15 kph. The flight ranges of neotropical forest species are usually less than 200 m, but some species move daily between the forest floor and canopy. Flight ranges of *P. argentipes* and *P. orientalis* may be 500 m or more, and those of *Lu. longipalpis* and *P. caucasicus* can reach 1,000 m. However, longer distance records include a female *Phlebotomus ariasi* recaptured 2.2 km from a release point in France, and a male *Phlebotomus perniciosus* captured on the island of Jersey (British Isles), 25 km from the nearest source on the mainland in France.

Adult sand flies feed on plant sap, nectar, and honeydew, storing sugar and protein-rich liquids in their crop. Females of *P. papatasi* can pierce the stems and leaves of plants to obtain sap. In addition, females require vertebrate blood for development of their eggs. Blood feeding is directed to more tender and exposed skin areas such as the ears, nose, neck, undersides, feet, and tail. Males and females of *Lu. longipalpis* aggregate on vertebrate hosts, where the males perform lekking behaviors to protect a mating site while the female is blood feeding. Males emit a terpenoid pheromone from glands on the abdomen to attract the female, which then feeds and mates within the male territory. Males also perform elaborate acoustic and visual displays during courtship and mating.

Most sand flies blood feed at dusk and throughout the night, although a few species, including several important vectors, will bite during the daylight hours (habitually or when disturbed). *Lutzomyia panamensis*, *Lu. pessoana*, *Lu. sanguinaria*, and *Lu. trapidoi* of the tropical lowlands of Panama blood feed at temperatures above 20°C, whereas *Lu. verrucarum* in the higher elevations of Peru feeds at temperatures as low as 10°C. Some species are endophilic (*Lu. verrucarum* and *P. papatasi*) and others are exophilic (*Lu. trapidoi* and *P. perniciosus*).

Although most sand flies have broad host preferences and are opportunistic feeders, some are narrowly restrictive. For example, *Lutzomyia gomezi* is known to feed on birds and on members of five different orders of mammals, but *Lu. vespertilionis* feeds exclusively on bats. *Phlebotomus papatasi* is anthropophagous, feeding preferentially on humans and domestic dogs throughout its range, whereas *P. argentipes* is anthropophagous in some areas and zoophilic in others, feeding preferentially on cattle, Similarly, *Lu. longipalpis* seems to prefer domestic animals, but in the absence of other hosts will feed avidly on humans.

Sand flies exhibit characteristic seasonal and biotopic patterns. In tropical areas, populations of most species increase during or shortly after the rainy season. For example, populations of *P. argentipes* and *P. papatasi* in India, as well as *Lu. longipalpis* in the Americas, typically increase during the rainy season and decrease or disappear during the dry season. In Africa, population densities of *Phlebotomus duboscqi* and *P. martini* vary seasonally with the activity cycles of their rodent hosts.

Clearly defined host, seasonal, and geographic associations are found throughout phlebotomine distribution ranges. Light-trap collections in Panama have revealed a primarily anthropophilic association represented by *Lu. gomezi*, *Lu. panamensis*, and *Lu. dysponeta*, and a primarily zoophilic association represented by *Lu. carpenteri*, *Lu. triramula*, and *Lu. camposi*. In some locations, the anthropophilic and zoophilic associations alternate by season. Similarly, collections in southern Turkey have revealed two overlapping altitudinal associations. *Sergentomyia theodori* and *P. tobbi* are the most numerous species at lowland altitudes around 350 m, whereas *Phlebotomus transcaucasicus* is the most numerous species in the 1,300-m, highland zone.

The distribution patterns of the approximately 400 species in the Americas are strongly correlated with the amount of forest cover, with species diversity increasing from grassy to secondary forest biotopes and from secondary to mature forest biotopes. Similarly, in East Africa, populations of *Sergentomyia bedfordi* and *Sergentomyia antennata* increase as one moves from thickets to open-canopy forests to closed-canopy forest; in West Africa, these species increase from the open plains to more heavily vegetated habitats. In Colombia, a zone dominated by *Lutzomyia evansi* exhibits greater species richness, diversity, and abundance in natural forest areas than in nearby agricultural areas. These examples illustrate how spatial and temporal differences in local geography can affect the distribution, abundance, diversity, and species composition of sand fly communities. This is particularly evident in tropical forests where the species diversity and abundance

are high, yet the identified species composition can vary considerably from locality to locality.

PUBLIC HEALTH IMPORTANCE

Psychodinae

The role of the Psychodinae in public health is minimal. Moth flies have been associated with cases of allergic rhinitis and asthma in western and southern Africa. Accidental myiasis caused by moth flies has been reported as well, including enteric, urogenital, bronchial, and even ocular infestations. *Psychoda alternata* is the species cited most frequently, but *Ps. cinerea* and *Ps. albipennis* also have been involved.

As pests of economic impact, larvae of *Psychoda* and *Clogmia* are the most commonly identified because of their wide distribution and affinity for decaying organic matter. They often occur in filter beds and settling tanks of sewage-treatment and water-treatment plants, where they clog filtering mechanisms and where the adults emerge in enormous numbers. Adult moth flies can be annoying pests in the neighborhood of these facilities. They also can appear in and around homes and buildings, where they emerge from sumps, sink and floor drains, cesspools, aquariums, and other larval feeding sites. As a result, moth flies are commonly referred to as **filter flies**, **drain flies**, and **sewer flies**. Nuisance problems also have been reported in connection with turf production in Florida, and greenhouse operations in California (USA), and malt production in England.

The moth fly *Ps. alternata* is the most common species associated with sewage treatment plants worldwide, occurring in North and South America, Europe, Asia, Africa, and Australia. *Psychoda albipennis* and *Ps. severini* also are common in sewage treatment plants in Europe. *Psychoda cinerea* and *Ps. pacifica* occur in the eastern and western United States, respectively. *Clogmia albipunctata* is widely distributed in North America and is common in Europe and Africa. Because of its morphological similarity to *Ps. alternata*, the two species may be commonly confounded with one another.

Phlebotominae

Phlebotomine sand flies are annoying biting pests in places where they are abundant, often probing and biting repeatedly before feeding to repletion, causing a sharp, pricking sensation each time. Biting by *Lu. verrucarum* reportedly makes sleep difficult in highly infested districts of Peru. In one study, the mean biting rate was estimated to be 20–50 bites per person per night, and one individual received an estimated 300 bites in a single night. Other highly anthropophilic species are *Lutzomyia diabolica* in the United States; *Lutzomyia gomezi*, *Lu. olmeca*, *Lu. panamensis*, *Lu. pessoana*, *Lu. sanguinaria*, *Lu. trapidoi*, and *Lu. ylephiletor* in Central America; *Lu. wellcomei* in Brazil; and *Phlebotomus sergenti*, *P. argentipes*, and *P. papatasi* in the Old World.

Initial bites received by an individual produce redness and swelling, but additional exposures can induce sensitization, resulting in immediate or delayed skin reactions. The typical reaction to the bite is a pink or red papule about 2–3 mm in diameter and 0.5 mm high, which remains prominent for 4–5 days before gradually disappearing. Moderate to severe itching usually occurs. Individuals who become hypersensitive may develop hives, with pronounced swelling of the eyelids and lips if those sites are bitten. Prolonged exposure to sand fly bites results in eventual desensitization. Chronically exposed individuals living in areas with high sand fly populations may exhibit little or no reaction to the bites.

The most important role of phlebotomines is in the transmission of cutaneous and visceral leishmaniasis around the globe. Many species of *Lutzomyia* and *Phlebotomus* are involved in this cycle. Several additional viral, bacterial, and parasitic pathogens are transmitted to humans by members of these genera (Table 12.2). The zoophilic genera *Brumptomyia*, *Warileya*, and *Sergentomyia* as well as species of *Lutzomyia* and *Phlebotomus* are probably involved in the maintenance of zoonotic diseases.

Vesicular Stomatitis Virus Disease

Vesicular stomatitis virus is a member of the family Rhabdoviridae, genus *Vesiculovirus*. Vesiculoviruses have a distinctive bullet shape like that of most rhabdoviruses of animals. Vesicular stomatitis virus is an important pathogen of livestock and an occasional human pathogen. In humans, it produces an acute, self-limiting illness with fever, chills, and myalgia. Pharyngitis, oral mucosal vesicular lesions, and cervical adenopathy are characteristic. The vesicular stomatitis serotypes Alagoas, Indiana, and New Jersey are widely distributed throughout North and South America. Information derived from studies on the structure and replication of vesicular stomatitis virus has served as a model for the related rabies virus (genus *Lyssavirus*).

Two species of sand flies are proven vectors of vesicular stomatitis virus in nature (Comer and Tesh, 1991). *Lutzomyia shannoni* transmits the New Jersey serotype among feral swine on Ossabaw Island, Georgia (USA) and *Lu. trapidoi* transmits the Indiana serotype in Latin America. The virus has been isolated repeatedly from both sand fly species; furthermore, the sand flies can be infected by normal feeding with infected blood and then successfully transmit the virus to experimental animals. Transovarial transmission of *Vesiculovirus* has also been demonstrated

TABLE 12.2 Sand Fly-Borne Diseases of Humans

Disease	Causative Agent	Geographic Distribution	Reservoirs	Sand Fly Vectors
Sand fly fever (New World)	Sand fly fever virus (Candiru, Chagres, Punta Toro serotypes)	Panama, Colombia	Rodents, Primates	*Lutzomyia trapidoi, Lu. ylephiletor*
Sand fly fever (Old World)	Sand fly fever virus (Naples, Sicilian serotypes)	Tropical and subtropical Europe, Asia, northern Africa	Rodents (Muridae)	*Phlebotomus papatasi, P. perfiliewi, P. perniciosus*
Changuinola virus disease	Changuinola fever virus	Central and South America	Sloths	*Lutzomyia umbratilis*
Vesicular stomatitis virus disease	Vesicular stomatitis virus (Alagoas, Indiana, New Jersey serotypes)	Tropical, subtropical and temperate North and South America	Opossums, monkeys, porcupines, raccoons, bobcats, horses, swine, pronghorns, cattle, sheep	*Lutzomyia shannoni, Lu. trapidoi, Lu. ylephiletor*
Chandipura virus disease	Chandipura virus	India, West Africa	Hedgehogs	*Phlebotomus papatasi*
Bartonellosis	*Bartonella bacilliformis* (bacterium)	Colombia, Ecuador, Peru	Rodents (*Rattus*)	*Lutzomyia verrucarum, Lu. peruensis, Lu. columbiana*
Cutaneous leishmaniasis (New World)	*Leishmania amazonensis,*[a,b] *Le. braziliensis,*[b] *Le. colombiensis, Le. garnhami, Le. guyanensis,*[a,b] *Le. lainsoni, Le. mexicana, Le. naiffi, Le. panamensis,*[b] *Le. peruviana, Le. pifanoi, Le. shawi, Le. venezuelensis*	Tropical and subtropical Central and South America, Mexico, United States (Texas)	Opossums, monkeys, sloths, armadillos, anteaters; various rodents (Sciuridae, Heteromyidae, Muridae, Dasyproctidae, Capromyidae, Echimyidae); mongooses, canines, cats, raccoons, horses	*Lutzomyia anduzei, Lu. anthophora, Lu. ayacuchensis, Lu. ayrozai, Lu. carrerai, Lu. christophei, Lu. diabolica, Lu. flaviscutellata, Lu. gomezi, Lu. hartmanni, Lu. intermedia, Lu. lichyi, Lu. llanosmartinsi, Lu. migonei, Lu. nuneztovari, Lu. olmeca, Lu. ovallesi, Lu. panamensis, Lu. paraensis, Lu. peruensis, Lu. pessoai, Lu. reducta, Lu. spinicrassa, Lu. squamiventris, Lu. townsendi, Lu. trapidoi, Lu. trinidadensis, Lu. ubiquitalis, Lu. umbratilis, Lu. verrucarum, Lu. wellcomei, Lu. whitmani, Lu. ylephiletor, Lu. youngi, Lu. yucumensis*
Cutaneous leishmaniasis (Old World)	*Leishmania aethiopica, Le. killicki, Le. major,*[b] *Le. tropica*[a]	Tropical and subtropical Europe, Asia and Africa	Monkeys, rodents (Sciuridae, Muridae), dogs, hyraxes	*Phlebotomus aculeatus, P. alexandri, P. ansarii, P. duboscqi, P. guggisbergi, P. longipes, P. papatasi, P. pedifer, P. rossi, P. salehi, P. sergenti*
Visceral leishmaniasis (New World)	*Le. infantum* (=*Le. chagasi*)	Tropical and subtropical Central and South America	Opossums, canines	*Lutzomyia antunesi, Lu. cruzi, Lu. evansi, Lu. longipalpis, Lu. pseudolongipalpis*
Visceral leishmaniasis (Old World)	*Leishmania archibaldi, Le. donovani,*[b,c] *Le. infantum*[b,c]	Tropical and subtropical Europe, Asia and Africa	Canines, rats (Muridae)	*Phlebotomus ariasi, P. alexandri, P. argentipes, P. caucasicus, P. celiae, P. chinensis, P. kandelakii, P. langeroni, P. longicuspis, P. longiductus, P. martini, P. neglectus, P. orientalis, P. perfiliewi, P. perniciosus, P. smirnovi, P. tobbi, P. transcaucasicus, P. vansomerenae*

The reservoirs and sand fly vectors listed include known and suspected species. Sand fly generic names after Young and Duncan (1994).
[a]*Also can cause visceral infections.*
[b]*Also associated with mucocutaneous infections.*
[c]*Also can cause cutaneous infections.*

for both species. A third sand fly species, *Lutzomyia apache* in southwestern United States, has been geographically associated with outbreaks of vesicular stomatitis in cattle.

Overall, however, the transmission of vesicular stomatitis virus is complex and poorly understood. Human infections typically occur as occupational hazards in association with livestock. Farmers, ranchers, and veterinarians are at risk when in contact with vesicular fluids and tissues of infected animals. Some evidence of transmission to humans by arthropods has been indicated. Vesicular stomatitis virus has been isolated from nonbiting flies as well, including eye gnats (Chloropidae), anthomyiid flies (Anthomyiidae), and houseflies (Muscidae). Even the migratory grasshopper, *Melanoplus sanguinipes* (order Orthoptera: family Acrididae), apparently can become infected by ingestion of grass contaminated with vesicular fluids of infected cattle.

The more likely vectors are biting flies, including sand flies, blackflies (Simuliidae), and biting midges (Ceratopogonidae). Oral infection with infected blood has produced viral amplification and transmission of vesicular stomatitis in *Simulium sanguinipes* (Simuliidae) and *Culicoides sanguinipes* (Ceratopogonidae). In addition, outbreaks of vesicular stomatitis in cattle have been associated with dense populations of blackflies (*Simulium* spp.).

In Panama, vesicular stomatitis virus was found to infect members of seven mammalian orders, including opossums, xenarthrans (sloths, anteaters, and armadillos), bats, primates, carnivores, rodents, and lagomorphs (rabbits). Induced infection studies have established that a variety of wild and domestic ungulates are susceptible, including deer, horses, pigs, cattle, and sheep. Many of these wild and domestic vertebrates undoubtedly function as amplifying hosts of the virus. Because phlebotomine sand flies can transmit the virus transovarially, the flies are also potential insect reservoirs.

Chandipura Virus Disease

Chandipura virus is a *Vesiculovirus* with a structure and replication similar to those of vesicular stomatitis virus. It was first isolated from the blood of two humans in the central Indian state of Maharashtra in 1965. In the following years, Chandipura virus was detected only occasionally as a pathogen of humans. In 2003, however, an outbreak of disease attributed to Chandipura virus occurred in the state of Andhra Pradesh that affected 329 children with 183 fatalities (case fatality rate, 56%). In 2004, a similar outbreak occurred in Gujarat State, affecting 23 children with 18 fatalities. Symptoms included fever, sensory disorders, convulsions, vomiting, diarrhea, and encephalitis leading to coma and death. A hospital-based study conducted in Andhra Pradesh in 2005 and 2006 concluded that Chandipura virus is the major cause of an acute viral encephalitis in children in endemic areas during the early monsoon months (Tindale et al., 2008). Chandipura virus and/or neutralizing antibody against it also have been found in humans in Sri Lanka and in the West African countries of Senegal and Nigeria. Clinical cases in humans, however, have not been reported outside India.

Chandipura virus has been isolated from unidentified *Phlebotomus* and *Sergentomyia* species in India and Senegal. Transovarial transmission has been demonstrated experimentally in *P. papatasi*. The virus also has been identified in primates (macaques) in Sri Lanka, insectivores (hedgehogs) in Nigeria, and even-toed ungulates (pigs, buffalo, cattle, goats, and sheep) in India. Although human disease has been detected only in central India, the evidence of widespread virus occurrence in South Asia and Africa indicates a much broader human risk.

Sand Fly Fever

The sand fly fever viruses are members of the viral family Bunyaviridae, genus *Phlebovirus*. Most phleboviruses are transmitted by sand flies, although a quarter are tick-borne, and one, Rift Valley fever, is transmitted by mosquitoes. Five species (serotypes) of sand fly−borne viruses that have been isolated from humans include the following two geographic groups: (1) Chandiru, Chagres, and Punta Toro viruses from Central and South America; and (2) the Naples and Sicilian viruses of southern Europe and North Africa that are found eastward as far as China.

Sand fly fever has been known since the early 19th century as one of the first identified arboviruses. In 1908, an Austrian military commission demonstrated that the agent of sand fly fever is a filterable agent transmitted by the bite of *P. papatasi*. In the 1950s, the United States tested the sand fly fever virus on human volunteers as a potential biological warfare agent. More recently, sand fly fever virus has been listed as a virus of bioterrorism importance.

Sand fly fever is also called **phlebotomus fever** and **papatasi fever**, from the scientific name of the first known vector. In older literature, it was called *pappataci* or *papataci fever*, from the Italian word for sand fly. Sand fly fever is a common, nonlethal, self-limiting illness usually of 3 days' duration. Sudden-onset headache, fever, malaise, and nausea; limb, back, and retro-orbital pain; and rapid abatement of fever are characteristic symptoms. Encephalitis may occur in infections with the Naples species. The intrinsic incubation period is usually 3−4 days and up to 6 days. The virus is present in the blood from 1 day before to 2 days after onset of fever.

The known New World vectors of sand fly fever viruses are *Lu. trapidoi* and *Lu. ylephiletor,* and in the Old World, *P. papatasi*, *P. perfiliewi,* and *P. perniciosus*. The extrinsic incubation period is about 7 days, after which the sand fly

remains infective for the remainder of its 4-week life expectancy. In Europe, epidemics of sand fly fever commonly occur in the summer and fall, corresponding with the two generations of *P. papatasi*.

Sand fly fever virus and antibody have been found in antbirds (Passeriformes: Formicariidae), opossums and sloths in the New World, and a variety of rodents worldwide. Relatively few positives have been found in other Old World vertebrates. For example, in France only two positive sera were found (a sheep and a deer) in a sample of 668 sera from large wild mammals. However, in Iran, these viruses were widespread among the rodent populations. Thirteen of 38 gerbils were Sicilian seropositive and 12 of the 38 were positive for the Naples serotype. Whereas gerbils are the suspected reservoirs in Iran, the flies themselves are the probable principal reservoirs elsewhere. Natural and experimental transovarial transmission has been demonstrated in a several species of *Lutzomyia* and *Phlebotomus*.

Changuinola Virus Disease

Changuinola virus is a member of the viral family Reoviridae, genus *Orbivirus*. Reoviruses have been recovered worldwide from a broad range of birds and mammals. Members of the genus *Orbivirus* are arboviruses that primarily infect mammals. Changuinola virus was first isolated in Panama in 1959 and occurs widely in Panama, Colombia, and Brazil. In humans, it is associated with only a single documented case of clinical illness in Panama, with the symptom of acute fever, which was resolved without treatment.

Changuinola virus is most commonly recovered from sand flies but also occasionally from mosquitoes. Numerous isolations have been made from *Lutzomyia trapidoi* and *Lu. ylephiletor* in Panama and from *Lu. dasipodogeton*, *Lu. davisi*, *Lu. ubiquilatis*, *Lu. umbratilis*, and other, unidentified, sand flies in Brazil. Several isolations have been made from male sand flies, which indicate that transovarial transmission occurs in nature. Changuinola virus also has been recovered from xenarthrans (sloths and armadillos) and rodents (rice rats). Antibodies against Changuinola virus are widespread in sloths but infrequent in other wild vertebrates, which indicates that sloths are the normal vertebrate hosts (Fig. 12.8).

Seroprevalence of Changuinola virus is high in some parts of South America, but its extent and frequency are not known. Serologic studies suggest that most infections occur in childhood and are asymptomatic or produce a mild illness. The incubation period in humans is unknown but is estimated at 6–9 days in animals. Little is known regarding the prognosis of orbiviral infections, but full recovery is expected in most if not all cases.

FIGURE 12.8 A sloth from a nearby scrub forest, Puerto Suarez, Bolivia. Sloths are sources of blood for sand flies, as well as reservoirs for a variety of viruses and *Leishmania* species. *Photograph by Leonard E. Munstermann.*

Bartonellosis

Bartonellosis is a disease of humans caused by an α-proteobacterium, *Bartonella bacilliformis*. The organism was named for Alberto Barton, who in 1905 first discovered it in patient blood. Bartonellosis is also called **Carrión disease**, in honor of Daniel Alcides Carrión, a Peruvian medical student who gave his life in 1885 in the course of research on the nature of the disease. The bacteria are motile, aerobic, gram-negative bacilli that vary in size and shape from minute coccoid bodies to short rods up to 3 μm in length (Fig. 12.9). Under natural conditions *B. bacilliformis* grows on red blood cells and in the cytoplasm of the endothelial cells. Agents of **trench fever** and **cat-scratch disease** are also assigned to the genus *Bartonella*; hence, the term "bartonellosis" is also applied to these two diseases.

The intrinsic incubation period of bartonellosis is usually 2–3 weeks. The agent appears in the blood before the onset of illness and may persist for years afterward. The

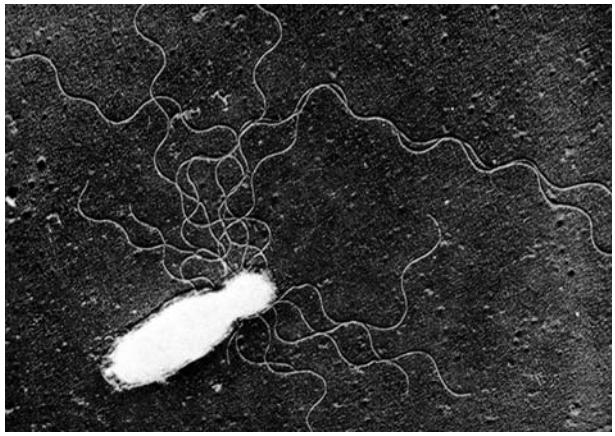


FIGURE 12.9 *Bartonella bacilliformis*, causative agent of Oroyo fever and verugana peruana. *Courtesy of U.S. Armed Forces Institute of Pathology (AFIP) 75–8592.*

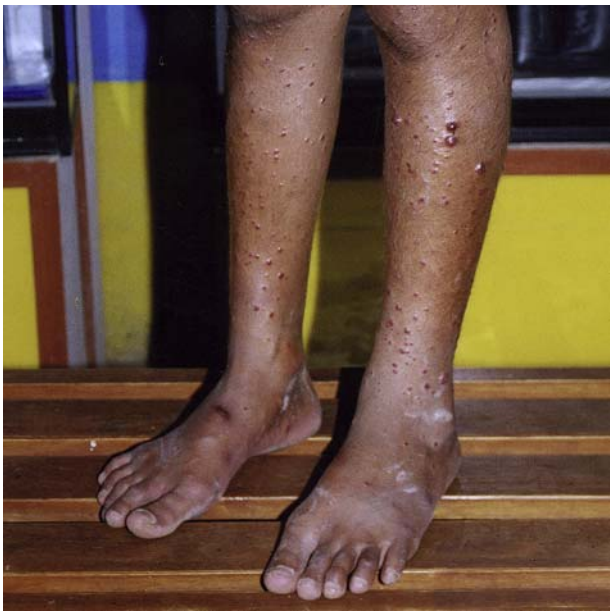


FIGURE 12.10 Verruga peruana, or Peruvian wart: nodular lesions on legs of a Peruvian male patient. *Photograph by Leonard E. Munstermann; courtesy of Abraham Caceres, Universidad Nacional Mayor de San Marcos, Lima, Peru.*

disease occurs in two distinct clinical forms: an acute, febrile anemia called **Oroya fever** (from La Oroya, the city where an epidemic killed thousands of railroad workers), and a benign dermal eruption called **verruga peruana** (Spanish for "Peruvian wart"). Oroya fever is characterized by fever, headache, muscle and joint pain, enlargement of the lymph nodes, and severe anemia. The case-fatality rate of untreated Oroya fever can range from 10% to 90%. Verruga peruana may be preceded by Oroya fever or by an asymptomatic infection, with an interval of weeks or months between. Verruga peruana has a preeruptive stage characterized by muscle, bone, and joint pain, followed by eruptions that may consist of widely disseminated small nodules, or with larger, deep-seated nodules most prominent on the limbs (Fig. 12.10). Individual nodules may enlarge and ulcerate. Verruga peruana may persist for months or years but is seldom fatal.

Bartonellosis occurs at altitudes between 500 and 3,000 m in the mountain valleys of Peru, Ecuador, and southwestern Colombia, as well as the coastal plains of Ecuador. Although bartonellosis has been known in Peru since the pre-Inca and Inca periods, its association with sand flies was first suspected by C. H. T. Townsend in 1913. Since then, Townsend's ideas about the sand fly association have been amply confirmed. Strong evidence has been presented that *Rattus* is a primary vertebrate reservoir (in addition to humans), but other details of the vector–parasite–vertebrate host relationship are not clear. The incriminated vectors in mountainous areas are *Lutzomyia verrucarum* and *Lu. peruensis* in Peru and *Lu. columbiana* in Colombia. The vector in lowland areas of Ecuador is not known.

Leishmaniasis

Leishmaniasis is a complex of sand fly-transmitted diseases widely distributed in tropical and subtropical areas of North and South America, Europe, Asia, and Africa (Fig. 12.11). The ecology of leishmaniasis varies widely in different geographic zones. In central Asia it occurs in semiarid and

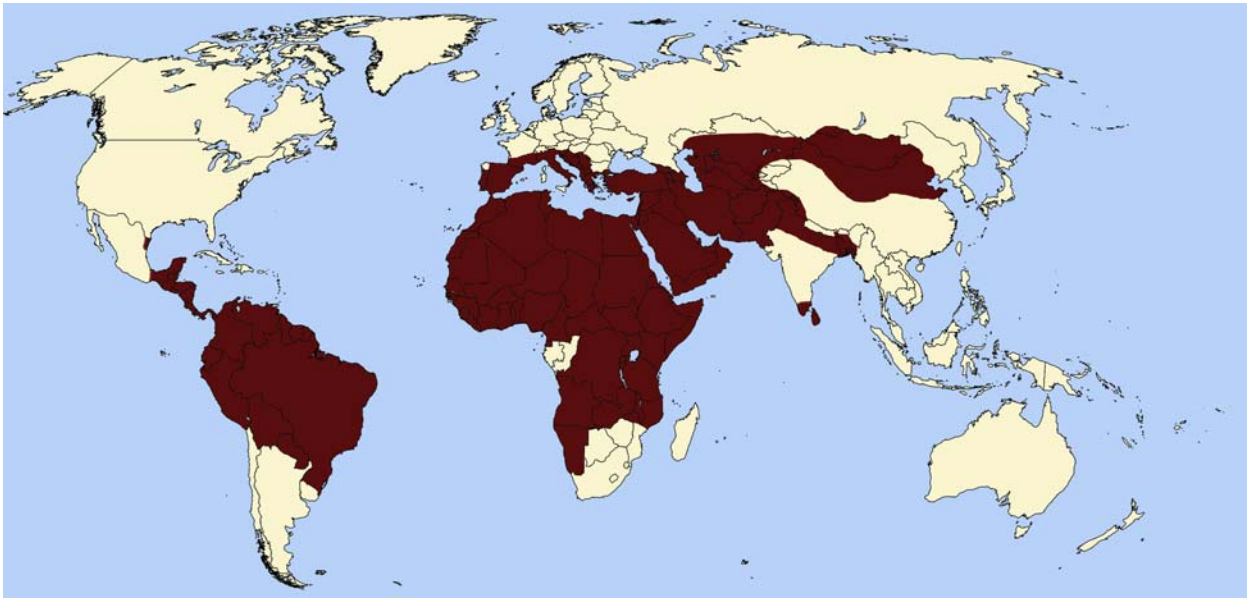


FIGURE 12.11 World distribution of human leishmaniases. *Based on data from the World Health Organization.*

arid situations. In the Mediterranean region and the Middle East, it is typically urban, and in Africa it is primarily rural. In the American tropics, it traditionally has been a forest disease but is advancing into urban areas. In Peru, it occurs in small towns and farms of high mountain valleys. Worldwide, about 200 million people in 98 countries are at risk of leishmaniasis with an estimated incidence of 12 million infections and some 2 million additional cases each year (WHO, 2017).

Leishmaniasis is caused by protozoan parasites of the genus *Leishmania*, a genus closely related to flagellated trypanosomes. Some 20 species have been recognized, largely based on genetic or immunologic criteria (Table 12.2). The disease and the genus were named for William B. Leishman, the British medical officer who discovered the organism early in the 19th century. The taxonomy of *Leishmania* remains controversial, largely because the molecular criteria have not been settled; this is despite investigations with electron micrography, enzyme electrophoresis, and nucleotide sequence analysis. Several taxa listed in Table 12.2 are relatively new; several others have been synonymized: e.g., *Leishmania chagasi* of the New World is the same as the Old World *Le. infantum*, and *Le. killicki* differs little from *Le. tropica* (for a detailed discussion, see Akhoundi et al., 2016). Identification of *Leishmania* species usually requires culture of the pathogen followed by immunological, biochemical, and molecular assay, although polymerase chain reaction and other diagnostics are under development.

The generalized life cycle of *Leishmania* species and the complexity of their developmental stages are shown in Figs. 12.12 and 12.13. In the vertebrate host, *Leishmania* is an obligate intracellular parasite of the macrophages of the

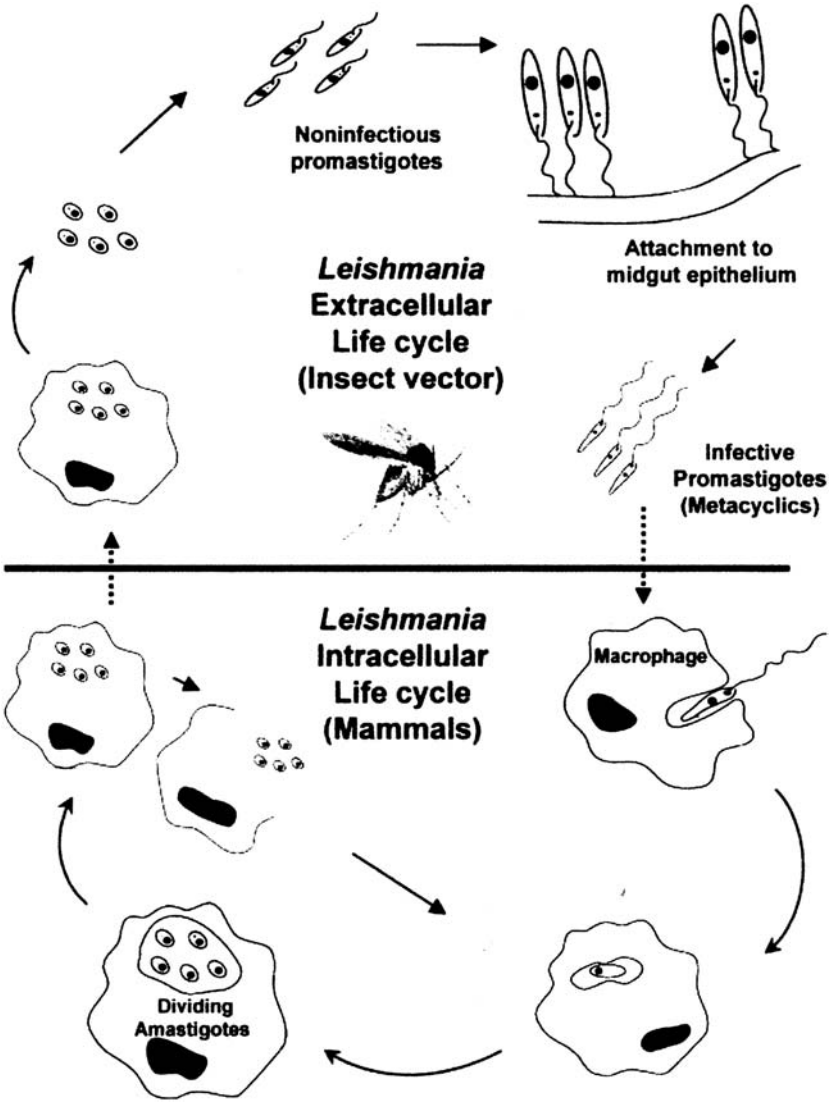


FIGURE 12.12 Generalized life cycle of *Leishmania* as it passes through the sand fly host and into the vertebrate host (Kamhawi et al., 2004).

lymphatic system and monocytes circulating in the blood. These forms represent the amastigote stage of development (Fig. 12.13A and B). Amastigotes are round or oval, 3−7 μm in diameter, with a round nucleus, rodlike kinetoplast (mitochondrion), and rudimentary, internal undulipodium (flagellum). The parasites multiply by binary fission within the macrophage, producing 50−200 new parasites. When the host cell ruptures, the *Leishmania* invade or are taken up by other cells.

In the sand fly, the parasites develop extracellularly from ingested amastigotes as they enter the alimentary canal (Fig. 12.13). Two morphological forms have been described, the promastigote and the paramastigote. Promastigotes are variable in shape, with a free undulipodium arising anteriorly. Promastigotes are elongate or pear-shaped and 5−24 μm long, with the kinetoplast situated anterior to the nucleus (Fig. 12.13C, E−G). Paramastigotes are round or oval and 3−7 μm in diameter, with the kinetoplast situated lateral to the nucleus (Fig. 12.13D and H). Promastigotes and paramastigotes may attach to the lining of the alimentary tract (haptomonad phase) or remain free-swimming (nectomonad phase). Haptomonads attach to cuticular surfaces of the foregut and hindgut by means of hemidesmosomes formed within the undulipodial tip

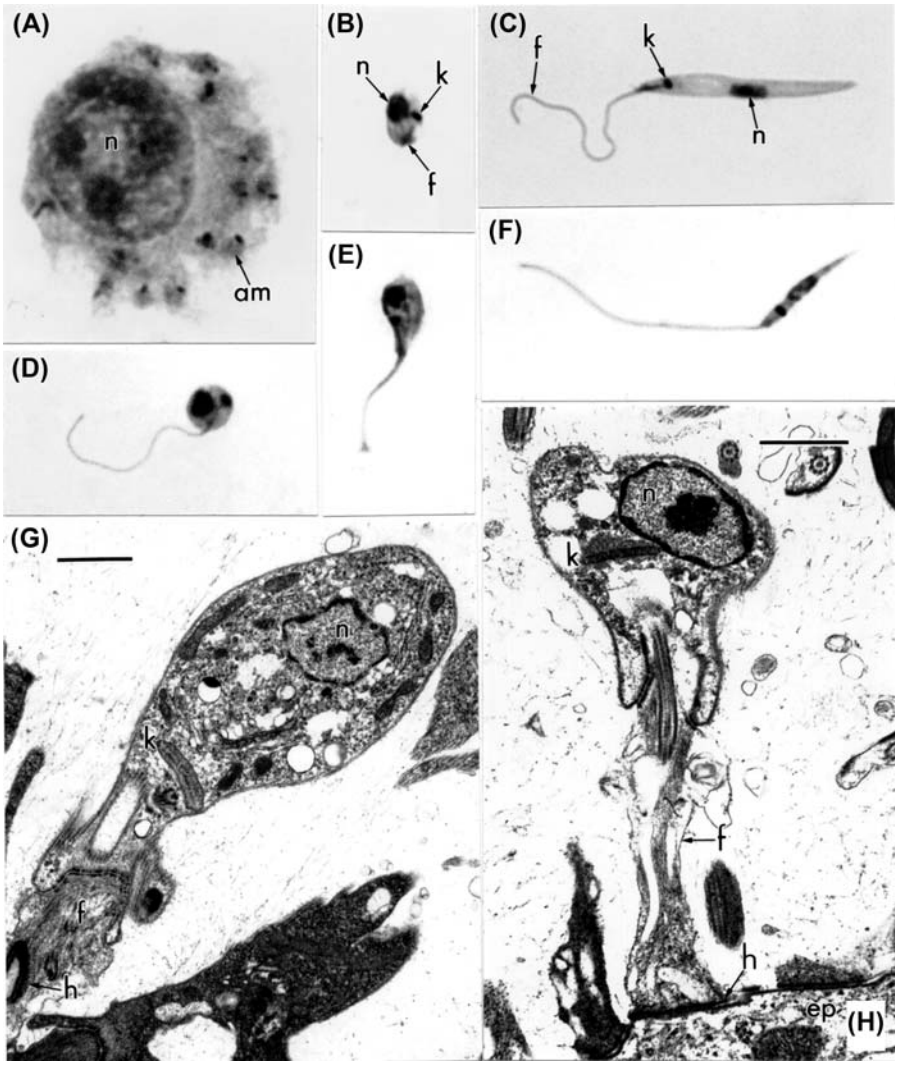


FIGURE 12.13 Developmental forms of *Leishmania* in sand flies. (A−F) Light micrographs; (G−H) electron micrographs, bars = 5 μm. (A) Ingested macrophage containing amastigotes; (B) amastigote; (C) elongate nectomonad promastigote; (D) nectomonad paramastigote; (E) pear-shaped haptomonad promastigote dissected from foregut intima; (F) metacyclic promastigote; (G) pear-shaped haptomonad promastigote attached in midgut; (H) haptomonad paramastigote attached in foregut. *am*, amastigote; *ep*, epithelium; *f*, flagellum; *h*, hemidesmosome; *k*, kinetoplast; *n*, nucleus. *Photographs by L. L. Walters; (B) Walters et al., 1989; (C) and (G) Walters et al., 1992; (D) and (E) Walters, L. L., 1993b. Life cycle of* Leishmania major *(Kinetoplastida; Trypanosomatidae) in the neotropical sand fly* Lutzomyia longipalpis *(Diptera: Psychodidae).* Journal of Medical Entomology, 30*, 669−718. 1993; (H) Walters, 1993a.*

(Fig. 12.13G and H). Nectomonads may temporarily attach in the midgut by interdigitation of the undulipodium with the epithelial microvilli.

In the sand fly blood meal, amastigotes transform into short promastigotes and mature into elongate promastigotes within 3 days of ingestion (Fig. 12.12) Initially, the peritrophic matrix surrounding the blood meal prevents establishment of the parasites in the gut. After natural or parasite-facilitated degradation of the matrix envelope, the parasites develop as promastigotes and paramastigotes in the midgut (as in the subgenus *Leishmania*) or in both hindgut and midgut (subgenus *Viannia*) and subsequently migrate to the foregut. The relatively short, slender, highly motile nectomonad promastigotes have very long undulapodia (flagella) and are regarded as the infective, or metacyclic, forms (Fig. 12.13F). Metacyclic promastigotes develop in the midgut, foregut, and mouthparts and are transmitted to new hosts during blood feeding. Parasite colonization of the foregut impedes the flow of blood during feeding, resulting in repeated probing and enhanced probability of transmission. The extrinsic incubation period is species- and temperature-dependent, ranging from 4 to 17 days.

During the feeding process, ample quantities of saliva are injected at the bite site. Thirty distinct proteins have been characterized in the sand fly saliva. Some are anticoagulants to stimulate blood flow and prevent clogging of the wound, the mouthparts, or pharynx. A well-studied protein, maxadilin, is a potent vasodilator for increasing blood flow. Several are immunogenic, i.e., the human and animal hosts develop antibodies to salivary proteins and seem to provide protection against the *Leishmania*. The precise effect of the immunogenic proteins on parasite infectivity is complicated, however. Other gut microbiota egested during feeding may produce an inflammatory response that facilitates parasite dissemination to host organs.

The relationship of parasite, vector, and vertebrate host in leishmaniasis is an ancient one. Fossil sand flies, *Paleomyia burmitis*, from the Cretaceous amber of Burma, have been found to contain blood that is believed to be dinosaur blood. Remarkably, when the blood meals of 21 of the fossil flies were examined microscopically, developmental stages of a fossil proto leishmania form, *Paleoleishmania proterus*, were identified in 10 of them (Poinar and Poinar, 2008).

Leishmania multiply by binary fission at many points in the life cycle. Clonal reproduction by mitosis has largely replaced segregation and recombination by meiosis in the evolution of the genus. Because of the degree of intraspecific genetic variability and patterns of variation in clinical manifestations, a degree of genetic recombination has been recognized for some time, particularly in the *Le. (Viannia)* species group. Direct evidence for recombination among microsatellite profiles has been documented in *Leishmania (Viannia) braziliensis* and *Leishmania (Viannia) guyanensis*. Kuhls et al. (2013) conclude that "Though clearly departing from panmixia, sporadic, but long-term sustained recombination might explain the tremendous genetic diversity and limited population structure found for [these] *Le.* (*Viannia*) strains."

Leishmaniasis, particularly the visceral form, has emerged as an opportunistic disease in immunocompromised people, who may acquire the disease by reactivation of latent infections or through exchange of infected needles and syringes among intravenous drug users. Coinfections of *Leishmania* and HIV have been reported from 35 countries, most notably Spain, Portugal, France, and Italy. Persons coinfected with *Leishmania* and HIV can infect sand flies and act as human reservoirs in zoonotic foci to shift the local epidemiology toward a human-to-human transmission pattern.

Cutaneous Leishmaniasis

Leishmaniasis occurs in two principal clinical forms, known as **cutaneous leishmaniasis** and **visceral leishmaniasis**. Whereas a given species of *Leishmania* typically produces one or the other clinical form, some, including *Leishmania amazonensis*, *Le. infantum*, and *Le. guyanensis* in the New World and *Leishmania donovani*, *Le. infantum*, and *Le. tropica* in the Old World, can produce both (Table 12.2). A third clinical form, **mucocutaneous leishmaniasis**, has a more serious external manifestation associated with, for example, severe erosion of the nasopharynx and eventual facial collapse. This form is produced by *Leishmania (Viannia)* species that are heavily coinfected with an RNA virus. Apparently, these viruses are able to deflect host immune response from the replicating *Leishmania*, leading to less restrained parasite invasion (Ives et al., 2011).

After an intrinsic incubation period of a week to many months, cutaneous leishmaniasis (dermal leishmaniasis) begins with a macule at the site of inoculation by the bite of an infected sand fly. The macule develops into a papule that enlarges and typically becomes a slow-developing and relatively painless ulcer. The lesion may be single or multiple (Fig. 12.14) or, occasionally, nonulcerative and diffuse (Fig. 12.15). The term "diffuse (disseminated) cutaneous leishmaniasis" is applied to a progressive, anergic (no immunity to an antigen), nonulcerative condition resulting from the lack of a delayed-type hypersensitivity in the patient. In some cases, however, lesions can persist to form large ulcers (Fig. 12.16). Gradually, infected lesions are replaced by scar tissue, which can be noticeably disfiguring, especially on the face (Fig. 12.17). New areas of the body become involved by extension of the primary lesions or by metastasis via the blood or lymph.

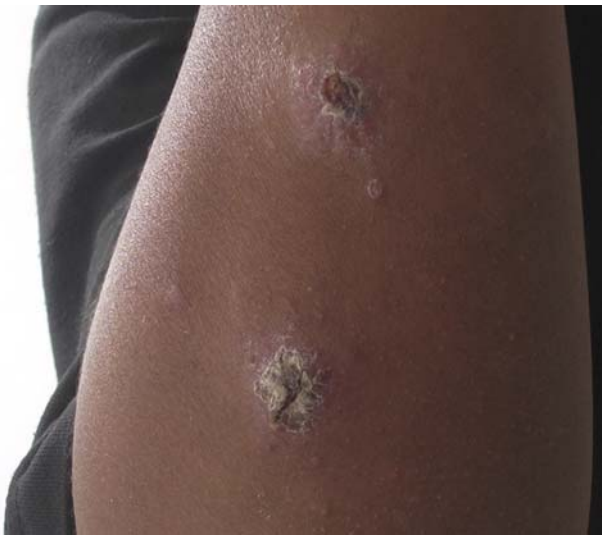


FIGURE 12.14 Ulcerous lesions with scabs at bite sites on forearm caused by *Leishmania major*. *Photograph by Karim Aoun and Aïda Bouratbine; Aoun and Bouratbine, 2014.*

Mucocutaneous leishmaniasis, involving lesions of the mucous membranes of the nose, mouth, and pharynx (mucosal or nasopharyngeal leishmaniasis) may develop after the primary lesion has healed, or in the absence of a recognized primary lesion. *Leishmania* species with known potential to cause mucocutaneous infections are indicated in Table 12.2.

Several clinical manifestations of cutaneous leishmaniasis have acquired specific common names: In the Old

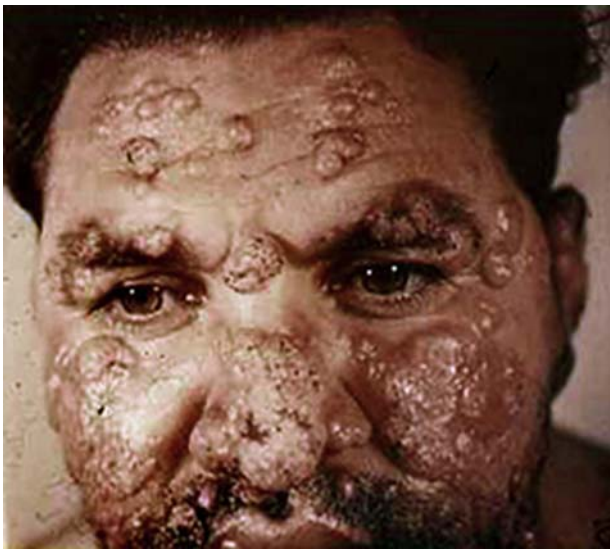


FIGURE 12.15 Severe case of diffuse (disseminated) leishmaniasis in a male patient. *Otis Historical Archives of "National Museum of Health & Medicine", OTIS Archive 1.*

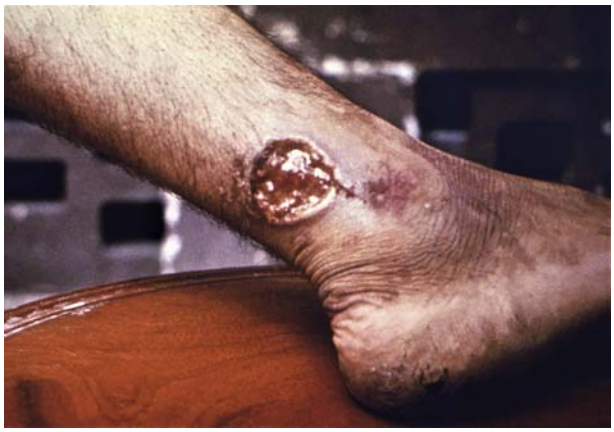


FIGURE 12.16 Case of cutaneous leishmaniasis lesion on right ankle of a male patient; characteristic chronic ulcer with erythema, nodular interior, and a raised border. *Courtesy of Public Health Image Library, Centers for Disease Control and Prevention, USA; PHIL#15609.*

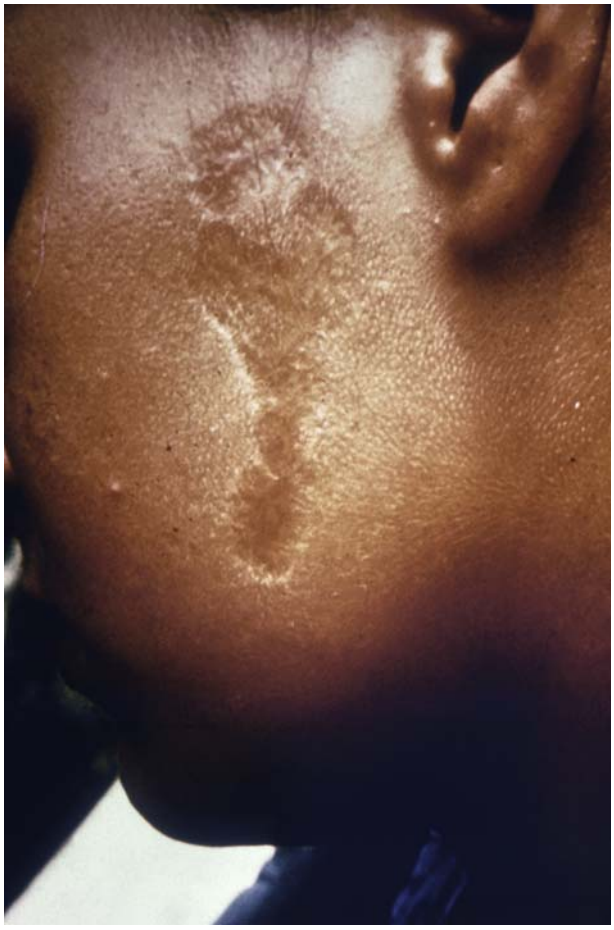


FIGURE 12.17 A characteristic scar of a healed leishmanial lesion on the face of a young female. *Courtesy of Public Health Image Library, Centers for Disease Control and Prevention, USA; PHIL# 15334.*

World, condition characterized by single or multiple cutaneous ulcers owing to *Leishmaniasis tropica* or *Le. major* has been called **oriental sore**, **tropical sore**, **Aleppo boil** (or Aleppo evil), **Baghdad boil**, or **Delhi boil**. In Central America, the condition characterized by single or multiple ulcers on the face or ears caused by *Leishmania mexicana* is known as **chiclero ulcer**. In French Guiana, the condition characterized by moderate ulcers due to *Leishmania amazonensis* or *Le. guyanensis* is known as *pian bois* (French for "forest yaws"). In Peru and Ecuador, the condition is characterized by numerous, small, benign lesions caused by *Leishmania peruviana* or *Le. mexicana* and is known as *uta*. In South America, mucocutaneous leishmaniasis caused by *Le. amazonensis* or *Le. braziliensis* is known as **espundia** (Spanish for "sponge").

Cutaneous leishmaniasis may be self-limiting or chronic. Chiclero ulcer and diffuse cutaneous leishmaniasis are chronic, and diffuse cutaneous leishmaniasis is resistant to treatment. Mucocutaneous leishmaniasis persists for many years and ultimately may be fatal. Infections of *Le. tropica* may recur at or near the site of the healed ulcer after apparent cure, a condition clinically known as **leishmaniasis recidivans** or **chronic relapsing leishmaniasis**. Lesions of cutaneous and mucocutaneous leishmaniasis are prone to secondary infections by bacteria and fungi and to infestation by fly larvae (myiasis). Disfiguring scars remain after healing.

Known and suspected vectors and reservoirs of cutaneous leishmaniasis are shown in Table 12.2. Transmission of *Le. major* by *Phlebotomus caucasicus* in semiarid regions of central Asia is a classic example of an endemic zoonosis. In this area, *P. caucasicus* develops in burrows of gerbils (*Rhombomys opimus*) and ground squirrels (*Spermophilopsis leptodactylus*) and transmits *Le. major* from animal to animal and from animals to humans. When gerbils were eradicated from the vicinity of a construction camp in Turkestan, leishmaniasis disappeared among the construction workers.

Cutaneous leishmaniasis due to *Le. mexicana* occurs widely but rarely in the United States in south-central Texas (Bexar, Cameron, Gonzales, Uvalde, and Wells counties) and the adjoining states of Coahuila, Nuevo Leon, and Tamaulipas in Mexico. Both typical and diffuse forms of the disease have been reported. *Lutzomyia anthophora* is believed to transmit the disease among woodrats (*Neotoma micropus*), and *Lu. diabolica* is suspected to be a bridge vector between wood rats and humans. Seropositive coyotes and an infected cat have been found in southern Texas. An additional focus of *Le. mexicana* in wood rats (*Neotoma albigula*) has been identified in Pima County, Arizona. *Lutzomyia anthophora* is considered to be the most likely vector.

Visceral Leishmaniasis

Visceral leishmaniasis also is known as **kala-azar** (from Hindi for "black fever") and **dumdum fever** (from Dum Dum, a village and former British arsenal near Calcutta, now the site of an international airport). It is a chronic systemic disease that begins with an inconspicuous cutaneous lesion at the site of inoculation by the bite of an infective sand fly. From this site, the parasites are distributed through the body in the bloodstream, producing chronic fever, enlargement of the lymph nodes, liver, and spleen, deficiency of red and white blood cells and blood platelets, and progressive emaciation and weakness. Early clinical manifestations are variable. Inapparent and subclinical infections caused by *Le. tropica* have been called **viscerotropic leishmaniasis**. Clinically evident visceral leishmaniasis is usually fatal if not treated. The intrinsic incubation period is usually 2–4 months. Cutaneous lesions may appear after apparent recovery or cure and may persist for up to 20 years in the absence of treatment. Such lesions are known as **post–kala-azar cutaneous leishmaniasis** or **post–kala-azar dermal leishmaniasis**.

Known and suspected vectors and reservoirs of visceral leishmaniasis are shown in Table 12.2. In India, Nepal, and Bangladesh, humans and sand flies are the only known hosts. Transmission of *Le. donovani* by *Phlebotomus argentipes* in the Ganges and Brahmaputra River basins of India is a classic example of an epidemic anthroponosis. Between 1824 and 1981, a series of nine major epidemics of visceral leishmaniasis occurred in this region. Some were so severe that entire villages were depopulated and extensive areas were abandoned. Similarly, between 1988 and 1993, a major epidemic of visceral leishmaniasis occurred in the Sudan, affecting 600,000–700,000 people and killing 40,000 where *Lu. donovani* was transmitted by *P. martini*. The Sudanese epidemic was exacerbated by conditions of malnutrition, famine, displacement of persons, and disruption of health services caused by civil war.

VETERINARY IMPORTANCE

Members of the Psychodinae have no known veterinary importance. Members of the Phlebotominae, however, can be pests of livestock, pets, and wildlife in places where they are abundant. Their contribution to the overall economic loss caused by its bites is unknown. In addition, sand flies can transmit *Leishmania* to dogs and cats and also may have a role in the transmission of vesicular stomatitis virus among livestock.

Leishmaniasis

Veterinary forms of leishmaniasis are **canine leishmaniasis** and **feline leishmaniasis**. Both dogs and cats are susceptible to cutaneous leishmaniasis. The lesions usually occur on the nose and ears. Dogs are also susceptible to visceral leishmaniasis and may be important reservoirs. The incubation period may be months or years. Infection in dogs is prevalent in Brazil, China, and the Mediterranean region.

Imported and autochthonous cases of canine leishmaniasis occur in the United States. Cases have occurred in dogs imported into the country and in dogs returning from foreign travel. Most are caused by *Le. infantum* or *Le. donovani*. Sporadic cases of autochthonous canine leishmaniasis have been reported from Texas, Oklahoma, Kansas, and Ohio for many years. The parasites have been variously identified as *Le. infantum* or *Le. mexicana*. The sand fly vectors are unknown.

In 1999, an outbreak of visceral leishmaniasis resulting from *Le. infantum* occurred among foxhounds in a New York foxhunting club, eventually resulting in 20 dog fatalities. Investigators uncovered a similar outbreak among foxhounds in a Michigan club in 1989. Subsequently, seropositive dogs have been found in clubs throughout the United States (21 states) and in the Canadian province of Ontario. Because cases have been limited to foxhounds, one inference is that direct dog-to-dog transmission may occur during annual foxhound shows. Direct transmission of *Le. infantum* has been demonstrated experimentally in mice. In addition, transplacental transmission of *Le. infantum* has been demonstrated experimentally in dogs, and a case was reported of transmission of *Le. donovani* to a dog by blood transfusion. Although *Lutzomyia vexator* is widespread and abundant in Dutchess County, New York, where the 1999 outbreak occurred, it apparently feeds only on reptiles. At present, no evidence other than sympatry implicates it as a vector of canine visceral leishmaniasis.

Vesicular Stomatitis Virus Disease

In veterinary practice, vesicular stomatitis virus disease is so named because of its oral symptoms in livestock. Vesicular stomatitis is an acute, febrile, weakening viral disease of horses, cattle, swine, and occasionally sheep and goats. It is characterized by small, superficial, erosive blisters that form in and about the mouth and on the feet, teats, and occasionally other parts of the body. Because the symptoms closely resemble those of foot-and-mouth disease, vesicular stomatitis also is known as **pseudo-foot and mouth disease**. Susceptibility depends on the immune status of the host animal. Nonimmune cattle are 100% susceptible, and up to 90% develop clinical disease. The intrinsic incubation period is 2–8 days. The disease is usually self-limiting, with recovery in about 2 weeks, but recrudescence or reinfection may occur. Economic losses result from the reduced condition of infected animals, reduced meat and milk production, and secondary bacterial infections. Vesicular stomatitis virus is on the US Department of Agriculture High Consequence list and the Australian Group Core list of potential biological warfare agents.

The Indiana, New Jersey, and Alagoas serotypes of vesicular stomatitis virus cause disease in domestic animals across the Americas. Vesicular stomatitis is endemic and occurs year-round in tropical regions. In temperate regions, it tends to be epidemic, occurring primarily in late summer and early fall. Opossums, monkeys, porcupines, raccoons, bobcats, and pronghorns are suspected reservoirs. Antibodies occur in domestic and wild dogs.

Lutzomyia shannoni is a proven vector of the New Jersey serotype among feral pigs on Ossabaw Island, Georgia, and *Lu. trapidoi* is a proven vector of the Indiana serotype in Latin America. *Lutzomyia ylephiletor* is an additional suspect vector. Other modes of transmission of vesicular stomatitis have been discussed earlier in connection with the public health importance of sand flies.

PREVENTION AND CONTROL

Psychodinae

Control of moth flies in buildings and homes depends on the removal of gelatinous films and slimes that form in household drains and condensation pans of refrigeration and air-conditioning units, as well as other situations in which oviposition and larval development occur. Mechanical cleaning is most effective, but infestations in drains can also be eliminated by flushing with cleaning materials followed by very hot water. Effective drain cleaners based on bacterial cultures have been developed to remove and prevent the formation of slime. An insect growth regulator, hydroprene, is also available to treat drains.

Larvae can be eliminated from the filters of sewage treatment plants by flooding for 24 h. Flooding does not affect the eggs and must be repeated periodically for continuous control. Larvae also can be eliminated from the filters by adding insecticides to the flow. An approved insecticide formulation and dose must be used to avoid harm to the filters and the downstream environment. Destruction of large numbers of larvae in sewage treatment plants may create an odor problem near the plant owing to decomposition. The bacterium *Clostridium bifermentans* serovar *malaysia* has been reported to be highly toxic to larvae of *Ps. alternata*, but its use as a biological control agent has not been demonstrated.

Adult moth flies can be controlled by applying insecticides to resting sites on structures and surrounding areas. Moth flies are susceptible to all classes of insecticides, but the use of approved nonpersistent pyrethroid insecticides is recommended. Residual insecticides are rarely necessary.

Phlebotominae

Methods for investigating larval habitats of phlebotomine sand flies include direct examination of soil and litter, extraction by Berlese funnel, wet sieving, flotation, and emergence trapping. These methods are tedious and largely unproductive. Surveillance and collection methods for adult sand flies include trapping and aspiration from resting sites, humans, and bait animals. Effective trap designs include light traps, bait traps, sticky traps, and flight traps. Smoke, insect repellent spray, or a twig or stick can be used to flush sand flies from inaccessible resting sites for collection.

Insect **repellents** and protective clothing can provide effective personal protection. Sand flies cannot bite through outdoor clothing because of their relatively short mouthparts. Long sleeves, trousers, and socks are recommended in areas where sand flies are active, as well as an effective repellent lotion or spray applied to exposed skin. The leading repellent for personal use is diethylmethylbenzamide, formerly known as diethyltoluamide (DEET). Where sand flies are present, campsites and outdoor sleeping areas are best selected to be dry, open to the wind, and away from potential larval sites. Sand flies can pass through or bite through untreated standard 16- and 18-mesh mosquito netting and screening. Fine-mesh nets and screens are not generally used because they impede circulation of the air. However, repellent- or insecticide-treated, standard- or wide-mesh net jackets and hoods, head nets, tent openings, screens, and bed nets provide effective protection. Treated bed nets are to be used when sleeping outdoors, and treated window screens, screen doors, and bed nets may be used for protection indoors. Synthetic pyrethroid insecticides, including permethrin, cypermethrin, cyfluthrin, and etofenprox, are recommended by the World Health Organization to treat bed nets. In connection with personal protection, the endophilic New World vector *Lu. verrucarum* has been reported to crawl beneath untreated clothing and bedding to reach the skin.

Aerosol formulations of natural and synthetic pyrethroid **insecticides** provide effective control of sand flies when used indoors during the daily period of sand fly activity. Insecticidal smoke produced by "mosquito coils" is a useful and popular control measure for indoor spaces, but these have no appreciable residual effect. Mosquito coils are made by combining natural pyrethrum or a synthetic pyrethroid (commonly allethrin or prallethrin) and a slow-burning organic filler (punk) with a binder, and then forming the mixture into a flat spiral. Mosquito coils are believed to have evolved as a folk remedy from the joss sticks burned as incense in Eastern temples.

Organochlorine, organophosphate, carbamate, and synthetic pyrethroid insecticides are effective for residual control of adult sand flies. Indoor treatments of houses are directed toward inside walls and ceilings, window and door frames, and screens. Pyrethroid-treated curtains for eaves, doors, windows, and walls have been used for sand fly control in homes in Sudan, Burkina Faso, Venezuela, and Italy. Outdoor treatments are directed toward putative larval habitats and adult resting sites such as buildings, walls, caves, animal burrows, and tree trunks. Insecticide resistance has been reported only for organochlorine insecticides applied to *P. argentipes* and *P. papatasi*.

From 1955 to 1969, the World Health Organization conducted a program for the global eradication of malaria by the indoor treatment of houses with dichlorodiphenyltrichloroethane (DDT). An unexpected collateral benefit of the program was a reduction in the incidence of leishmaniasis in areas where domiciliary transmission occurred. When the program was discontinued, the incidence of leishmaniasis increased, along with the incidence of malaria, in at least five countries: India, Bangladesh, Nepal, Greece, and Colombia. An especially notable deadly epidemic of visceral leishmaniasis occurred in India from 1973 to 1981. These events provided a dramatic demonstration of the effectiveness of residual insecticides in the control of vector-borne disease.

Environmental measures for sand fly control include eliminating larval habitats and adult resting sites. Resting sites of peridomestic *Phlebotomus papatasi* can be reduced by filling crevices in walls, ceilings, and floors of houses, outbuildings, and masonry structures and by clearing outdoor areas of accumulations of refuse, stone, and other materials. In Italy, *P. perfiliewi* was eliminated from farmhouses by relocating storage piles of farm manure in which the sand fly larvae were developing to safe distances from houses. In Kenya, *P. martini* was eliminated from houses by destroying termite mounds located within 20 m of homes. The mounds provided sites for both sand fly larvae and resting adults. In Panama and French Guiana, larval and adult sites of local forest species were eliminated by deforestation of areas around small towns and settlements.

Biological and genetic methods for sand fly control have not been demonstrated. The bacteria *Bacillus sphaericus* and *Bacillus thuringiensis* var. *israelensis*

are potential agents for biological control of the larval stage. Rickettsial endosymbionts of the genus *Wohlbachia* have been found in New and Old World sand flies. In the mosquito *Culex pipiens*, *Wohlbachia* causes a form of male sterility known as cytoplasmic incompatibility; however, this interaction has not been observed in sand flies. Several gregarines (Apicomplexa), microsporidia (Microspora), and roundworms (Nematoda) are parasites in New and Old World sand flies. Adult sand fly predators include a cricket, *Anaxipha gracilis* (Orthoptera: Gryllidae) in the Americas and a thread-legged bug *Ploiaria domestica* (Hemiptera: Reduviidae) in the Old World.

Elimination of **animal reservoirs** of zoonotic leishmaniasis is feasible in some situations. Culling of infected dogs has been practiced in China and Brazil. Although stray dogs are a major reservoir in the Americas, this practice has not had a pronounced effect on human incidence. Another approach has been the mandatory treatment of all infected dogs or mandatory pyrethroid-impregnated collars. The reduction of feral dog and jackal populations by proper disposal of offal from slaughterhouses and poultry farms has effectively reduced the incidence of visceral leishmaniasis in Iraq. The fat sand rat *Psammomys obesus* has been controlled in Jordan by flooding, digging, or deep plowing of burrows. Populations of the great gerbil *R. opimus* have been reduced in Kazakhstan by deep plowing of burrows and dispersal of bait consisting of wheat treated with zinc phosphide or anticoagulant rodenticides.

For bacterial diseases, penicillin, streptomycin, chloramphenicol, and tetracyclines are effective in reducing fever and bacteremia (e.g., in Oroya fever and bartonellosis). Specific treatments for bartonellosis include streptomycin and rifampin. First-line drugs for treatment of leishmaniasis are pentavalent antimonials, most importantly sodium stibogluconate and meglumine antimonite. An extended course of multiple treatments is always required.

A kind of folk **vaccination** called leishmanization has been practiced in southwest Asia since ancient times. Typically, children are inoculated on a part of the body normally covered by clothing. The inoculum consists of living amastigotes from an active lesion of cutaneous leishmaniasis. The lesion that develops at the site of inoculation eventually heals, leaving the child immune to reinfection and protected from unsightly scarring of the face or another visible body part. Large-scale government-sponsored programs of leishmanization of children and adults have been conducted, sometimes using promastigotes from culture in lieu of amastigotes from lesions. This method was used most notably in Uzbekistan and in Iran during its 1980–1988 war with Iraq. Although ongoing research is directed toward developing effective vaccines for the leishmanias, successful vaccines have not been forthcoming.

REFERENCES AND FURTHER READING

Akhoundi, M., Kuhls, K., Cannet, A., Votýpka, J., Marty, P., Delaunay, P., et al. (2016). A historical overview of the classification, evolution, and dispersion of *Leishmania* parasites and sandflies. *PLoS Neglected Tropical Diseases, 10*(3), e0004349. https://doi.org/10.1371/journal.pntd.0004349.

Alexander, B. (1995). A review of bartonellosis in Ecuador and Colombia. *The American Journal of Tropical Medicine and Hygiene, 52*, 354–359.

Aoun, K., & Bouratbine, A. (2014). Cutaneous leishmaniasis in North Africa: A review. *Parasite, 21*, 14. https://doi.org/10.1051/parasite/2014014.

Artemiev, M. M. (1991). A classification of the subfamily Phlebotominae. *Parassitologia, 33*(Supplement 1), 69–77.

Azar, D., Huang, D., Cai, C., & Nel, A. (2015). The first trichomyiine from Burmese Cretaceous amber (Diptera, Psychodidae, Trichomyiinae). *Cretaceous Research, 53*, 48–58.

Beati, L., Cáceres, A. G., Lee, J. A., & Munstermann, L. E. (2004). Systematic relationships among *Lutzomyia* sand flies (Diptera: Psychodidae) of Peru and Colombia based on the analysis of 12S and 28S ribosomal DNA sequences. *International Journal for Parasitology, 34*, 225–234.

Belen, A., & Alten, B. (2006). Variation in life table characteristics among populations of *Phlebotomus papatasi* at different altitudes. *Journal of Vector Ecology, 31*, 35–44.

Cárdenas, E., Corredor, D., Munstermann, L. E., & Ferro, C. (1999). Reproductive biology of *Lutzomyia shannoni* (Dyar) (Diptera: Psychodidae) under experimental conditions. *Journal of Vector Ecology, 24*, 158–170.

Chang, K.-P., & Bray, R. S. (Eds.). (1985). *Leishmaniasis*. London: Elsevier.

Charlab, R., Valenzuela, J. G., Rowton, E. D., & Rebeiro, J. M. C. (1999). Toward an understanding of the biochemical and pharmacological complexity of the saliva of a hematophagous sand fly, *Lutzomyia longipalpis*. *Proceedings of the National Academy of Science U. S. A., 96*, 15155–15160.

Comer, J. A., & Tesh, R. B. (1991). Phlebotomine sand flies as vectors of vesiculoviruses: A review. *Parassitologia, 33*(Supplement 1), 143–150.

Davidson, I. H. (1990). *Sandflies of Africa south of the Sahara: Taxonomy and systematics of the genus* Sergentomyia. Johannesburg: South African Institute for Medical Research.

De Almeida, D. N., Oliveira, R. D. S., Brazil, B. G., & Soares, M. J. (2004). Patterns of exochorion ornaments on eggs of seven South American species of *Lutzomyia* sand flies (Diptera: Psychodidae). *Journal of Medical Entomology, 41*, 819–825.

GBIF. Global Biodiversity Information Facility. (1999). *Report of the Biodiversity Informatics Subgroup of the Organization for economic Cooperation and development*. http://www.gbif.org/species/9164.

Galati, E. A. B. (2003). Classificação de Phlebotominae. In E. F. Rangel, & R. Lainson (Eds.), *Flebotomíneos do Brasil* (pp. 23–176). Rio de Janeiro: Editora Fiocruz. Ch. 2.1-2.

Gutiérez, M. A. C., Vivero, R. J., Vélez, I. D., Porter, C. H., & Uribe, S. (2015). DNA barcoding for the identification of sand fly species (Diptera. Psychodidae. Phlebotominae) in Colombia. *PLoS One*. http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0085496.

Ives, A., Ronet, C., Prevel, F., Ruzzante, G., Fuertes-Marraco, S., Schutz, F., et al. (2011). *Leishmania* virus controls severity of mucocutaneous leishmaniasis. *Science, 331*(6018), 775–778.

Jobling, B. (1987). *Anatomical drawings of biting flies*. British Museum of Natural History, 119 pp.

Kamhawi, S., Ramalho-Ortigao, M., Pharn, V. M., Kuman, S., Lawyer, P. G., Turco, S. J., et al. (2004). A role for insect galectins in parasite survival. *Cell, 119*, 329–341.

Kasap, O. E., & Alten, B. (2006). Comparative demography of the sand fly *Phlebotomus papatasi* (Diptera: Psychodidae) at constant temperatures. *Journal of Vector Ecology, 31*, 378–385.

Kerr, S. F. (2000). Palaearctic origin of *leishmania*. *Memoirs do Instituto Oswaldo Cruz, Rio de Janeiro, 95*, 75–80.

Kuhls, K., Cupolillo, E., Silva, S. O., Schweynoch, C., Boité, M. C., Mello, M. N., et al. (2013). Population structure and evidence for both clonality and recombination among Brazilian strains of the subgenus *Leishmania (Viannia)*. *PLoS Neglected Tropical Diseases, 7*(10), e2490. https://doi.org/10.1371/journal.pntd.0002490.

Lewis, D. J. (1982). A taxonomic review of the genus *Phlebotomus* (Diptera: Psychodidae). *Bulletin of the British Museum of Natural History, 45*, 121–209.

Llanes, A., Restrepo, C. M., Del Vecchio, G., Anguizola, F. J., & Lleonart, R. (2015). The genome of *Leishmaniasis panamensis*: Insights into genomics of the *L. (Viannia)* subgenus. *Scientific Reports, 5*(8550). https://doi.org/10.1038/srep08550.

Munstermann, L. E. (2004a). Care, maintenance, and experimental infection of phlebotomine sand flies. Ch. 56. In W. C. Marquardt, W. C. Black, J. Freier, H. Hagedorn, J. Hemingway, S. Higgs, et al. (Eds.), *Biology of disease vectors* (2nd ed., pp. 757–762). San Diego, CA: Elsevier Science (USA).

Munstermann, L. E. (2004b). Phlebotomine sand flies, the psychodidae. Ch. 12. In W. C. Marquardt, W. C. Black, J. Freier, H. Hagedorn, J. Hemingway, S. Higgs, et al. (Eds.), *Biology of disease vectors* (2nd ed., pp. 141–151). San Diego, CA: Elsevier Science (USA).

Nieves, E., & Pimenta, P. F. P. (2000). Development of *Leishmania (Viannia) braziliensis* and *Leishmania (Leishmania) amazonensis* in the sand fly *Lutzomyia migonei* (Diptera: Psychodidae). *Journal of Medical Entomology, 37*, 134–140.

Perfil'ev, P. P. (1968). Phlebotomidae (Sandflies). In *Fauna of the U.S.S.R* (Vol. III, No. 2). Jerusalem: Israel Program for Scientific Translations. Translated from the Russian.

Poinar, G., & Poinar, R. (2008). *What bugged dinosaurs? Insects, disease, and death in the Cretaceous*. Princeton, NJ: Princeton University Press.

Quate, L. W. (1955). A revision of the Psychodidae (Diptera) in America north of Mexico. In *University of California Publications in Entomology* (Vol. 10, pp. 103–273).

Quate, L. W., & Vickeroth, J. R. (1981). Psychodidae. In J. F. McAlpine, B. V. Peterson, G. E. Shewell, H. J. Tesky, J. H. Vickeroth, & D. M. Wood (Eds.), *Agriculture Canada Monograph 27: Vol. 1. Manual of nearctic Diptera* (pp. 293–300). Quebec: Canadian Government Publishing Center.

Rutledge, L. C., Walton, B. C., & Ellenwood, D. A. (1976). A transect study of sand fly populations in Panama (Diptera: Psychodidae). *Environmental Entomology, 5*, 1149–1154.

Santamaría, E., Munstermann, L. E., & Ferro, C. (2002). Estimating carrying capacity in a newly colonized sand fly *Lutzomyia serrana* (Diptera: Psychodidae). *Journal of Economic Entomology, 95*(1), 149–154.

Tindale, B. V., Tikute, S. S., Arankalle, V. A., Sathe, P. S., Joshi, M. V., Ranadive, S. N., et al. (2008). Chandipura virus: A major cause of acute encephalitis in children in North Telangana, Andhra Pradesh, India. *Journal of Medical Virology, 80*, 118–124.

Triplehorn, C., & Johnson, N. (2005). *Borror and Delong's Introduction to the study of insects* (7th ed.). Belmont, CA: Thompson-Brooks/Cole. ISBN 0-03-096835-6. 864 pp.

VectorBase. Genonic, phenotypic, and population-centric data for invertebrate vectors of human pathogens. National Institute of Allergy and Infectious Diseases (NAIAD). http//www.vectorbase.org.

Walters, L. L. (1993). *Leishmania* differentiation in natural and unnatural sand fly hosts. *The Journal of Eukaryotic Microbiology, 40*, 196–206.

Werneck, G. L. (2014). Visceral leishmaniasis in Brazil: Rationale and concerns related to reservoir control. *Revista de Saúde Pública, 48*(5), 851–856. https://doi.org/10.1590/S0034-8910.2014048005615.

WHO (World Health Organization). (2017). *Leishmaniasis. Epidemiological situation*. http://www.who.int/leishmaniasis/burden/magnitude/burden_magnitude/en/.

Young, D. G., & Duncan, M. A. (1994). *Guide to the identification and geographic distribution of* Lutzomyia *sand flies in Mexico, the West Indies, central and south America (Diptera: Psychodidae)*. Gainesville, Florida, USA: Associated Publishers: American Entomological Institute, 881 pp.

Young, D. G., & Perkins, P. V. (1984). Phlebotomine sand flies of North America (Diptera: Psychodidae). *Mosquito news, 44*, 263–304.

This page intentionally left blank

Chapter 13

Biting Midges (Ceratopogonidae)

Gary R. Mullen[1] and C. Steven Murphree[2]

[1]*Department of Entomology and Plant Pathology, Auburn University, Auburn, AL, United States;* [2]*Biology Department, Belmont University, Nashville, TN, United States*

Biting midges are minute bloodsucking flies represented by only a few of the many genera in the family Ceratopogonidae. They are commonly known as **no-see-ums** owing to their small size and the fact that they often go unnoticed despite their discomforting bites. Another name for this group, especially in the northeastern United States, is ***punkies***. It is derived from a Dutch corruption of the Algonquin (Native American) root *punkwa*, which means ashlike, referring to the appearance of the fly as it is biting. The associated burning sensation is likened to that of a hot ash from a fire in contact with skin. The early French Canadians called them **brulôt** from *bruler*, meaning "to burn." They also are called **sand flies**, particularly in the coastal areas of the southeastern United States, the West Indies, and adjacent parts of the Caribbean and Latin America. This name should not be confused with the same term applied to phlebotomine flies of the family Psychodidae. Along the Gulf Coast of Alabama and Florida, local residents refer to biting midges as **five-o's** because of their biting activity that commences late in the afternoon about 5 o'clock. Other names for biting midges in various parts of the world include **moose flies** in Alaska, *jejenes* in Latin America, *maruins* in Brazil, *kuiki* in India, *makunagi* and *nukaka* in Japan, *nyung noi* in Laos, *agas* and *merutu* in Indonesia, *merotoe* in Sumatra, and *no-no's* in Polynesia.

Biting midges can be annoying pests to humans and both domestic and wild animals. In addition to the discomfort that they cause, biting midges serve as vectors of a number of viruses, protozoans, and nematodes. Among the more important viral diseases are Oropouche fever in humans, bluetongue disease (BT) and epizootic hemorrhagic disease in ruminants, and African horse sickness (AHS) in equids. Blood protozoans transmitted by biting midges cause diseases in poultry, whereas certain nematodes are the cause of Mansonellosis in humans and onchocerciasis in various domestic and wild animals.

TAXONOMY

The Ceratopogonidae are represented worldwide by approximately 123 genera and 6,267 extant described species. Ceratopogonids are divided into four subfamilies, the Leptoconopinae, Forcipomyiinae, Dasyheleinae, and Ceratopogoninae. Catalogs of the species of ceratopogonids worldwide are provided by Borkent and Wirth (1997, 2016, http://wwx.inhs.illinois.edu/files/4514/6410/0252/CeratopogonidaeCatalog.pdf) and Yu and Liu (2006). A catalog of New World species north of Mexico was provided by Borkent and Grogan (2009). For a world list of the species and subspecies within the genus *Culicoides*, see Boorman and Hagan (1996).

With the exception of the Dasyhelinae, each subfamily includes species that feed on vertebrate blood. Species in only four genera are known to attack humans and other animals. The most important genus in this respect is *Culicoides*. It includes most of the troublesome species throughout the world and those that serve as the principal vectors of animal disease agents. *Leptoconops* occurs primarily in the subtropics and tropics; a few species are annoying biters in the Caribbean area and along the coast of the southeastern United States. *Forcipomyia* species in the subgenus *Lasiohelea* attack vertebrates, particularly in subtropical and tropical rain forests. Two extant species of *Austroconops*, a blood-feeding genus known from the Lower Cretaceous period, have been reported in Western Australia (Borkent, 2004b). No members of the Dasyheleinae are of medical or veterinary importance.

Because of its importance in transmitting animal viruses in North America, the *Culicoides variipennis* complex warrants special comment. For many years, *C. variipennis* was thought to consist of five subspecies: *Culicoides variipennis albertensis*, *Culicoides variipennis australis*, *Culicoides variipennis occidentalis*, *Culicoides variipennis sonorensis*, and *Culicoides variipennis*. However, based on

Medical and Veterinary Entomology. https://doi.org/10.1016/B978-0-12-814043-7.00013-3

morphological and electrophoretic analyses, this complex is now regarded as three species (*Culicoides occidentalis*, *Culicoides sonorensis*, and *C. variipennis*), with *C. v. albertensis* and *C. v. australis* being synonyms of *C. sonorensis* (Holbrook et al., 2000). *C. sonorensis* (rather than *C. variipennis*, as widely reported in the earlier literature) is the principal vector of the viruses causing BT and epizootic hemorrhagic disease in North American ruminants.

The *Culicoides obsoletus* complex and *Culicoides pulicaris* complex have become increasingly implicated as vectors of Bluetongue virus (BTV) in the Palearctic region. Species-specific polymerase chain reaction primers are available to identify members of both of these groups (Nolan et al., 2007). The *Culicoides imicola* complex includes 10 morphological species (Sebastiani et al., 2001). This complex is confined to the Old World, where it has an important role in transmitting the viruses that cause African horse sickness and bluetongue disease.

For further discussion of *Culicoides* vector complexes, see Meiswinkel et al. (2004).

Keys to the genera of ceratopogonid adults are provided by Wirth et al. (1974) and Downes and Wirth (1981). For keys to adults of North American *Culicoides* species, see Jamnback (1965), Battle and Turner (1971), and Blanton and Wirth (1979). A generic key to the pupae of the world fauna was published by Borkent (2014). Generic keys for larvae are provided by Glukhova (1977, 1979). For larval keys to North American species of *Culicoides*, see Jamnback (1965), Blanton and Wirth (1979) and Murphree and Mullen (1991). Major taxonomic works for identifying ceratopogonid fauna in other parts of the world include Central America (Spinelli and Borkent, 2004), South America (Spinelli et al., 2005; Borkent and Spinelli, 2007), former Union of Soviet Socialist Republics (Glukhova, 1989), China (Yu, 2005, 2006), Southeast Asia (Wirth and Hubert, 1989), Australia (Bellis et al., 2015) and Africa (Glick, 1990; Rawlings et al., 2003).

MORPHOLOGY

Ceratopogonid larvae (Fig. 13.1B), as represented by *Culicoides* species, are typically long and slender, ranging from 2 to 5 mm in length when mature. The body has a white, translucent appearance in contrast to the yellow-to-brown head capsule (Fig. 13.2). The thorax often is marked by a characteristic pattern of subcutaneous pigmentation. Thoracic and abdominal segments are similar in size, contributing to their elongate, cylindrical body shape. Although larvae of other genera may possess distinctive setae and abdominal projections, the larval chaetotaxy of *Culicoides* and related genera is generally inconspicuous, except for four pairs of setae that may be apparent at the caudal end. These setae are especially long in tree-hole species and are believed to increase larval mobility. A pair of narrow, bifid anal papillae that function in osmoregulation can be everted through the anus; in most preserved specimens, however, they are retracted into the rectum. Larvae generally lack spiracles and depend on cutaneous respiration. Whereas *Culicoides* and *Leptoconops* larvae lack thoracic and abdominal appendages, *Forcipomyia* and *Atrichopogon* larvae possess a well-developed, ventral prothoracic proleg and associated apical hooklets or setae.

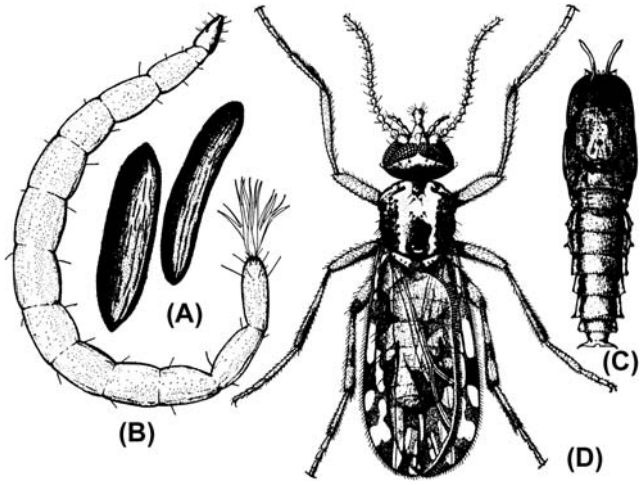


FIGURE 13.1 Developmental stages of the salt-marsh biting midge *Culicoides furens*. (A) eggs; (B) larva; (C) pupa; (D) adult female. *Modified from Hall, 1932.*

The mouthparts are characterized by a pair of mandibles that are not opposable; they move vertically or partially rotate while the larva feeds and are used to scrape, tear, or seize items depending on the species involved (Mullen and Hribar, 1988; Murphree and Mullen, 1991). The shape of the mandibles and the number of teeth, if present, as well as the number of lateral teeth of the ventrally located hypostoma have been used as taxonomic characters. Located within the buccal cavity is a complex, sclerotized internal structure called the

FIGURE 13.2 *Culicoides* larva, fourth instar. Note the slender, cylindrical body and distinct head capsule. *Photograph by Richard C. Lancaster.*

epipharynx, which is best observed in cleared, slide-mounted specimens. It consists of a pair of lateral arms and a median region supporting two to four combs that overlay one another. The epipharynx together with the trough-like hypopharynx comprise the pharyngeal apparatus. The epipharynx is rocked back and forth by muscles attached to the lateral arms and functions to shred solid food and move food items posteriorly into the alimentary tract. In species that feed primarily on detritus and microorganisms, the combs apparently serve to strain material entering the mouth cavity. The number of pharyngeal combs and degree of sclerotization of the epipharynx is highly variable, reflecting the diversity of ingested food items and feeding behaviors exhibited by ceratopogonid larvae.

Pupae are typically brownish with a pair of relatively short but conspicuous prothoracic respiratory horns arising at the anterior end (Fig. 13.1C). Close inspection reveals numerous tiny spiracular openings at the tip. The respiratory tubes repel water, enabling aquatic forms to hang at the water surface, where they can obtain air during metamorphosis to the adult stage. A pocket of air beneath the developing wings provides additional buoyancy to keep the pupa at the water surface. Cuticular features in the form of tubercles, spines, and setae provide valuable taxonomic characters to identify pupae to species. For a comprehensive account of pupal characters, standardized terminology, and relationships among species, see Borkent (2014).

Adult *Culicoides* midges (Fig. 13.1D) are tiny, usually 1.0−2.5 mm in body length. Their mouthparts are adapted for biting or piercing tissues and are especially well-developed in bloodsucking species (Fig. 13.3). In females, the mouthparts are surrounded by a prominent extension of the labium called a proboscis, which is relatively short, about as long as the head. It consists of an upper labrum-epipharynx, a pair of bladelike mandibles, a pair of laciniae (maxillae), and a ventral hypopharynx bearing a median, longitudinal groove along which saliva is passed as the female feeds. The mandibles bear a row of teeth along the inner edge near the tip, which is used to lacerate the skin while biting. The mouthparts of males are generally reduced and are not used in blood feeding.

Associated with the mouthparts are a pair of five-segmented maxillary palps. The third segment is typically enlarged and bears a specialized group of sensilla located in a depression, or sensory pit, which serves as a sensory organ. The adult antennae are 15-segmented; each consists of a basal scape, an enlarged pedicel containing a Johnston's organ, and 13 flagellomeres. The antennal segments bear differing numbers of small sensory pits (sensilla coeloconica), the number and pattern of which provide important taxonomic characters. The number of segments bearing sensory pits appears to be correlated

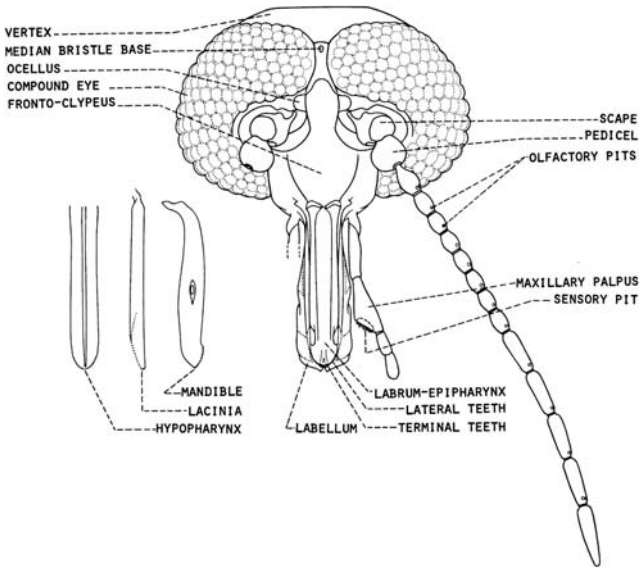


FIGURE 13.3 Morphology of head, mouthparts, and other associated structures of a female biting midge, *Culicoides* species. *From Blanton and Wirth, 1979.*

with host feeding; species that feed primarily on birds generally have more sensory pits than those that feed on mammals. In males, flagellomeres 1−8 possess whorls of long setae that increase their sensitivity as mechanoreceptors and give them their plumose appearance. The wings have one or two radial-vein branches reaching the wing margin and two median-vein branches. The distinctive wing patterns of most members of the genus *Culicoides* provide valuable taxonomic characters for identifying species in this large, important group (Fig. 13.4). The darker areas of the wings are not pigmented but indicate the density of tiny setae (micro- and macrotrichia) on the wing surface.

LIFE HISTORY

Adult females typically require a blood meal to develop their eggs (Fig. 13.1A). However, some are autogenous and carry over enough nutrients from the larval stage to develop eggs during the first gonotrophic cycle without feeding on blood. Development of the eggs usually requires 7−10 days but may be as short as 2−3 days. The eggs are deposited in batches on moist substrates. The number of eggs per female varies from 30 to 450 or more, depending on the species and size of the blood meal. Autogenous females tend to produce fewer eggs. The eggs are small and elongate (250−500 μm in length) and often banana-shaped and are covered with minute projections that apparently function in plastron respiration. They are white when first deposited but gradually turn brown. The eggs hatch in 2−7 days. The larvae develop through four instars with a development time that varies from 2 weeks to more than a

FIGURE 13.4 Biting midge (*Culicoides glabrior*), female; showing distinctive wing pattern characteristic of the species, Brazil. *Photograph by Luis Paulo Carvalho.*

year, reflecting different species, latitudes, and seasons. Many species overwinter as larvae and thus commonly pass 7 or 8 months in this stage. In other cases, larvae become dormant during the hot summer months, prolonging their developmental time. Larval development of some arctic species may take as long as 2 years. Pupation generally occurs near the surface of the substrate, where the prothoracic horns of the pupae can penetrate the water film. Pupae of *Culicoides* species that develop in water-filled tree holes may remain afloat at the water surface, loosely adhering to the sides of the tree cavity.

Overwintering larvae pupate in the spring or early summer, producing the first generation of adults. Autogenous females usually oviposit about a week after emergence; thereafter they must obtain a blood meal each time before they can develop another batch of eggs. Newly emerged, anautogenous females oviposit after their initial blood meal. The small percentage of the females that are successful in obtaining a second blood meal can produce a second batch of eggs, but they seldom do so a third time under field conditions. In the laboratory, however, *C. variipennis* is capable of completing up to seven gonotrophic cycles. Longevity of captive adults varies from 2 to 7 weeks, with most individuals probably surviving only a few weeks at most under natural conditions. The generation time may be as short as 2 weeks for members of the *C. variipennis* complex, but more typical is 6 weeks or longer for most species.

Although some species are univoltine, most biting midges are multivoltine, producing two or more generations per year. Because of overlapping generations and multiple oviposition cycles by individual females, populations of a given species may be present throughout the warm months of the year. Usually, however, each species exhibits a general seasonal pattern with characteristic peaks of adult abundance. Some species are abundant only in the spring (e.g., *Culicoides biguttatus*, *C. niger*, *C. travisi*), whereas others may exhibit high spring populations and be present in lower numbers throughout the summer and fall (e.g., *C. spinosus*). Others tend to be abundant throughout the spring, summer, and fall (e.g., *C. crepuscularis*, *C. furens*, *C. haematopotus*, *C. stellifer*, and *C. venustus*). Still others are bivoltine, with peaks in the spring and fall (e.g., *C. hollensis*).

BEHAVIOR AND ECOLOGY

Ceratopogonid larvae develop in a wide range of aquatic and semiaquatic habitats in biomes ranging from the tropics to the arctic tundra. *Leptoconops* species occur primarily in sandy or claylike, alkaline soils of arid regions and along tidal margins or coastal marshes and beaches. *Forcipomyia* species are generally found in mosses and algae in shallow water and in more terrestrial habitats, such as rotting wood. The larval habitat of *Austroconops* is wet soil and detritus. It is difficult to make generalizations about the breeding sites of *Culicoides* species except to say that they occur primarily in organically rich substrates (Fig. 13.5). As a group, they use a broad diversity of habitats including freshwater marshes and swamps; shallow margins of ponds, streams, and rivers; bogs and peat lands; tree holes and other natural cavities in rotting wood; tidal marshes and mangroves; and more specialized habitats such as rotting cacti, animal manure (Fig. 13.6), and highly alkaline or saline inland pools.

The diversity of ceratopogonid larvae is similarly reflected in their feeding behavior. Many are predaceous, feeding on protozoans, rotifers, oligochaetes, nematodes,

FIGURE 13.5 Sampling organically enriched substrate of freshwater marsh for ceratopogonid larvae. *Photograph by Gary R. Mullen.*

FIGURE 13.6 Typical breeding site of *Culicoides sonorensis* in wet, manure-contaminated soil surrounding leaking water trough. *Photograph by Gary R. Mullen.*

immature stages of insects, and various other small aquatic or semiaquatic invertebrates. Others feed on detritus, bacteria, fungi, green algae, diatoms, and other organic materials. Based on feeding experiments and direct observations, it is apparent that many species are omnivorous and feed opportunistically on a variety of food items. Members of the *C. variipennis* complex, for example, are generally reared on a diet of microorganisms; however, it can also complete its development when fed only nematodes. For most species, the natural diet and nutritional requirements remain unknown, precluding the establishment of laboratory colonies for most of the economically important species. Among the North American species of *Culicoides* that have been laboratory cultured are *C. furens*, *C. guttipennis*, *C. melleus*, *C. mississippiensis*, *C. sonorensis*, and *C. wisconsinensis*. In addition are *C. nubeculosus* (England) and *C. oxystoma* (Japan).

Despite their small size, ceratopogonid larvae often can be recognized by their serpentine locomotion consisting of side-to-side lashing movements of the body as they propel themselves through water or moist organic substrates. *Culicoides* larvae are generally considered to be good swimmers, especially the later instars. *Culicoides circumscriptus* can lash back and forth an estimated 9 cycles/s, whereas members of the *C. variipennis* complex are capable of sustained, directed swimming at speeds up to 1.7 cm/s. Species such as *C. denningi* that burrow in the bottom of streams and rivers are excellent swimmers, enabling them to make their way to shore where they pupate.

Males typically emerge a short time before females and are ready to mate by the time the females are produced. Mature sperm are already present within 24 h of eclosion. Unlike mosquitoes, ceratopogonid males do not undergo permanent rotation of the genitalia. Instead, the genital structures are temporarily rotated 180 degrees to facilitate clasping the female in an end-to-end position just before mating takes place.

Mating usually involves swarming, in which large numbers of males form aerial aggregations, often near water or in open areas near potential breeding sites. Females fly into the swarm where males recognize them as being the same species by their wing-beat frequency. Sex pheromones have been shown to be involved in some species. If a female is receptive, she couples with the male and typically drops to the ground or vegetation where copulation takes place. A few species mate without forming swarms. In such cases, the male and female locate one another by crawling about on the ground or some other substrate where coupling occurs. In other cases, both sexes are attracted to a host, where the male seeks out and mates with the female shortly after she has taken a blood meal. Most species are believed to mate only once, although members of the *C. variipennis* complex and others may mate repeatedly. Sufficient sperm from a single mating is stored by females of the *C. variipennis* complex to fertilize up to three batches of eggs.

As adults, both males and females feed on nectar of flowering plants. This serves as an energy source for flight activity and increased longevity, especially in females. Only females feed on vertebrate blood (Fig. 13.7). As in other hematophagous insects, usually blood is required for egg development and subsequent oviposition. Females are pool feeders (telmophages). They lacerate the skin and underlying capillaries with the serrated tips of their mandibles, causing blood to seep into the surrounding tissues. From there, it is drawn into the foregut by action of the pharyngeal pump and passed back into the midgut. After feeding, the blood-laden female flies to nearby vegetation or another sheltered site, where she rests for several days while her eggs develop.

Many species of biting midges feed primarily on mammals, whereas others feed preferentially on birds, reptiles, or amphibians. Those that feed on a given class of hosts often show preferences for certain groups within that

FIGURE 13.7 *Culicoides sonorensis*, adult female feeding on human arm. *Photograph by P. Kirk Visscher.*

class, such as small versus large mammals or certain types of birds. Whereas some are host-specific, others are considered generalists and may feed, for example, on both birds and mammals, depending on host availability.

In general, *Culicoides* adults are crepuscular and/or nighttime feeders, whereas *Leptoconops* adults tend to be more active during the daytime. The activity periods of most biting midges occur during twilight, particularly an hour before to an hour after sunrise and sunset. Whereas some species exhibit bimodal activity at dawn and dusk (e.g., *Culicoides barbosai*, *C. furens*, and *C. stellifer*) or during morning and late afternoon (e.g., *Leptoconops becquaerti*), others tend to be active during only one of these periods (e.g., *C. debilipalpis* in the early dawn hours; *C. furens*, *C. paraensis*, and *Leptoconops linleyi* in the late afternoon.). Certain species will readily bite during the daytime and can be particularly annoying in the late afternoon. Activity periods for a given species may vary at different times of the year, reflecting seasonal changes in temperature and light intensity. The effects of daily and seasonal temperatures on flight activity are evident among salt-marsh species along the Florida coast. Whereas *C. barbosai*, *C. floridensis*, and *L. becquaerti* are not active below 14°C (57°F), *C. mississippiensis* remains active all year, even at winter temperatures as low as 4°C (37°F).

In addition to temperature, a number of other factors can influence flight activity by biting midges (Lillie et al., 1987; Kettle, 1972). They include light intensity, lunar cycles, relative humidity, changes in barometric pressure, and other weather conditions. Wind velocity is especially important. Because of their small size, most species do not tolerate appreciable air movement and are seldom troublesome at wind speeds above 2.5 m/s. Such velocities interfere with normal flight activity and the ability to orient to a host. There are exceptions, however, such as *L. becquaerti*, which will continue to bite at wind speeds of 5.0 m/s or more.

Biting midges are most abundant in proximity to productive breeding sites. From there, they disperse into surrounding areas in search of mates and suitable hosts. How far they travel is highly variable depending on their success in finding a mate, availability of hosts, and prevailing weather conditions. Mark-recapture studies, in which adults are released at a given location and are subsequently recovered by trapping at increasing distances from the release point, indicate that the mean distance traveled by many *Culicoides* females is about 2 km. The distance traveled by males is usually much shorter, often less than half that of females of the same species. The flight distance for any individual, however, can be much lower or higher than this mean value implies. Members of the *C. variipennis* complex have been recovered up to 2.8 km from release sites within 12 h and nearly 5 km within 36 h. Salt-marsh species such as *C. mississippiensis* have been shown to disperse more than 3 km within 24 h, whereas *Leptoconops kerteszi* in semiarid regions of the southwestern United States has been reported to fly 15 km or more in this same period.

PUBLIC HEALTH IMPORTANCE

Most complaints about biting midges relate to the annoyance caused by their persistent biting. This is especially a problem in coastal areas where salt-marsh species are notorious pests, often creating great discomfort for local residents and beachgoers and discouraging tourism during the summer months. In the United States, *C. furens*, *C. hollensis*, and *C. melleus* are the most troublesome species attacking humans along the Atlantic Coast. *Culicoides mississippiensis* is a problem along the Gulf Coast, whereas *C. barbosai* is commonly a problem near the mangrove forests of southern Florida. Certain *Leptoconops* species are especially pestiferous along coastal beaches; e.g., *L. linleyi* along the Gulf Coast and *L. becquaerti* in the Caribbean, whereas members of the *L. kerteszi* group are annoying biters in semiarid regions of the southwestern United States.

A common pest throughout much of the eastern United States is *C. paraensis*, a tree-hole species that readily bites humans, especially during the late afternoon and early evening. It causes considerable discomfort to hunters, campers, and hikers and is typically the species involved in annoyance to homeowners while picnicking or trying to work in their yards bordering deciduous woods.

Because of their small size, biting midges frequently are overlooked; their bites are often blamed on mosquitoes. Reactions to their bites generally consist of a localized stinging or burning sensation, producing a well-defined reddened area about the bite site without the formation of a wheal. The discomfort usually lasts from only a few minutes to a few hours. In individuals who develop hypersensitivity, the bites may continue to itch for 2 or 3 days. In the tropics, certain *Leptoconops* and *Lasiohelea* species can cause more severe reactions resulting in blisters and serous exudates at the bite sites of sensitized individuals.

Viruses and filarial nematodes are the only disease agents known to be transmitted to humans by the bite of ceratopogonid midges (Tables 13.1 and 13.2). They are primarily subtropical or tropical in distribution; no associated diseases have been reported in North America. Although several viruses have been isolated from *Culicoides* adults, Oropouche virus is the only significant viral agent transmitted to humans by ceratopogonid midges. Biting midges also transmit three filarial nematode species that parasitize humans, causing a disease known as ***mansonellosis*** or ***mansonelliasis***. The causative agents are *Mansonella ozzardi* in the Americas, *Mansonella perstans* in Africa and South America, and *Mansonella streptocerca*

TABLE 13.1 Species and Strains of Arboviruses of Medical and Veterinary Interest Transmitted by Biting Midges (*Culicoides* Species)*

Virus	Vertebrate Host	Geographic Area	Known or Suspected Vectors
Peribunyaviridae			
Akabane *orthobunyavirus*			
Akabane	Cattle, sheep, goats, horses, buffalo, camels	Africa, Middle East, Japan, Australia	*Culicoides brevitarsis*
Sabo	Cattle, goats	Nigeria	*C. imicola*
Tinaroo	Cattle, sheep, goats, buffalo	Australia	*C. brevitarsis*
Bunyawera orthobunyavirus			
Lokern	Lagomorphs (*Lepus, Sylvilagus*)	North America	*Culicoides variipennis* complex, *Culicoides* (*Selfia*) spp.
Main drain orthobunyavirus			
Main drain	Lagomorphs (*Lepus*)	North America	*C. variipennis* complex
Manzanilla orthobunyavirus			
Buttonwillow	Lagomorphs (*Lepus, Sylvilagus*)	United States	*C. variipennis* complex
Oropouche orthobunyavirus			
Oropouche	Humans, forest primates, sloths	South America, Caribbean	*C. paraensis*
Utive	Sloths	Panama	*C. diabolicus*
Utinga	Sloths	Panama, Brazil	*C. diabolicus*
Rift Valley Fever phlebovirus			
Rift Valley Fever	Humans, cattle, buffalo, sheep, goats, antelope, camels	Africa	*Culicoides* spp. (primarily mosquitoes)
Sathuperi orthobunyavirus			
Douglas	Cattle, sheep, goats, horses, buffalo deer	Australia, New Guinea	*C. brevitarsis*
Schmallenberg	Cattle, sheep, goats, deer, mouflon, bison	Europe	*C. obsoletus, C. scoticus, C. punctatus*
Sathuperi	Cattle	Nigeria, Kenya, India	*Culicoides* spp.
Shamonda orthobunyavirus			
Peaton	Cattle	Australia	*C. brevitarsis*
Sango	Cattle	Nigeria, Kenya	*Culicoides* spp.
Shamonda	Cattle	Nigeria	*C. imicola*
Shuni orthobunyavirus			
Aino	Cattle, sheep, buffalo	Japan	*C. brevitarsis*
Shuni	Humans, cattle	Nigeria, South Africa	*Culicoides* spp.
Thimiri orthobunyavirus			
Thimiri	Birds	Egypt, India, Australia	*C. histrio*
Crimean-Congo hemorrhagic fever nairovirus			
Crimean-Congo hemorrhagic fever	Humans, cattle	Africa, Asia	*Culicoides* spp. (primarily ticks)
Dugbe nairovirus			
Dugbe	Humans, cattle	Africa	*Culicoides* spp. (primarily ticks)

Continued

TABLE 13.1 Species and Strains of Arboviruses of Medical and Veterinary Interest Transmitted by Biting Midges (*Culicoides* Species)[*]—cont'd

Virus	Vertebrate Host	Geographic Area	Known or Suspected Vectors
Reoviridae			
Sedoreovirinae *(Orbivirus)*			
African horse sickness	Horses, mules	Africa, Middle East, India, Europe, Asia	*Culicoides imicola*
Bluetongue	Cattle, sheep, other domestic and wild ruminants	Africa, Middle East, Europe, Japan, Australia, North America, South America	*C. fulvus, C. gulbenkiani, C. imicola, C. insignis, C. milnei, C. obsoletus, C. sonorensis*
Epizootic hemorrhagic disease	Deer, cattle	North America, Africa, Asia, Australia	*C. sonorensis, C. schultzei*
Equine encephalosis	Cattle	Africa, Australia	*C. imicola, C. bolitinos*
Nairoviridae			
Eubenangee Group			
Eubenangee	Humans, cattle, kangaroos, wallabies	Australia	*C. marksi* (also mosquitoes)
Palyam Group			
Abadina	Cattle, sheep	Nigeria	*Culicoides* spp.
Bunyip Creek	Cattle, buffalo, sheep, deer	Australia	*C. brevitarsis, C. oxystoma*
CSIRO Village	Cattle, buffalo	Australia	*C. brevitarsis*
D'Aguilar	Cattle, sheep	Australia	*C. brevitarsis*
Kasba (= Chuzan Kagoshima)	Cattle	Japan	*C. oxystoma*
Marrakai	Buffalo?	Australia	*C. oxystoma, C. peregrinus*
Nyabira	Cattle	Zimbabwe	*Culicoides* sp.?
Wallal Group			
Mudjinbarry	Marsupials	Australia	*C. marksi*
Wallal	Marsupials	Australia	*C. marksi*
Wallal K	Marsupials	Australia	*Culicoides* spp.
Warrego Group			
Mitchell River	Cattle, marsupials	Australia	*Culicoides* spp.
Warrego	Cattle, marsupials	Australia	*C. dycei, C. marksi* (also mosquitoes)
Wongorr Group			
Wongor	Humans, cattle, wallabies	Australia	*C. pallidothorax*
Rhabdoviridae			
Ephemerovirus			
Bovine ephemeral fever	Humans, cattle, horses, sheep, kangaroos, wallabies, rats	Africa, Asia, Australia	*C. kingi, C. nivosus, C. bedfordi, C. pallidipennis, C. cornutus*
Kotonkan	Humans, cattle, horses, sheep, rats, hedgehogs, bats	Africa	*Culicoides* spp.
Tibrovirus			
Tibrogargan	Cattle, water buffalo	Australia	*C. brevitarsis*

[*]*Classification of the viruses in this table are based on the International Committee on Taxonomy of Viruses (ICTV), Master Species List, v1.0. (2017). https://talk.ictvonline.org/files/master-species-lists/m/msl/7185*

TABLE 13.2 Filarial Nematodes Transmitted by Biting Midges to Humans and Domestic Animals (Vectors Include *Culicoides*, *Forcipomyia*, and *Leptoconops* Species)

Nematode	Vertebrate Host	Geographic Area	Known or Suspected Vectors
Mansonella ozzardi	Human	South America, Caribbean Basin	*Culicoides barbosai, C. furens, C. paraensis, C. phlebotomus, Leptoconops becquaerti*
M. perstans	Human	Sub-Saharan West Africa; Central Africa to Kenya and Mozambique Northern coast of South America; Caribbean Islands	*C. austeni, C. grahamii, C. inornatipennis* *Culicoides* spp.
M. streptocerca	Human	West and Central Africa (rain forests)	*C. austeni, C. grahamii*
Onchocerca cervicalis	Horses	North America, Australia	*C. variipennis, C. victoriae, Forcipomyia townsvillensis*
O. gibsoni	Cattle	India, Sri Lanka, Malaysia, northern Australia, South Africa	*C. pungens, C.* spp.
O. gutturosa	Cattle	Australia	*Culicoides* spp.
O. reticulata	Horses, ponies	Australia	*C. nubeculosus, C. obsoletus*
O. sweetae	Water buffalo	Unknown	Unknown

in Africa. Linley (1983) has provided an excellent overview of the various human pathogens and parasites transmitted by this group of flies.

Oropouche Fever

Oropouche fever is caused by a virus in the family Peribunyaviridae. Since it was first isolated from a charcoal worker in Trinidad in 1955, this virus has been documented in numerous epidemics in the Amazon region of Brazil. Outbreaks before 1980 were largely restricted to Pará State, where over 165,000 human cases of this disease were estimated to have occurred between 1961 and 1980. Since that time, outbreaks have been reported in the Brazilian states of Amazonas, Goias, and Marahnao, and in the Amapa Territory. These epidemics have taken place primarily in urban areas, where surveys indicate that up to 44% of local populations have been seropositive for antibodies to the virus.

Oropouche virus causes a nonfatal, acute febrile illness with general muscular and joint pains usually lasting 2–5 days. More than half of cases involve symptoms such as headaches, dizziness, photophobia, and severe myalgia and arthralgia, which can occasionally lead to prostration. Recurrence of symptoms often prolongs the illness up to 2 weeks. The incubation period for this disease is believed to be 4–8 days.

The epidemiology of Oropouche fever is complicated by multiple strains of the virus and uncertainty about which animals serve as reservoirs. Antibody levels in potential reservoir hosts tend to be highly variable, with some evidence indicating that urban and sylvatic cycles are involved. During nonepidemic periods, several species of monkeys have been found to have high antibody levels implicating them as important reservoirs. Other likely reservoirs in the sylvatic cycle are wild birds and sloths. During epidemics, high antibody levels among various carnivores and domestic birds suggest that they have a role as reservoirs in urban outbreaks of this disease.

The principal vector in urban outbreaks is *C. paraensis*. This forest species breeds in tree holes and decaying cacao and calabash pods. It readily feeds on humans both inside and outside houses. Infected *C. paraensis* females can transmit the virus as early as 4–6 days after a blood meal. Although Oropouche virus has also been isolated from naturally infected mosquitoes such as *Culex quinquefasciatus*, *Aedes serratus*, and *Coquillettidia venezuelensis*, the role of these species as vectors remains uncertain.

Other Viral Agents

Many other arboviruses have been isolated from *Culicoides* adults. Four of them are members of the Bunyaviridae: Crimean-Congo, Rift Valley fever, Dugbe and Shuni viruses. In addition, three mosquito-borne viruses that cause eastern equine encephalitis, Japanese B encephalitis, and Venezuelan equine encephalitis have been isolated from *Culicoides* and *Lasiohelea* species. There is no evidence,

however, to indicate that biting midges have a significant role in transmitting any of these viruses. For a list of other viruses and organisms transmitted by ceratopogonids, see Borkent (2004).

Mansonellosis

Three filarial nematode species in the genus *Mansonella* cause infestations in humans called mansonellosis (Table 13.2). Cases occur widely throughout the tropical and subtropical regions of both the Old World and New World, where *Culicoides*, *Forcipomyia*, and *Leptoconops* species serve as arthropod vectors. Although infestations involving these parasites are generally mild or asymptomatic, they sometimes cause serious medical problems.

Mansonella ozzardi

Mansonella ozzardi is the only native New World ceratopogonid-borne nematode of humans (Fig. 13.8). It is indigenous to the Americas, occurring in the Amazon Basin (Brazil), along the northern coast of South America (Colombia, Venezuela, Guyana, Surinam, and French Guiana), on Trinidad, Haiti, and other islands of the West Indies, Panama, and in parts of Peru, Bolivia and Argentina. It particularly affects coastal fishing communities near breeding sites of associated vectors. The infection rate among local inhabitants is highly variable, ranging from as low as 5% or less in northern Brazil and some of the Caribbean islands to over 95% among Amerindians in Colombia and Venezuela. Infection rates are generally highest among men and women in older age groups, reflecting chronic exposure to infection in endemic areas.

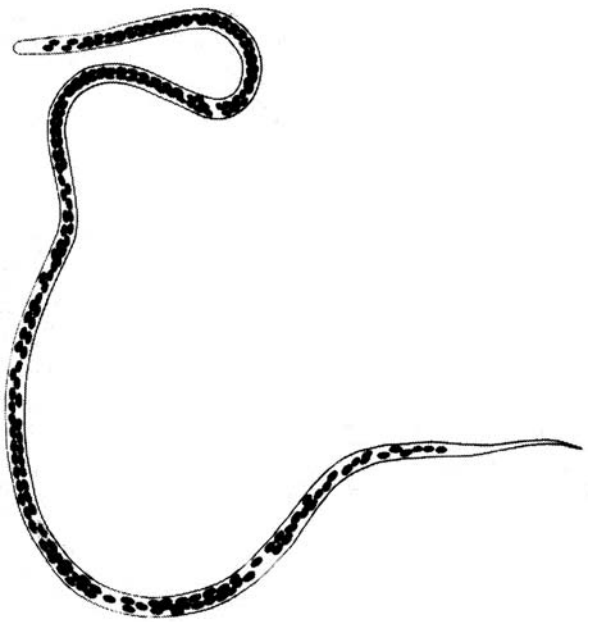


FIGURE 13.8 Filarial nematode (*Mansonella ozzardi*), microfilarial stage. *Courtesy of Lea and Febiger.*

Infections with *M. ozzardi* usually do not result in significant pathological effects. The microfilariae typically remain in the capillaries of the skin and surrounding dermal tissues, where they cause relatively little harm. Surveys are usually conducted by taking skin biopsies or blood samples and examining them for the presence of microfilariae. The adult worms are found primarily in fat tissue associated with the peritoneum and various body cavities, occasionally causing conjunctivitis and swelling of the eyes. In some cases, this nematode can cause more serious problems such as severe joint pains, eosinophilia, enlargement of the liver, and blockage or inflammation of the lymphatic vessels, resulting in conditions similar to Bancroftian filariasis and elephantiasis. Ivermectin has been successfully used to treat patients with *M. ozzardi*, whereas the widely used filarial nematocide diethylcarbamazine is ineffective in killing this parasite.

Vectors of *M. ozzardi* include both biting midges and blackflies; different taxa have important roles in different areas. *Culicoides furens* and *C. phlebotomus* are the principal vectors in Haiti and Trinidad, respectively. In Argentina, *Culicoides lahillei* is believed to be the primary vector; *C. paraensis* and the blackfly *Simulium exiguum* have a secondary role in transmission. Other species that support the development of microfilariae to infective larvae and are generally considered to have a secondary role in transmission are *C. barbosai*, *C. paraensis*, and *L. becquaerti*. After ingestion by a biting midge as it feeds on an infected host, microfilariae are carried into the midgut, where they penetrate the midgut wall and make their way to the thoracic muscles within 24 h. There, they develop to third-stage larvae during the next 6–9 days before moving to the head and mouthparts. Infective third-stage larvae enter the bite wound when the midge subsequently feeds on another host. Typically, only one to three larvae successfully complete development to the infective stage in a host insect, regardless of the number of microfilariae initially ingested.

The role of blackflies as vectors of *M. ozzardi* remains unclear. Species in the *Simulium amazonicum* group and the *Simulium sanguineum* group have been found to be naturally infected with this nematode and probably have a role in transmission, particularly in the Amazon Basin. Other species incriminated as potential vectors based on field collections and experimental infection studies include *Simulium sanchezi* and *S. pintoi*. Despite earlier suggestions that there may be two different forms or species of nematode involved, one transmitted by biting midges and the other by blackflies, evidence indicates that they are morphologically identical and represent a single species.

Mansonella perstans

This nematode (formerly placed in the genera *Acanthocheilonema*, *Dipetalonema*, and *Tetrapetalonema*) is the most widely distributed of the three human filarial nematodes transmitted by biting midges. It is indigenous to the Old World, where it occurs in sub-Saharan Africa, extending primarily from West African countries bordering the Gulf of Guinea (Ivory Coast, Nigeria, and Equatorial Guinea) and from Gabon and Angola east through Central Africa to Kenya and Mozambique. Infection rates are commonly 50% or higher in some communities. *Mansonella perstans* was introduced to Central and South America with the slave trade and now occurs along the northern coast of South America (Colombia, Venezuela, Guyana, Suriname, and French Guiana), in the Yucatán area of Mexico and on Trinidad and other Caribbean Islands. Prevalence of infection exceeding 50% has been reported among the Curripaco (Native Americans) of Venezuela. Although there is evidence to suggest that *M. perstans* represents a complex of species, this issue remains unresolved.

Mansonella perstans is typically regarded as nonpathogenic. The microfilariae remain primarily in the circulating blood, whereas the adult worms occur freely in the body cavities. Some infested individuals develop problems such as joint pains, fever, fatigue, transient edema, elephantoid scrota, mild urticarial skin reactions, and eosinophilia. Various ocular problems including swelling of the eyelids, excessive lacrimation, pruritus, and conjunctival granulomas or nodules have been reported. The latter is the result of adult worms coiled within the connective tissue of the conjunctiva, causing a condition known as **bulge-eye** or **bung-eye**. Adult worms also have been removed from connective tissue of the pancreas, kidneys, rectum, and mesenteric lymph nodes of infested patients with little evidence of serious harm. Mebendazole has been used successfully to treat patients with *M. perstans*, whereas diethylcarbamazine and ivermectin are ineffective in killing either the microfilariae or adult worms.

The principal vectors of *M. perstans* remain virtually unknown in the New World. In Africa, however, several *Culicoides* species have been implicated as vectors based on natural infections and support of development to the infective stage in experimental studies. They include: *C. austeni*, *C. grahamii*, and *C. inornatipennis* as probable vectors, and *C. fulvithorax*, *C. hortensis*, *C. krameri*, *C. kumbaensis*, *C. milnei*, *C. pycnostictus*, *C. ravus*, *C. rutshuruensis*, and *C. vitshumbiensis* as possible vectors. As in the case of *M. ozzardi*, the microfilariae of *M. perstans* move from the midgut of the biting midge to the thoracic musculature, where they complete their development to infective third-stage larvae 8–10 days after the infective blood meal.

Mansonella streptocerca

This filarial nematode (formerly placed in the genera *Dipetalonema* and *Tetrapetalonema*) occurs only in the rain forests of West and Central Africa, extending from the Ivory Coast and Burkina Faso to the Congo and Zaire. Little information on prevalence is available for this species, although a figure of 13%–14% has been reported in certain villages of the Central African Republic based on peripheral blood smears. Although it is regarded as nonpathogenic to humans, *M. streptocerca* occasionally causes mild skin reactions owing to the activity of microfilariae in dermal tissues, usually involving the trunk and upper arms. Adult worms typically occur subcutaneously in upper parts of the body. Diethylcarbamazine is effective as a treatment. *Culicoides grahamii* is regarded as the principal vector.

VETERINARY IMPORTANCE

Biting midges serve as vectors of more than 35 arboviruses that infect domestic animals (Table 13.1). Only a few of these viruses cause significant clinical disease. Cattle, sheep, and horses usually are the most seriously affected. Most of these viral agents are members of the Reoviridae and Bunyaviridae, including the pathogens that cause bluetongue disease epizootic hemorrhagic disease, and African horse sickness. Two other families of viruses for which *Culicoides* species have been implicated as vectors are the Rhabdoviridae and Poxviridae. For most of these viral agents, the principal ceratopogonid species involved as vectors remain largely unknown. Other disease agents transmitted by biting midges include blood protozoans of birds such as *Haemoproteus meleagridis* in turkeys, *Leucocytozoon caulleryi* in chickens, and the nematode *Onchocerca cervicalis* in horses. Biting midges also can cause discomforting skin reactions in horses, known as equine allergic dermatitis.

Bluetongue Disease

Bluetongue disease is caused by Bluetongue virus (BTV) in the genus *Orbivirus* (family Reoviridae, subfamily Sedoreovirinae), which infects ruminants, notably sheep and cattle. It was first recognized in South Africa in the early 1930s after the introduction of European breeds. Historically, it has been limited between latitudes 40 degrees north and 35 degrees south, including North America, parts of Central America and South America bordering the Caribbean, southern Europe and countries bordering the Mediterranean Sea, the Middle East, Asia, Australia, and southern Africa. However, it has made excursions from northern Africa and the eastern Mediterranean into northern Europe, where it had not been documented previously.

Outbreaks occurred among cattle and sheep in the Netherlands, Belgium, France, and Germany in 2006, and in Denmark, Luxembourg, Switzerland, and the United Kingdom in 2007. This northern expansion of bluetongue serotype 8, with successful overwintering of the virus in northern Europe, has been attributed in part to wind-borne BTV-infected *Culicoides* females from endemic areas in the Mediterranean and to global climate changes. BTV-8 was not reported again in Europe until 2015 in central France.

In the United States, Bluetongue virus was first isolated in 1952 in Texas from sheep exhibiting a condition known as **soremuzzle**. It is now known to occur throughout most of the southern and western states, where the prevalence of antibody to BTV in cattle is commonly 20%−50%. No cases have been reported in Alaska or Hawaii. Canada remains largely bluetongue-free; detection of infected animals was reported there only in the Okanagan Valley of British Columbia during localized outbreaks in 1975 and 1987, apparently originating from cattle imported from the United States.

Historically, the global distribution of bluetongue has been relatively well-defined, with many parts of the world remaining largely bluetongue-free. This has led to restrictions on international trade and the movement of cattle and sheep from endemic areas to bluetongue-free zones. The regions where bluetongue occurs can be divided into fairly discrete epidemiological systems, or **episystems**, reflecting the geographic occurrence of major vector species. These include, for example, North America (*C. sonorensis*), South America (*C. insignis*), Africa (*C. imicola*), and Australia (*C. brevitarsis*).

BTV represents an **antigenic complex** with at least 27 currently recognized serotypes that vary significantly in their pathogenicity. The BTV particle is a nonenveloped, double-capsid structure consisting of seven structural proteins (VP1 to VP7) and a genome of 10 double-stranded RNA segments (Celma et al., 2017).

Occurrence of clinical bluetongue disease in cattle tends to be sporadic, often involving only one or a few animals in a given herd. Occasionally, however, epizootics occur. After outbreaks in Cyprus in 1943, Turkey in 1944, and Israel in 1951, major epizootics occurred in Europe in the late 1950s, when an estimated 179,000 sheep died in Spain and Portugal. The mortality rate among infected animals was 75%. BTV has since been introduced to the Caribbean islands, bordering countries of South and Central America, and Australia, where it was first detected in 1974.

Serosurveys for detecting antibodies to BTV indicate that most sheep and cattle that are exposed to the virus do not develop clinical signs. As a result, animals typically remain asymptomatic and often serve as unrecognized sources of infection for other animals. However, under circumstances that are poorly understood, some animals develop varying degrees of illness ranging from mild infections to acute, fatal disease. In severe cases, animals develop lesions about the mouth and muzzle (Fig. 13.11) with ulceration and sloughing of skin tissues, inflammation of the coronary band at the base of the hooves, lesions between the toes (Fig. 13.12), respiratory difficulties caused by the accumulation of fluids in the lungs, and internal hemorrhaging. The term "bluetongue" gets its name from the dusky blue appearance of the tongue and mucosal membranes lining the mouth resulting from cyanosis. Acutely infected animals often exhibit lameness and a characteristic arched back resulting from efforts to keep weight off their painful hooves (Figs. 13.9 and 13.10). Death results primarily from congestion of the lungs and massive internal hemorrhaging.

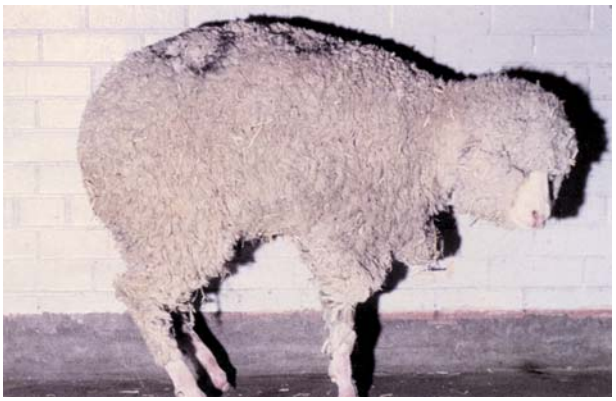


FIGURE 13.9 Sheep with bluetongue disease; infected host with characteristic arched back, tender hooves, and hanging head. *Courtesy of U.S. Department of Agriculture, Animal and Plant Health Inspection Service.*

Reproduction is also affected and can result in underweight calves at birth, congenital deformities, stillbirths, and abortions. The severity of reproductive impact is due in large part to the time during gestation when the infection occurs. Infections in the early stages of fetal development

FIGURE 13.10 Black Angus calf in late stage of bluetongue disease, with general depression, hanging head, labored breathing, and difficulty standing. *Photograph by Lloyd L. Lauerman.*

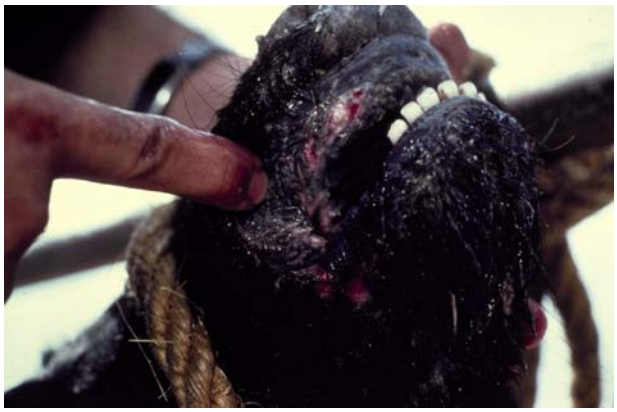


FIGURE 13.11 Oral and muzzle lesions in Black Angus calf with bluetongue disease. *Photograph by Lloyd L. Lauerman.*

can result in aborted or stillborn calves. Infections that occur later in gestation are more likely to result in congenital deformities and underweight calves at birth. The virus is also found in semen of infected bulls. Consequently, restrictions are placed on the exportation of semen and live animals from endemic bluetongue areas for artificial insemination and other breeding purposes. The resulting economic impact on the livestock industry in the United States for mandatory testing of animals and losses in the foreign market are substantial, totaling millions of dollars annually.

The common occurrence of multiple serotypes of BTV in the same geographic area has hampered the use of immunization to protect livestock from infection. Polyvalent vaccines have been developed but generally have not been effective. Natural immunity is acquired by sheep after their recovery from this disease, but it is limited to the particular serotype with which the animal was infected. Cattle apparently do not develop significant immunity after infection.

The primary mode of transmission of BTV is by the bite of infected *Culicoides* midges. Based on experimental studies with *C. sonorensis*, the virus acquired while feeding on a viremic animal invades the salivary glands of the midge and multiplies there. The intrinsic incubation period is temperature-dependent (10—20 days), after which the midge can transmit the virus at subsequent feedings. After infection, the *Culicoides* host remains infective throughout its life. Infection rates are highly variable, depending on the *Culicoides* species and the geographic populations involved. Selective breeding in laboratory colonies has produced both susceptible and highly resistant lines, indicating the complexity of factors influencing the vector competence of *Culicoides* species involved in the epizootiology of this disease.

Transmission of BTV also occurs venereally via semen of infected rams and bulls. When introduced into the female genital tract, the virus potentially can infect the adult animal and, if she is pregnant, the developing embryo or fetus. No methods have been developed to destroy the virus in semen of infected animals. The virus can survive indefinitely in frozen semen samples.

Biting midges of the genus *Culicoides* are the only vectors of BTV. The following are known or suspected vectors. In North America, the primary vector is *C. sonorensis*, with *C. insignis*, *C. debilipalpis*, *C. obsoletus*, and *C. stellifer* being possible secondary species. In the Caribbean Basin, the primary vector is *C. insignis*, although *C. filarifer* and *C. pusillus* also may be involved. In Europe, the Mediterranean region and Middle East the primary vector is *C. imicola* with *C. obsoletus*, *C. pulicaris*, and *C. scoticus* as probable secondary vectors. In Africa, the primary vectors are *C. imicola* and *C. bolitinos*, in addition to *C. gulbenkiani*, *C. magnus*, *C. pycnostictus*, *C. zuluensis*, and members of the *C. shultzei* group. In Asia and the Indian subcontinent, vectors include *C. actoni*, *C. brevitarsis*, *C. fulvus*, and *C. wadi*. In Australia the primary vector is *C. brevitarsis, with C. actoni*, *C. fulvus*, and *C. wadai* considered to be likely vectors and *C. brevipalpus*, *C. oxystoma*, and *C. peregrinus* probably having minor roles in transmission.

FIGURE 13.12 Hoof lesions in Black Angus calf in late stage of bluetongue disease. *Photograph by Lloyd L. Lauerman.*

Epizootic Hemorrhagic Disease

Epizootic hemorrhagic disease (EHD) is similar to bluetongue disease in many respects; the major difference is that it occurs primarily in wild ruminants, notably deer. It also is caused by a species in the genus *Orbivirus* (Reoviridae) and is closely related to BTV. Three strains, or serotypes, of the virus are recognized in North America, designated EHD-1, EHD-2, and EHD-6. EHD-1, known as the New Jersey strain, was first isolated from white-tailed deer in New Jersey during an outbreak in 1955. EHD-2, commonly referred to as the Alberta strain, was first isolated during an epizootic in that Canadian province in 1962. At least five other EHD serotypes have been

isolated in South America, the Caribbean, Africa, the Middle East, Australia, and Asia.

The clinical signs in EHD cases are virtually indistinguishable from bluetongue disease. Isolation and identification of the etiologic agent are usually required to determine with certainty which virus is involved. Because of the similarities of these two diseases in wild ruminants, cases are often referred to simply as **hemorrhagic disease**. It also is referred to as **black tongue disease** by deer hunters in the southeastern United States.

Clinical disease in white-tailed deer and other ruminants varies from sudden death without apparent signs of illness to mild infections from which animals fully recover. Typically the disease is characterized by rapid onset of fever, loss of appetite, disorientation and weakness, a hanging head, labored breathing with the tongue often protruding (Fig. 13.13), swelling of the head and neck, arched back, and painful hooves (Fig. 13.14). As the virus multiplies in endothelial cells lining the blood vessels, it spreads to various organ systems causing extensive internal hemorrhaging (Fig. 13.15), intravascular coagulation, and thrombosis. In acute cases, death usually occurs in 4–10 days after the initial infection. In animals that survive, recovery can be prolonged and debilitating, resulting in permanent lameness owing to deformed hooves (Fig. 13.16) and difficulty eating because of damage to the oral tissues.

EHD is the most important infectious disease of wild deer in the United States. It primarily affects white-tailed deer, causing sporadic die-offs. Mule deer, pronghorns, and domestic cattle also can develop fatal infections, but less commonly. Other wild ruminants that have been found to be infected during EHD epizootics include elk, bison, bighorn sheep, Rocky Mountain goat, and several species of exotic animals such as yak and ibex. Wapiti and moose do not appear to be adversely affected by this virus.

FIGURE 13.14 White-tailed deer buck in late stage of epizootic hemorrhagic disease, with characteristic tender hooves, difficulty walking, arched back, laid-back ears, and general depression. *Photograph by Gary R. Mullen.*

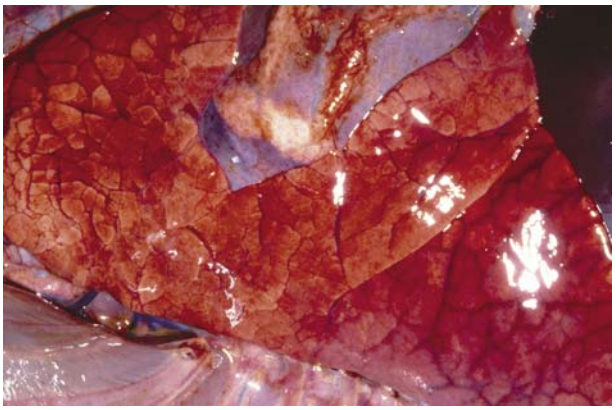


FIGURE 13.15 Extensive hemorrhaging and edema of lung tissue in white-tailed deer that died of epizootic hemorrhagic disease. *Courtesy of C. S. Roberts State Veterinary Diagnostic Laboratory, Auburn, AL.*

FIGURE 13.13 White-tailed deer fawn infected with epizootic hemorrhagic disease virus. Note hanging head and protruding tongue. *Photograph by Gary R. Mullen.*

FIGURE 13.16 Foot lesions, swelling, and deformed hooves in white-tailed deer with epizootic hemorrhagic disease. *Courtesy of Southeastern Cooperative Wildlife Disease Study, Athens, GA.*

Although this disease is endemic throughout the United States where white-tailed deer populations are established, it is more prevalent in the southeast, midwest, and northwest, and along the Pacific Coast. Epizootics, with sudden die-offs in local deer herds, tend to occur in more temperate areas, whereas asymptomatic and subclinical infections are more common in the coastal endemic areas of the southeastern states where infection rates may be as high as 70% or more. Outbreaks of EHD have also been reported in the western provinces of Canada, notably, in southeastern Alberta (1962), the Okanagan Valley of British Columbia (1975), and southern Saskatchewan (1986–1987). In each case, the source of infection has been attributed to *Culicoides* species from adjacent endemic areas in the United States. The only serotype isolated in Canada has been EHD-2.

Cattle are commonly exposed to EHD virus. Based on serologic surveys, infections in cattle are widespread throughout the United States. In most cases, these are silent infections or involve only mild clinical disease. Occasionally, however, epizootics occur in cattle, as in central Oregon in 1969 and eastern Tennessee in 1972. Infections of cattle with EHD virus also have been reported in the South American countries of Guyana, Suriname, and Colombia, and in Taiwan, Malaysia, and Indonesia.

Biting midges of the genus *Culicoides* are the only known **vectors** of EHD virus. The most important species and only proven vector in North America is *C. sonorensis*. Other species have been implicated as potential vectors based on isolations of the virus from field-collected midges and limited experimental studies. The high prevalence of seropositive deer for EHD virus in areas where *C. sonorensis* is uncommon or absent supports the belief that other *Culicoides* species are involved in transmitting this virus in the United States. The vectors in other parts of the world remain unknown.

African Horse Sickness

African horse sickness (AHS) is a viral disease of horses, donkeys, and mules, which can be highly fatal in susceptible animals (Fig. 13.17). It is known by various names including *la pesta equine* (Spain), *pesta ecvina* (Romania), equine plague, and horse sickness fever. The disease was first recognized in South Africa in the early 1700s. The etiologic agent was first isolated from infected horses nearly two centuries later in 1899. It is endemic particularly in central-tropical Africa, regularly spreading to southern Africa and less commonly to northern Africa, also intermittently to the Arabian Peninsula, southwestern Asia, and southern Europe.

The etiologic agent of AHS is a species in the genus *Orbivirus* (Reoviridae), closely related to the viruses that cause bluetongue disease and epizootic hemorrhagic disease. Nine AHS serotypes are recognized, all nine of which occur in eastern and southern Africa. Only serotypes 4 and 9 occur in West Africa, from which occasionally they spread to countries bordering the Mediterranean. The occurrence of multiple serotypes in a given geographic region and simultaneous infections of animals with more than one serotype underscores the epizootic complexity of this disease.

FIGURE 13.17 Horse that died with African horse sickness; death attributed to pulmonary edema. *Courtesy of USDA-APHIS, Foreign Animal Disease Diagnostic Laboratory, Plum Island, NY.*

Four clinical forms of AHS are recognized: pulmonary (peracute), cardiac (subacute), mixed pulmonary-cardiac (acute), and horse sickness fever. The **pulmonary form** is the most fatal, with mortality rates as high as 95%. Clinical signs develop within 3–5 days of the initial infection. The onset of symptoms is sudden, usually beginning with fever followed by congestion of the mucous membranes of the eyes, nose, and mouth. Animals sweat profusely, experience increased respiratory rates, and cough spasmodically owing to the accumulation of fluids in the lungs. Froth is commonly emitted from the nostrils in the terminal stage. Death usually occurs within a few days of the onset of clinical signs.

The **cardiac form** is similarly characterized by initial fever and congestion of the mucous membranes after an incubation period of 7–14 days. Animals subsequently develop extensive subcutaneous edema that is often apparent in the neck and jugular area, muscles along the back and hips, about the eyes and eyelids and the jaws. Other signs include depression and petechial hemorrhages on the underside of the tongue. Infected animals continue to feed and drink throughout the course of the disease. Death usually occurs in 4–8 days after the onset of fever, with mortality rates approaching 50%. The **mixed pulmonary-cardiac form** of AHS is characterized by clinical signs associated with each of the previous two syndromes. The onset of symptoms typically occurs 5–7 days after infection, with death ensuing 3–6 days later. The mortality is approximately 80%, intermediate

between the pulmonary and cardiac forms. **Horse sickness fever** is the mildest form of AHS. Infected animals usually recover after a low-grade fever, congested mucous membranes, loss of appetite, and mild depression over a 1-week period.

The principal vertebrate hosts of AHS virus are wild and domestic equids such as horses, mules, donkeys, and zebras. Mortality rates are highest in horses (70%−90%), intermediate in mules (50%), and low in donkeys (10%). Most infections in donkeys and zebras are subclinical or limited to mild fever. Zebras are believed to be the major reservoir hosts.

In endemic areas, native breeds seldom exhibit overt signs of infection; apparently, they develop a natural or acquired immunity. Most AHS outbreaks have occurred in European breeds of equids introduced to endemic areas or as a result of exposure of susceptible equids to infected animals imported from endemic areas in Africa and parts of the Middle East. Other animals in which the presence of antibodies indicates exposure to AHS virus are goats, sheep, domestic cattle, buffaloes, dromedaries, and elephants. None of these hosts develop more than mild clinical signs, but they may serve as potential reservoirs for the virus. Infected dogs, however, can develop clinical disease and are believed to be important reservoirs in urban areas. Six strains of AHS virus have been isolated from street dogs in Egypt, where a number of dogs died after consuming the uncooked meat of infected horse carcasses. The progression of the disease in dogs is similar to the pulmonary form in horses.

Until the mid-1900s, epizootics of AHS were largely confined to South Africa. Beginning in 1944 with an outbreak among horses in several Middle East countries, major epizootics have occurred in other parts of Africa, the Middle East, India, and Europe. The most devastating outbreak occurred in 1959−1960, in which over 300,000 horses died or had to be destroyed in six Middle East countries and Cyprus, Afghanistan, Pakistan, and India. Although cases of AHS were reported in Spain as early as 1965, the most severe epizootic to occur in Europe took place in Spain and Portugal in 1987−1990, in which more than 160 horses, mules, and donkeys died or had to be destroyed. Some of those animals were valuable thoroughbred horses participating in international equestrian competitions being held in Spain at the time. Ten zebras imported to a zoological park near Madrid, Spain, from Namibia in southern Africa are believed to have been the source of the infection. The virus was subsequently transmitted by indigenous *Culicoides* populations to Portugal and Morocco. Wind-borne midges, particularly *C. imicola*, are believed to have had a role in spreading the virus. Wind dispersal of *Culicoides* vectors may explain the spread of AHS virus from endemic areas of Africa, causing outbreaks in various parts of the Middle East, Cyprus, and Turkey in the Mediterranean region, and the Cape Verde Islands off the northwestern coast of Africa.

There currently is no cure for this disease, which leaves supportive therapy as the only means of treatment. Commercially available vaccines, however, have been helpful in protecting equines from infection in areas of Africa where AHS is endemic. Annual vaccinations are effective in maintaining immunity, reflecting the natural and complete immunity acquired by animals chronically exposed to this virus over extended periods. Regular vaccination of susceptible equines and strict control of the movement of unvaccinated animals are currently the only practical means of containing this disease.

The major vectors of AHS virus are *Culicoides* species. *Culicoides imicola* is the principal vector in Africa and the Middle East; *C. bolitinos* has a secondary role. Since the first isolation from field-collected *C. imicola* during an outbreak in South Africa, other species have been implicated as vectors. A few species have been shown to support replication of AHS virus after experimental inoculation with members of the *C. variipennis* complex, for example. The virus has been successfully transmitted 12−13 days after an infected blood meal. Some mosquitoes also are believed to be potential vectors, even though the virus has not been isolated from them under field conditions. *Culex pipiens*, *Aedes aegypti*, and *Anopheles stephensi* have been experimentally infected with the virus, but there is no strong evidence to indicate that these particular mosquitoes are natural vectors.

AHS virus has been isolated from naturally infected camel ticks (*Hyalomma dromedarii*) in Egypt, which raises a question about the possible involvement of this tick as a secondary vector. The brown dog tick *Rhipicephalus sanguineus* has been shown to be capable of biologically transmitting AHS virus between horses and dogs. However, the virus has not been isolated from naturally infected ticks of this species.

For a review of the history, transmission, and status of AHS, see Carpenter et al. (2017).

Schmallenberg Virus

Schmallenberg virus (SBV) is a species in the genus *Orthobunyavirus* of the family Peribunyaviridae. It is named after Schmallenberg, Germany, where it was discovered in 2011 and has since spread across Europe. Although it was first described in sheep, goats, and cattle, further epidemiological studies have linked SBV to wild ruminants such as deer, mouflon, and bison. In affected herds, SBV infection has been associated with diarrhea, fever, and decreased milk production. Although symptoms are generally mild and short-lived in adult ruminants, SBV infections can result in widespread abortions and developmental malformations in newborn domestic

ruminant livestock. SBV has been shown to be transmitted by *C. obsoletus*, *C. scoticus*, and *C. punctatus* (Balenghien et al., 2014; Larska et al., 2013a, 2013b).

For further information on this viral disease in European livestock, see Hoffmann et al., 2012; Larska et al., 2013a, 2014; Wernike et al., 2013, 2014; Bayrou et al., 2014; Yilmaz et al., 2014; Lievaart-Peterson et al., 2015.

Other Viral Agents

Culicoides species are suspected to be potential vectors of other viruses affecting livestock (Table 13.1), although their role in most cases remains uncertain. *Culicoides sonorensis*, for example, supports replication of Vesicular stomatitis virus and has been shown experimentally to be capable of transmitting the virus to cattle (Drolet et al., 2005; Leon and Tabachnick, 2006). Several other bovine arboviruses have been isolated from field-collected *C. oxystoma*, e.g., Akabane, Aino, Chuzan, D'Aguliar, and Ibaraki viruses in Japan (Yanase et al., 2005) and Shamonda virus in Japan and Nigeria. However, vector competence studies remain to be done before meaningful conclusions can be drawn regarding the importance of *Culicoides* species in the natural transmission of these viruses. *Culicoides imicola* and *C. bolitinos* are suspected vectors of Equine encephalosis virus among horses in southern Africa (Venter et al., 2002; Paweska and Venter, 2004).

Blood Protozoans

Biting midges are biological vectors of a number of protozoans called ***haemosporidians***, which are blood parasites of reptiles, birds, and mammals. Three genera that are transmitted by biting midges are *Haemoproteus* (Fig. 13.18), *Hepatocystis*, and *Leucocytozoon*. Most of the species are avian parasites that cause little or no apparent harm to their hosts. A few, however, such as *H. meleagridis* of turkeys and *L. caulleryi* of chickens, can cause significant problems for poultry producers. In addition, *Culicoides* species may serve as hosts for several avian trypanosomes (e.g., *Herpetomonas*, *Sergeia*).

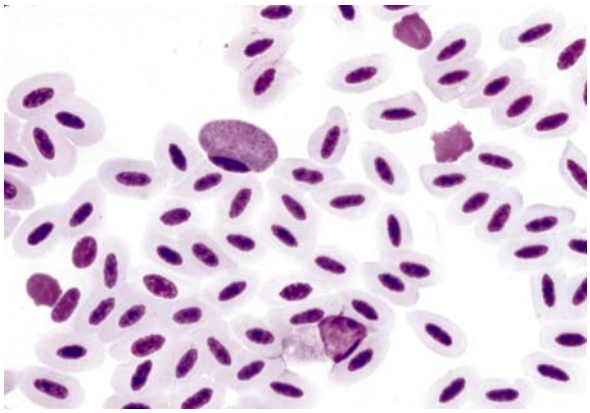


FIGURE 13.18 *Haemoproteus* sp., avian protozoan developing in blood cells of mourning dove (*Zenaida macroura*), transmitted by biting midges. *Photograph by Mary E. Hayes/Rogers.*

Haemosporidians transmitted by biting midges are related to malarial parasites (*Plasmodium* species) with which they share a similar life cycle and developmental stages. While feeding on an infected vertebrate host, female midges ingest red blood cells containing ***gametocytes***, the sexual stage of the parasite. In the midgut of the midge, the gametocytes are released, where they unite to form a motile zygote, the **ookinete**. The ookinete typically penetrates the peritrophic membrane and midgut tissue to form a cystlike structure or **oocyst** on the outer midgut wall. Within the oocyst, **sporozoites** are produced asexually, eventually rupturing from the mature oocysts into the haemocoel, where they make their way to the salivary glands and accumulate there. The sporozoite is the infective stage that is transmitted via the saliva to suitable hosts when the biting midge subsequently blood feeds. Development of the parasite in *Culicoides* species usually takes about 6–10 days.

Upon entering the vertebrate host, sporozoites invade cells of fixed tissues, notably the endothelium of various organs, and myofibroblasts, precursor cells that form muscle fibers. There they undergo one or more cycles of asexual reproduction called **schizogony** to produce **merozoites**. The merozoites then invade the blood and penetrate circulating erythrocytes. There, they develop into gametocytes and thus complete the life cycle.

Species of blood protozoans that are known to be transmitted by biting midges are summarized in Table 13.3. With the exceptions of *Haemoproteus kochi* that parasitizes Old World monkeys, and *Haemoproteus brayi* that parasitizes Malaysian squirrels, these haemosporidians are parasites of birds. The species are primarily members of the genus *Haemoproteus*, all of which are transmitted by *Culicoides* species. Relatively few details are known about most of these arthropod-borne haemosporidians and their associated *Culicoides* vectors. What is known is based primarily on studies of *H. meleagridis* and *L. caulleryi* as parasites of poultry.

Haemoproteus meleagridis

Haemoproteus meleagridis is primarily a parasite of wild and domestic turkeys. It can also cause at least transient infections in pheasants and chukars but apparently does not infect chickens, guinea fowl, bobwhite quail, and other gallinaceous birds.

This parasite is generally regarded as nonpathogenic. Even in cases in which large numbers of circulating red blood cells are infected with gametocytes, birds usually exhibit few signs of stress or other pathologic effects. This suggests that compensatory mechanisms are operative in

TABLE 13.3 Protozoans Transmitted by Biting Midges

Protozoan	Vertebrate Hosts	Geographic Area	Known or Suspected *Culicoides* Vectors
Haemoproteus			
H. danilewskyi	Crows, jays (Corvidae)	North America	*Culicoides arboricola, C. crepuscularis, C. edeni, C. stilobezzioides, C. sphagnumensis*
H. desseri (= *H. handai*)	Parakeets (Psittacidae)	Thailand	*C. nubeculosus* (experimental)
H. fringillae	Finches, sparrows (Fringillidae)	North America	*C. crepuscularis, C. stilobezzioides*
H. mansoni (= *H. canachites*)	Grouse (Tetraonidae)	North America	*C. sphagnumensis*
H. meleagridis	Turkey (Meleagrididae)	North America	*C. edeni, C. arboricola, C. haematopotus, C. hinmani, C. knowltoni*
H. nettionis	Ducks, geese, (Anatidae), other waterfowl	Canada	*C. downesi*
H. velans	Woodpeckers (Picidae)	North America	*C. sphagnumensis*
Hepatocystis			
H. brayi	Squirrels (Sciuridae)	Malaysia	*Culicoides* spp.
H. kochi	Monkeys (*Cercopithecus*)	Kenya	*C. adersi*
Leucocytozoon			
Leucocytozoon caulleryi	Chickens	Southeast Asia, Japan	*C. arakawae, C. circumscriptus, C. guttifer, C. schultzei*

which the replacement rate of erythrocytes is sufficient to maintain a stable hematocrit despite high parasitemia. In other cases, however, there is evidence that *H. meleagridis* harms its avian hosts, especially domestic turkeys. Heavy infections can result in anemia, reduced weight gain and growth rates, inflammation of skeletal and cardiac muscles, lameness, damage to the spleen and liver, and a wasting condition associated with chronic infections. Young birds are particularly vulnerable.

Five *Culicoides* species have been identified as vectors of *H. meleagridis* based primarily on studies in Florida (United States). *Culicoides edeni* is regarded as the most important vector; *C. hinmani*, *C. arboricola*, *C. haematopotus*, and *C. knowltoni* have secondary roles in transmission. Other species such as *C. baueri*, *C. nanus*, and *C. paraensis* have been shown to support the development of the parasite only to the oocyst stage. Transmission of *H. meleagridis* occurs throughout the year in southern Florida, whereas it is limited to the warmer months of the year throughout the rest of the United States where turkeys occur.

Other Haemoproteus *Species*

Haemoproteus danilewskyi, an avian parasite of the blue jay *Cyanocitta cristata* in Florida (United States), is capable of sporogonic development in *C. arboricola* and *C. edeni* (Garvin and Greiner, 2003). In California (United States), *Haemoproteus lophortyx*, a parasite of the California quail (*Callipepla californica*), is believed to be transmitted by *C. bottimeri* (Mullens et al., 2006). In Europe, *C. impunctatus* appears to be a likely vector of several *Haemoproteus* species that infect passerine birds, whereas other *Haemoproteus* species have been shown to have a detrimental effect on *C. impunctatus*, significantly decreasing its longevity (Valkiunas et al., 2002; Valkiunas and Lezhova, 2004).

Leucocytozoon caulleryi

This is the only *Leucocytozoon* species known to be transmitted by biting midges. It has been recognized for many years as causing a serious poultry disease of chickens in Japan and Southeast Asia, where it is known as **poultry leucocytozoonosis** and by the earlier name **Bangkok hemorrhagic disease**, where it occurred in Thailand. The principal vector of *L. caulleryi* is *Culicoides arakawae*, which commonly breeds in rice paddies.

Equine Onchocerciasis

Equine onchocerciasis is caused by the filarial nematode *Onchocerca cervicalis* (Fig. 13.19), the most widely

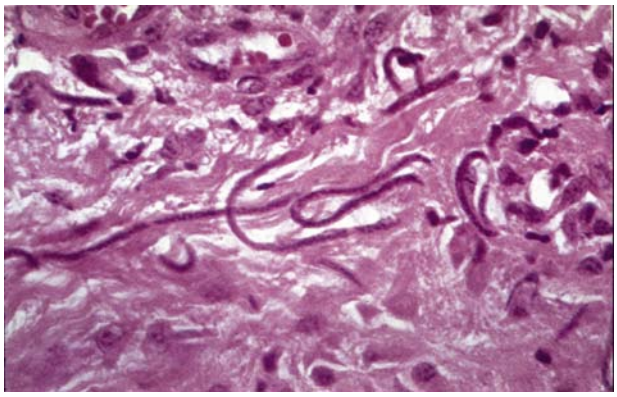


FIGURE 13.19 *Onchocerca cervicalis*, histological preparation showing microfilariae in skin of infested horse (Montes and Vaughan, 1983).

distributed nematode transmitted to domestic animals by biting midges. Horses are the only known host. Although it occurs worldwide, most problems associated with this nematode have been reported in the United States and Australia, where it commonly causes dermatitis. Various names that refer to infestations by *O. cervicalis* include **cutaneous equine onchocerciasis**, **equine ventral midline dermatitis**, **equine nuchal disease**, and **fistulous withers**. Prevalence of *O. cervicalis* is high in many regions of the United States; up to 85% or more of older horses have been reported to be infected with this parasite in New York, Kentucky, and the Gulf Coast states.

Adult worms occur primarily in the nuchal ligament of the neck and between the shoulder blades, or withers. Microfilariae produced by the females move to the skin, where they are active in the dermal tissues, often eliciting a host response in the form of localized inflammation and pruritus. The highest concentrations of microfilariae tend to be along the ventral midline of the horse. High numbers of microfilariae also may occur in skin of the inner thighs, chest region, withers, and eyelids. The density of microfilariae in skin tissue varies seasonally; it is highest during the spring and summer months and lowest in the winter, when they move to the deeper dermal layers. This is correlated with seasonal activity of most biting midges.

Horses that become sensitized to *O. cervicalis* develop various types of skin lesions including depigmentation, pruritus, scaling, and hair loss. This usually occurs on the face, chest, withers, and ventral midline, where microfilariae are most abundant. Ocular lesions have also been reported. Diagnosis is based on clinical signs and the detection of microfilariae in skin biopsies. Treatment with ivermectin has been found to be effective in killing microfilariae but not the adults. In most cases, the skin lesions show significant improvement or are completely resolved within a few weeks after treatment.

Members of the *C. variipennis* complex are the only known vectors of *O. cervicalis* in North America. Based primarily on laboratory studies, *Culicoides victoriae*, *Forcipomyia townsvillensis*, and the blackfly *Austrosimulium pestilens* also have been identified as potential vectors of *O. cervicalis* in Australia. Because these biting midges tend to ingest few microfilariae while feeding on an infected animal, only one or two infective third-stage larvae are typically found in field-collected flies.

Other Filarial Nematodes

At least three other *Onchocerca* species that infest bovine and equine hosts are believed to be transmitted by biting midges (Table 13.2): *Onchocerca gibsoni* of cattle in Southeast Asia, Malaya, Australia, and South Africa; *O. gutturosa* of cattle; and *O. reticulata* of horses and ponies in Australia. They are considered to be nonpathogenic.

Equine Allergic Dermatitis

Horses exposed to bites of certain *Culicoides* species commonly exhibit an allergic skin reaction. This typically occurs as a seasonal dermatitis affecting the withers, mane, tail, and ears. The back, ventral midline, and other body regions also can be affected, presumably reflecting the feeding sites of different biting midges involved. Equine allergic dermatitis was first attributed to *Culicoides* bites in Australia in the early 1950s, where it was known as **Queensland itch**. It is now known to occur widely throughout the world by various names such as **sweet itch**, **summer dermatitis**, **summer recurrent dermatitis**, **summer eczema**, **equine *Culicoides* sensitivity**, **Dhobie itch** (Philippines), and **Kasen disease** (Japan). A similar seasonal dermatitis in response to *Culicoides* bites also occurs in sheep.

The dermal response apparently is a sensitivity reaction to components of salivary fluids introduced to the bite wound while the flies are feeding (Fig. 13.20). Normal,

FIGURE 13.20 Allergic dermatitis in neck region of horse in response to injections of *Culicoides* extracts. *Courtesy of Yehuda Braverman, Kimron Veterinary Institute, Israel.*

nonsensitized horses usually react to these bites by developing small welts with relatively little associated discomfort. Sensitized horses, however, react more severely by developing intense local inflammation and pruritus; this can result in irritability, rubbing and scratching of involved areas, open wounds and secondary infections. Ponies are especially sensitive. Affected animals often are unsuitable for riding and, in the case of show horses, may have a substantial decrease in commercial value because of their irritable behavior, hair loss, and skin blemishes.

Once sensitized, horses experience either an immediate hypersensitivity response that peaks within 4 h or a delayed hypersensitivity response in which large welts develop after 24 h, with inflammation persisting up to 3 weeks or more. There is good evidence to show that *Culicoides*-induced hypersensitivity is a polygenic hereditary trait that predisposes certain animals to this response. This sensitivity occurs primarily in older horses, usually after 4−5 years of age.

A number of *Culicoides* species have been implicated as the cause of equine allergic dermatitis. Most are based on correlations between seasonal occurrences of the midges and clinical signs, biting sites on horses, and positive reactions to intradermal injections of horses with extracts of the respective biting midges. The following species are suspected of being involved: *C. insignis*, *C. obsoletus*, *C. spinosus*, *C. stellifer* and *C. venustus* in the United States; *C. pulicaris* in England; *C. nubeculosus* and *C. punctatus* in Ireland; *C. chiopterus*, *C. impunctatus*, and *C. obsoletus* in Norway; *C. imicola* in Israel; and *C. brevitarsis* in Australia.

Treatments for equine allergic dermatitis in the form of antihistamines and corticosteroids usually provide only temporary relief of symptoms. Desensitization of animals with injections of *Culicoides* extracts has not proved to be effective. Horse owners in areas where this condition is recognized as a problem should avoid breeding their animals with lineages of known sensitivity. Insecticides applied directly to horses to repel or kill biting midges affords some protection and can substantially reduce the severity if administered on a regular basis throughout the fly season. Ivermectin is ineffective, however. Stabling horses at night or pasturing them away from the attack of biting midges can also alleviate the problem.

PREVENTION AND CONTROL

Larviciding generally has not been effective in reducing populations of biting midges. Often the breeding sites are not easily located and may be so dispersed that the application of insecticides to kill the immature stages is not practical. In some situations, modifications of the habitat can reduce breeding sites by filling low-lying areas, diking, and regulating water levels to disrupt breeding and larval development. Eliminating seepage areas and leaking water troughs in or around livestock facilities can discourage the breeding of important species such as *C. sonorensis* and *C. variipennis*. Proper maintenance of farm ponds and fluctuation of the water level in dairy ponds and waste lagoons can reduce the numbers of adult biting midges that emerge. Disking of low-lying crop lands and the use of appropriate irrigation schedules have been effective in reducing adult populations of some pest species.

Adulticides have been used with limited success in suppressing adults. To be effective, they are usually applied as mists or fogs in the evening hours when the insects are most active. In coastal areas where problems can be especially severe, aerial applications of ultralow volume formulations of insecticides to salt marshes bordering populated areas can provide some relief. Ground applications using truck-mounted mist sprayers for control of biting midges are sometimes conducted in conjunction with municipal mosquito control programs in problem areas.

Individual protection of humans and other animals is often the only practical means of discouraging ceratopogonid midges from biting. Scheduling outdoor activities to avoid the peak biting periods of troublesome species is advisable. Animals such as horses can be stabled at night to protect them from species that do not readily enter buildings and shelters to feed. The mesh sizes of most window and door screens are not effective in excluding biting midges, especially in the case of species such as *C. furens* and *C. paraensis* that will enter buildings in search of hosts. Treatment of resting surfaces, such as the walls and roofs of animal shelters, with residual insecticides has been shown to reduce the numbers of certain *Culicoides* species. Insect repellents applied to exposed skin and the use of jackets and other clothing impregnated with compounds such as *N*,*N*-diethyl-meta-toluamide can provide effective protection from the bites of many of the more troublesome species.

For a review of techniques to control biting midges, see Carpenter et al. (2008); and for an assessment of mermithid nematodes as biological control agents, see Mullens et al. (2008).

REFERENCES AND FURTHER READING

Akiba, K. (1960). Studies on the *Leucocytozoon* found in the chicken in Japan. II. On the transmission of *Leucocytozoon caulleryi* by *Culicoides arakawae*. *The Japanese Journal of Veterinary Science, 22*, 309−317.

Akiba, K. (1970). Leucocytozoonosis of chickens. *National Institute of Animal Health, Quarterly, 10*(Suppl.), 131−147.

Anderson, G. S., Belton, P., & Kleider, N. (1993). Hypersensitivity of horses in British Columbia to extracts of native and exotic species of *Culicoides* (Diptera: Ceratopogonidae). *Journal of Medical Entomology, 30*, 657–663.

Atchley, W. R., Wirth, W. W., Gaskins, C. T., & Strauss, S. L. (1981). *A bibliography and keyword index of the biting midges (Diptera: Ceratopogonidae)*. USDA, Sci. and Edu. Admin., Bibliographies and Literature of Agriculture No. 13, 544 p.

Atkinson, C. T. (1988). Epizootiology of *Haemoproteus meleagridis* (Protozoa: Haemosporina) in Florida: Potential vectors and prevalence in naturally infected *Culicoides* (Diptera: Ceratopogonidae). *Journal of Medical Entomology, 25*, 39–44.

Atkinson, C. T. (1991). Vectors, epizootiology, and pathogenicity of avian species of *Haemoproteus* (Haemosporina: Haemoproteidae). *Bulletin of the Society for Vector Ecology, 16*, 109–126.

Balenghien, T., Pages, N., Goffredo, M., Carpenter, S., Augot, D., Jacquier, E., et al. (2014). The emergence of Schmallenberg virus across *Culicoides* communities and ecosystems in Europe. *Preventive Veterinary Medicine, 116*, 360–369.

Barber, J., Harrup, L. E., Silk, R., Veronesi, E., Gubbins, S., Bachanek-Bankowska, K., & Carpenter, S. (2018). Blood-feeding, susceptibility to infection with Schmallenberg virus, and phylogenetics of *Culicoides* (Diptera: Ceratopogonidae) from the United Kingdom. *Parasites and Vectors, 11*, 1–13.

Bluetongue and related orbiviruses. In Barber, T., & Jochim, M. J. (Eds.), *Progress in Clinical and Biological Research* (Vol. 178), (1985), 746 p.

Battle, F. V., & Turner, E. C., Jr. (1971). A systematic review of the genus *Culicoides* (Diptera: Ceratopogonidae) of Virginia. In *, Vol. 44. The Insects of Virginia No. 3*. VA: Polytechnic Instit. and State Univ., Bull., 129 p.

Bayrou, C., Garigliany, M. M., Sarlet, M., Sartelet, A., Cassart, D., & Desmecht, D. (2014). Natural intrauterine infection with Schmallenberg virus in malformed newborn calves. *Emerging Infectious Diseases, 20*, 1327–1330.

Bellis, G. A., Halling, L., & Anderson, S. J. (2015). Pictorial key to adult female *Culicoides* Latreille, 1809 (Diptera: Ceratopogonidae) from the northern territory, western Australia and south Australia. *Austral Entomology, 54*, 28–59.

Blanton, F. S., & Wirth, W. W. (1979). The sand flies (*Culicoides*) of Florida (Diptera: Ceratopogonidae). *Arthropods of Florida and Neighboring Land Areas, 10*, 294 p.

Boorman, J. (1993). Biting midges (Ceratopogonidae). In R. P. Lane, & R. W. Crosskey (Eds.), *Medical insects and arachnids* (pp. 288–301). London: Chapman & Hall.

Boorman, J., & Hagan, D. V. (1996). A name list of world *Culicoides* (Diptera: Ceratopogonidae). *International Journal of Dipterological Research, 7*, 161–192.

Borkent, A. (2004a). Ceratopogonidae. In W. C. Marquart (Ed.), *Biology of disease vectors* (pp. 113–126). San Diego: Elsevier Inc.

Borkent, A. (2004b). *Austroconops* Wirth and Lee, a lower cretaceous genus of biting midges yet living in western Australia: A new species, first description of the immatures and discussion of their biology and phylogeny (Diptera: Ceratopogonidae). *American Museum Novitates, 3449*, 1–67.

Borkent, A. (2014). The pupae of the biting midges of the world (Diptera: Ceratopogonidae), with a generic key and analysis of the phylogenetic relationships between genera. *Zootaxa, 3879*, 1–327.

Borkent, A., & Grogan, W. L., Jr. (2009). Catalog of the new world biting midges north of Mexico (Diptera: Ceratopogonidae). *Zootaxa, 2273*, 1–48.

Borkent, A., & Spinelli, G. (2007). *Neotropical Ceratopogonidae, Diptera, Insecta (Aquatic Biodiversity in Latin America)*. Pensoft Publishers.

Borkent, A., & Wirth, W. W. (1997). World species of biting midges (Diptera: Ceratopogonidae). *Bulletin of the American Museum of Natural History, 233*, 1–257.

Carpenter, S., Mellor, P. S., Fall, A. G., Garros, C., & Venter, G. (2017). African horse sickness virus: History, transmission, and current status. *Annual Review of Entomology, 62*, 343–358.

Carpenter, S., Mellor, P., & Torr, S. J. (2008). Control techniques for *Culicoides* biting midges and their application in the U.K. and northwestern Palaearctic. *Medical and Veterinary Entomology, 22*, 1–13.

Celma, C. C., Stewart, M., Wernike, K., Eschbaumer, M., Gonzalez-Molleda, L., Breard, E., et al. (2017). Replication-deficient Particles: New insights into the next generation of bluetongue virus vaccines. *Journal of Virology, 91*, 1–16.

Coetzer, J. A. W., & Tustin, R. C. (Eds.). (2005). *Infectious diseases of livestock, with special reference to South Africa* (2nd ed., 3 *Vols*. Oxford University Press.

da Rosa, J. F. T., de Souza, W. M., Pinheiro, F. D., Figueiredo, M. L., Cardoso, J. F., Acrani, G. O., & Nunes, M. R. T. (2017). Oropouche virus: Clinical, epidemiological, and molecular aspects of a neglected Orthobunyavirus. *American Journal of Tropical Medicine and Hygiene, 96*, 1019–1030.

DeHaven, W. R., Valle Molina, J. A., & Evans, B. (2004). Bluetongue viruses and trade issues: A North American perspective. *Veterinaria Italiana, 40*, 683–687.

Downes, J. A., & Wirth, W. W. (1981). Ceratopogonidae. In J. F. McAlpine (Ed.), *Vol. 1. Manual of Nearctic Diptera* (pp. 393–421). Research Branch, Agriculture Canada. Monogr. 17.

Drolet, B. S., Campbell, C. L., Stuart, M. A., & Wilson, W. C. (2005). Vector competence of *Culicoides sonorensis* (Diptera: Ceratopogonidae) for vesicular stomatitis virus. *Journal of Medical Entomology, 42*, 409–418.

Dulac, G. C., Sterritt, W. G., Dubuc, C., Afshar, A., Myers, D. J., Taylor, E. A., et al. (1991). Incursions of orbiviruses in Canada and their serologic monitoring in the native animal populations between 1962 and 1991. In T. E. Walton, & B. I. Osburn (Eds.), *Bluetongue, African horsesickness, and related orbiviruses* (pp. 120–128). Boca Raton, FL: CRC Press.

Eberhard, M. L., & Orihel, T. C. (1984). The genus *Mansonella* (syn. *Tetrapetalonema*). A new classification. *Annales de Parasitologie Humaine et Comparée, 59*, 483–496.

Foil, L., Stage, D., & Klei, T. R. (1984). Assessment of wild-caught *Culicoides* (Ceratopogonidae) species as natural vectors of *Onchocerca cervicalis* in Louisiana. *Mosquito News, 44*, 204–206.

Garnham, P. C. C., Desser, S. S., & Khan, R. A. (1974). On species of *Leucocytozoon*, *Haemoproteus* and *Hepatocystis*. In J. P. Kreier (Ed.), *Vol. 3. Parasitic Protozoa* (pp. 239–266).

Garvin, M. C., & Greiner, E. C. (2003). Ecology of *Culicoides* (Diptera: Ceratopogonidae) in Southcentral Florida and experimental *Culicoides* vectors of the avian hematozoan *Haemoproteus danilewskyi* Kruse. *Journal of Wildlife Diseases, 39*, 170–178.

Gerdes, G. H. (2004). A South African overview of the virus, vectors, surveillance and unique features of bluetongue. *Veterinaria Italiana, 40*, 39–42.

Gibbs, E. P. J., & Greiner, E. C. (1988). Bluetongue and epizootic hemorrhagic disease. In T. P. Monath (Ed.), *The arboviruses: Epidemiology and ecology* (Vol. 2, pp. 39–70). Boca Raton, FL: CRC Press.

Gibbs, E. P. J., & Greiner, E. C. (1994). The epidemiology of bluetongue. *Comparative Immunology, Microbiology & Infectious Diseases, 17*, 207–220.

Glick, J. I. (1990). *Culicoides* biting midges (Diptera: Ceratopogonidae) of Kenya. *Journal of Medical Entomology, 27*, 87–195.

Gloster, J., Mellor, P. S., Manning, A. J., Webster, H. N., & Hort, M. C. (2007). Assessing the risk of windborne spread of bluetongue in the 2006 outbreak of disease in northern Europe. *Veterinary Record, 160*, 54–56.

Glukhova, V. M. (1977). The subgeneric classification of the genus *Culicoides* Latreille, 1809 (Diptera: Ceratopogonidae), including morphological characters of the larvae (in Russian). *Parazitol Sbornik, 27*, 112–128.

Glukhova, V. M. (1979). *Larval midges of the subfamilies Palpomyiinae and Ceratopogoninae of the fauna of the U.S.S.R. (in Russian)*. Leningrad: Nauka Publishers, 230 p.

Glukhova, V. M. (1989). *Blood-sucking midges of the genera Culicoides and Forcipomyia (Ceratopogonidae) of the fauna of the U.S.S.R. (in Russian)* (p. 407). Leningrad: Nauka Publishers.

Gomez-Tejedor, C. (2004). Brief overview of the bluetongue situation in Mediterranean Europe, 1998-2004. *Veterinaria Italiana, 40*, 57–60.

Greiner, E. C. (1995). Entomological evaluation of insect hypersensitivity in horses. *Veterinary Clinics of North America: Equine Practice, 11*, 29–41.

Greiner, E. C., Fadok, V. A., & Rabin, E. B. (1990). Equine *Culicoides* hypersensitivity in Florida: Biting midges aspirated from horses. *Medical and Veterinary Entomology, 4*, 375–381.

Greiner, E. C., Mo, C. L., Tanya, V., Thompson, L. H., & Oviedo, M. T. (1991). Vector ecology of bluetongue viruses in Central America and the Caribbean. In T. E. Walton, & B. I. Osburn (Eds.), *Bluetongue, African horsesickness, and related orbiviruses* (pp. 320–324).

Halldorsdottir, S., & Larsen, H. J. (1991). An epidemiological study of summer eczema in Icelandic horses in Norway. *Equine Veterinary Journal, 23*, 296–299.

Hess, W. R. (1988). African horse sickness. In T. P. Monath (Ed.), *The arboviruses: Epidemiology and ecology* (Vol. 2, pp. 1–18). Boca Raton, FL: CRC Press.

Hoffmann, B., Scheuch, M., Hoper, D., Jungblut, R., Holsteg, M., Schirrmeier, H., et al. (2012). Novel orthobunyavirus in cattle, Europe, 2011. *Emerging Infectious Diseases, 18*, 469–472.

Holbrook, F. R., Tabachnick, W. J., Schmidtmann, E. T., McKinnon, C. N., Bobian, R. J., & Grogan, W. L. (2000). Sympatry in the *Culicoides variipennis* complex (Diptera: Ceratopogonidae): A taxonomic reassessment. *Journal of Medical Entomology, 37*, 65–76.

Hunt, G. J. (1994). *A procedural manual for the large-scale rearing of the biting midge, Culicoides variipennis (Diptera: Ceratopogonidae)*. U.S. Dept. Agric., Agric. Res. Ser., ARS-121, 68 p.

Jamnback, H. A. (1965). *The Culicoides of New York State (Diptera: Ceratopogonidae)*. New York State Mus. Sci. Serv. Bull. No. 399, 154 p.

Jones, R. H., Luedke, A. J., Walton, T. E., & Metcalf, H. E. (1981). Bluetongue in the United States; an entomological perspective toward control. *World Animal Review, 38*, 2–8.

Kettle, D. S. (1965). Biting ceratopogonids as vectors of human and animal diseases. *Acta Tropica, 22*, 356–362.

Kettle, D. S. (1972). The biting habits of *Culicoides furens* (Poey) and *C. barbosai* Wirth and Blanton. III. Seasonal cycle, with a note on the relative importance of ten factors that might influence the biting rate. *Bulletin of Entomological Research, 61*, 565–576.

Kettle, D. S. (1984). *Medical and veterinary entomology* (pp. 536–541). John Wiley & Sons.

Lager, I. A. (2004). Bluetongue in South America: Overview of viruses, vectors, surveillance and unique features. *Veterinaria Italiana, 40*, 89–93.

Larska, M., Krzysiak, M. K., Kesik-Maliszewska, J., & Rola, J. (2014). Cross-sectional study of Schmallenberg virus seroprevalence in wild ruminants in Poland at the end of the vector season of 2013. *BMC Veterinary Research, 10*(967), 1–7.

Larska, M., Krzysiak, M., Smreczak, M., Polak, M. P., & Zmudzinski, J. F. (2013a). First detection of Schmallenberg virus in elk (*Alces alces*) indicating infection of wildlife in Bialowieza National Park in Poland. *The Veterinary Journal, 198*, 279–281.

Larska, M., Lechowski, L., Grochowska, M., & Zmudzinski, J. F. (2013b). Detection of the Schmallenberg virus in nulliparous *Culicoides obsoletus/scoticus* complex and *C. punctatus*–the possibility of transovarial virus transmission in the midge population and of a new vector. *Veterinary Microbiology, 166*, 467–473.

LeDuc, J. W., Hoch, A. L., Pinheiro, F. P., & Travassos da Rosa, A. P. A. (1981). Epidemic Oropouche virus disease in northern Brazil. *Bulletin of the Pan American Health Organization, 15*, 97–193.

Leon, A., & Tabachnick, W. J. (2006). Transmission of vesicular stomatitis New Jersey virus to cattle by the biting midge *Culicoides sonorensis* (Diptera: Ceratopogonidae). *Journal of Medical Entomology, 43*, 323–329.

Lievaart-Peterson, K., Luttikholt, S., Peperkamp, K., Van den Brom, R., & Vellema, P. (2015). Schmallenberg disease in sheep or goats: Past, present and future. *Veterinary Microbiology, 181*, 147–153.

Lillie, T. H., Kline, D. L., & Hall, D. W. (1987). Diel and seasonal activity of *Culicoides* spp. (Diptera: Ceratopogonidae) near Yankeetown, Florida, monitored with a vehicle-mounted insect trap. *Journal of Medical Entomology, 24*, 503–511.

Linley, J. R. (1983). Biting midges (Diptera: Ceratopogonidae) and human health. *Journal of Medical Entomology, 20*, 347–364.

Linley, J. R. (1985). Biting midges (Diptera: Ceratopogonidae) as vectors of nonviral animal pathogens. *Journal of Medical Entomology, 22*, 589–599.

Linley, J. R., & Adams, M. (1972). A study of the mating behavior of *Culicoides melleus* (Diptera: Ceratopogonidae). *Transactions of the Royal Entomological Society of London, 124*, 81–121.

Lubroth, J. (1988). African horsesickness and the epizootic in Spain 1987. *Equine Practice, 10*, 26–33.

Lubroth, J. (1991). The complete epidemiologic cycle of African horse sickness: Our incomplete knowledge. In T. E. Walton, & B. I. Osburn (Eds.), *Bluetongue, African horse sickness, and related orbiviruses* (pp. 197–204). Boca Raton, FL: CRC Press.

Mehlhorn, H., Walldorf, V., Klimpel, S., Jahn, B., Jaeger, F., Eschweiler, J., et al. (2007). First occurrence of *Culicoides obsoletus*-transmitted bluetongue virus epidemic in central Europe. *Parasitology Research, 101*, 219–228.

Meiswinkel, R., Gomulski, L. M., Delecolle, J. C., Goffredo, M., & Gasperi, G. (2004). The taxonomy of *Culicoides* vector complexes – unfinished business. *Veterinaria Italiana, 40*, 151–159.

Meiswinkel, R., Venter, G. J., & Neville, E. M. (2004). Vectors: *Culicoides* spp. In J. A. W. Coetzer, & R. C. Tustin (Eds.), *Vol. 1. Infectious diseases of livestock, with special reference to South Africa* (pp. 93–136). Oxford University Press.

Mellor, P. S. (1990). The replication of bluetongue virus in *Culicoides* vectors. In P. Roy, & B. M Gorman (Eds.), *Current topics in microbiology and immunology: Vol. 162. Bluetongue viruses* (pp. 143–161). Berlin: Springer-Verlag.

Mellor, P. S., & Hamblin, C. (2004). African horse sickness (special issue). In *Equine infectious diseases* (Vol. 4, pp. 445–466).

Miura, Y., Goto, Y., Kubo, M., & Kono, Y. (1988). Isolation of Chuzan virus, a new member of the Palyam subgroup of the genus *Orbivirus*, from cattle and *Culicoides oxystoma* in Japan. *American Journal of Veterinary Research, 49*, 2022–2025.

Mo, C. L., Thompson, L. H., Homan, E. J., Oviedo, M. T., Greiner, E. C., González, J., et al. (1994). Bluetongue virus isolations from vectors and ruminants in Central America and the Caribbean. *American Journal of Veterinary Research, 55*, 211–215.

Mullen, G. R., & Hribar, L. J. (1988). Biology and feeding behavior of ceratopogonid larvae (Diptera: Ceratopogonidae) in north America. *Bulletin of the Society for Vector Ecology, 13*, 60–81.

Mullens, B. A. (1991). Integrated management of *Culicoides variipennis*: A problem of applied ecology. In T. E. Walton, & I. Osburn (Eds.), *Bluetongue, African horsesickness, and related orbiviruses* (pp. 896–905). Boca Raton, FL: CRC Press.

Mullens, B. A., Cardona, C. J., McClellan, L., Szijj, C. E., & Owen, J. P. (2006). *Culicoides bottimeri* as a vector of *Haemoproteus lophortyx* to quail in California, USA. *Veterinary Parasitology, 140*, 35–43.

Mullens, B. A., Sarto I Monteys, V., & Przhiboro, A. A. (2008). Mermithid parasitism in the Ceratopogonidae: A literature review and critical assessment of host impact and potential for biological control. *Russian Entomological Journal, 17*, 87–113.

Murphree, C. S., & Mullen, G. R. (1991). Comparative larval morphology of the genus *Culicoides* Latreille (Diptera: Ceratopogonidae) in north America with a key to species. *Bulletin of the Society for Vector Ecology, 16*, 269–399.

Nolan, D. V., Carpenter, S., Barber, J., Mellor, P. S., Dallas, J. F., Mordue, A. J., et al. (2007). Rapid diagnostic PCR assays for members of the *Culicoides obsoletus* and *Culicoides pulicaris* species complexes, implicated vectors of bluetongue virus in Europe. *Veterinary Microbiology, 124*, 82–94.

Ottley, M. L., Dallemagne, C., & Moorhouse, D. E. (1983). Equine onchocerciasis in Queensland and the northern territory of Australia. *Australian Veterinary Journal, 60*, 200–203.

Paweska, J. T., & Venter, G. J. (2004). Vector competence of *Culicoides* species and the seroprevalence of homologous neutralizing antibody in horses for six serotypes of equine encephalosis virus (EEV) in South Africa. *Medical and Veterinary Entomology, 18*, 398–407.

Paweska, J. T., Venter, G. J., & Mellor, P. S. (2002). Vector competence of South African *Culicoides* species for bluetongue virus serotype 1 (BTV-1) with special reference to the effect of temperature on the rate of virus replication in *C. imicola* and *C. bolitinos*. *Medical and Veterinary Entomology, 16*, 10–21.

Pearson, J. E., Gustafason, G. A., Shafer, A. L., & Alstad, A. D. (1991). Distribution of bluetongue in the United States. In T. E. Walton, & B. I. Osburn (Eds.), *Bluetongue, African horsesickness, and related orbiviruses* (pp. 128–138). Boca Raton, FL: CRC Press.

Pinheiro, K. F., Travassos da Rosa, A. P., Travassos da Rosa, J. F., Ishak, R., Freitas, R. B., Gomez, M. L., et al. (1981). Oropouche virus I. A review of clinical, epidemiological, and ecological findings. *The American Journal of Tropical Medicine and Hygiene, 30*, 149–160.

Purse, B. V., Mellor, P. S., Rogers, D. J., Samuel, A. R., Mertens, P. P. C., & Baylis, M. (2005). Climate change and the recent emergence of bluetongue in Europe. *Nature Reviews Microbiology, 3*, 171–181.

Rawlings, P., Meiswinkel, R., Labuschange, K., Welton, N., Baylis, M., & Mellor, P. S. (2003). The distribution and species characteristics of the *Culicoides* biting midges of South Africa. *Ecological Entomology, 28*, 559–566.

Sebastiani, F., Meiswinkel, R., Gomulski, L. M., Guglielmino, C. R., Mellor, P. S., Malacrida, A. R., et al. (2001). Molecular differentiation of the Old World *Culicoides imicola* species complex (Diptera: Ceratopogonidae), inferred using random amplified polymorphic DNA markers. *Molecular Ecology, 10*, 1773–1786.

Spinelli, G. R., & Borkent, A. (2004). New species of central American *Culicoides* Latreille (Diptera: Ceratopogonidae) with a synopsis of species from Costa Rica. *Proceedings of the Entomological Society of Washington, 106*, 361–395.

Spinelli, G. R., Ronderos, M. M., Diaz, F., & Marino, P. I. (2005). The bloodsucking biting midges of Argentina (Diptera: Ceratopogonidae). *Memorias do Instituto Oswaldo Cruz, Rio de Janeiro, 100*, 137–150.

Tabachnick, W. J. (1992). Genetic differentiation among populations of *Culicoides variipennis* (Diptera: Ceratopogonidae), the North American vector of bluetongue virus. *Annals of the Entomological Society of America, 85*, 140–147.

Tabachnick, W. J. (1996). *Culicoides variipennis* and bluetongue-virus epidemiology in the United States. *Annual Review of Entomology, 41*, 23–43.

Tabachnick, W. J. (2004). *Culicoides* and the global epidemiology of bluetongue virus infection. *Veterinaria Italiana, 40*, 135–150.

Thomas, F. C. (1981). Hemorrhagic disease. In W. R. Davidson, F. A. Hayes, V. F. Nettles, & F. E. Kellogg (Eds.), *Diseases and parasites of white-tailed deer* (pp. 87–96). Tallahassee, FL: Miscellaneous Publications No. 7. Tall Timbers Research Station.

Valikiunas, G., & Lezhova, T. A. (2004). Detrimental effects of *Haemoproteus* infections on the survival of the biting midge *Culicoides impunctatus* (Diptera: Ceratopogonidae). *The Journal of Parasitology, 90*, 194–196.

Valikiunas, G., Liutkevicius, G., & Lezhova, T. A. (2002). Complete development of three species of *Haemoproteus* (Haemosporidia, Haemoproteidae) in the biting midge *Culicoides impunctatus* (Diptera, Ceratopogonidae). *The Journal of Parasitology, 88*, 864–868.

Venter, G. J., Groenewald, D., Venter, E., Hermanides, K. G., & Howell, P. G. (2002). A comparison of the vector competence of the biting midges, *Culicoides* (*Avaritia*) *bolitinos* and *C.* (*A.*) *imicola*, for the Bryanson serotype of equine encephalosis virus. *Medical and Veterinary Entomology, 16*, 372–377.

Walton, T. E. (2004). The history of bluetongue and a current global overview. *Veterinaria Italiana, 40*, 31–38.

Walton, T. E., & Osburn, B. I. (Eds.). (1991). *Bluetongue, African horsesickness, and related orbiviruses*. Boca Raton, Fla: CRC Press, 1042 p.

Ward, M. P. (1994). The epidemiology of bluetongue virus in Australia – a review. *Australian Veterinary Journal, 71*, 3–7.

Wernike, K., Conraths, F., Zanella, G., Granzow, H., Gache, K., Schirrmeier, H., et al. (2014). Schmallenberg virus–two years of experiences. *Preventive Veterinary Medicine, 116*, 423–434.

Wernike, K., Hoffmann, B., Breard, E., Botner, A., Ponsart, C., Zientara, S., et al. (2013). Schmallenberg virus experimental infection of sheep. *Veterinary Microbiology, 166*, 461–466.

Wirth, W. W., Dyce, A. L., & Peterson, B. V. (1985). An atlas of wing photographs, with a summary of the numerical characters of the Nearctic species of *Culicoides* (Diptera: Ceratopogonidae). *Contributions of the American Entomological Institute, 22*(4), 46 p.

Wirth, W. W., Dyce, A. L., & Spinelli, G. R. (1988). An atlas of wing photographs, with a summary of the numerical characters of the Neotropical species of *Culicoides* (Diptera: Ceratopogonidae). *Contributions of the American Entomological Institute, 25*(1), 72 p.

Wirth, W. W., & Hubert, A. A. (1989). The *Culicoides* of Southeast Asia (Diptera: Ceratopogonidae). *Memoirs of the American Entomological Institute, 44*, 508 p.

Wirth, W. W., Ratanaworabhan, N. C., & Blanton, F. S. (1974). Synopsis of the genera of Ceratopogonidae (Diptera). *Annales de Parasitologie Humaine et Comparée, 49*, 595−613.

Yanase, T., Kato, T., Kubo, T., Yoshida, K., Ohashi, S., Yamakawa, M., et al. (2005). Isolation of bovine arboviruses from *Culicoides* biting midges (Diptera: Ceratopogonidae) in southern Japan: 1985-2002. *Journal of Medical Entomology, 42*, 63−67.

Yilmaz, H., Hoffmann, B., Turan, N., Cizmecigil, U. Y., Richt, J. A., & Van der Poel, W. H. (2014). Detection and partial sequencing of Schmallenberg virus in cattle and sheep in Turkey. *Vector-Borne and Zoonotic Diseases, 14*, 223−225.

Yu, Y. (2005). *Catalog and keys of Chinese Ceratopogonidae (Insecta, Diptera) (in Chinese)*. Beijing: Military Medical Science Press.

Yu, Y. (2006). *Ceratopogonidae of China (Insecta, Diptera) (in Chinese)* (Vol. 2). Beijing: Military Medical Science Press.

Yu, Y., & Liu, J. (2006). *World species of bloodsucking midges (Diptera: Ceratopogonidae)*. Beijing: Military Medical Science Press.

Chapter 14

Black Flies (Simuliidae)

Peter H. Adler[1] and John W. McCreadie[2]

[1]*Department of Plant and Environmental Sciences, Clemson University, Clemson, SC, United States;* [2]*Department of Biological Sciences, University of South Alabama, Mobile, AL, United States*

As small, powerful fliers adapted for blood-feeding, black flies can be formidable pests of humans, domestic animals, and wildlife, affecting virtually all facets of outdoor life. They are distributed worldwide, with the exception of Antarctica and some oceanic islands (e.g., Hawaii), and inhabit elevations from below sea level to at least 5,000 m above sea level. Areas with large altitudinal variation have more species. Their distribution is largely influenced by the availability of flowing water, which is required for development of the immature stages, probably driven by respiratory needs and the filter-feeding mode of life. Many of the worst pest species breed in large rivers, some of which can produce nearly a billion flies per kilometer of riverbed per day (Adler et al., 2016). Other pest species inhabit the myriad small streams of heavily wooded terrain, making management efforts difficult.

Often ranked third globally among arthropods in importance as vectors of disease agents, black flies also are among the few arthropods that have killed animals via exsanguination during massive attacks. Even when not biting, their persistent swarming behavior can create an intolerable nuisance as the blood-seeking females dart into facial orifices and crawl on the skin. As often is the case, the behavior of a minority defines the reputation of the group. So it is with black flies, for only about 10%–20% of the world's species are actually pests of humans and their animals. Among these species, however, are the vectors of the agents of human onchocerciasis and mansonellosis, bovine onchocerciasis, and avian leucocytozoonosis. The majority of species go unnoticed, because either they do not feed as adults or their hosts are of little economic concern.

TAXONOMY

More than 2,200 species of black flies have been described worldwide (Adler and Crosskey, 2018). The Palearctic Region contains the most described species, about 700, followed by the Oriental and Neotropical Regions with well over 500 and 300 species, respectively (Currie and Adler, 2008; Takaoka, 2017). The Nearctic Region has about 256 known species (Adler et al., 2004).

The Simuliidae consist of two subfamilies. The most primitive subfamily, **Parasimuliinae,** includes one genus and four described and one undescribed species endemic to the Pacific Northwest. The females of these species do not have biting mouthparts. The subfamily **Simuliinae** contains all remaining species and is divided into two tribes, the **Prosimuliini** and the **Simuliini**, the latter including the majority of pest species. The most universally accepted classification system below the tribal level is summarized by Adler and Crosskey (2018), who recognize 25 extant genera in the subfamily Simuliinae. The largest genus of black flies is *Simulium*, which contains 37 subgenera and about 81% of all species, including more than 90% of the major pest and vector species.

The morphological uniformity of black flies creates difficulty for species identification. For this reason, a holistic approach to identification is typically used, relying on characters from larvae, pupae, males, females, the polytene chromosomes, and DNA barcodes, as well as distributional and ecological information. The need for accurate identifications, particularly in programs for pest and vector management, has driven the taxonomy of black flies. As a result, black flies are taxonomically one of the best known groups of arthropods at the species level; for example, about 98% of North American species are known as larvae and pupae.

More than 150 identification keys exist for black flies in various parts of the globe. Crosskey and Howard (1997) provide a comprehensive list of identification keys by zoogeographic region. Keys to the genera and species of adults, pupae, and larvae of the Nearctic Region are provided by Adler et al. (2004). The most comprehensive

Medical and Veterinary Entomology. https://doi.org/10.1016/B978-0-12-814043-7.00014-5

English-language treatment of the Palearctic fauna is by Rubtsov (1956), and of the Neotropical fauna, by Coscarón and Coscarón Arias (2007) and Shelley et al. (2010). Keys to the supraspecific taxa of the Australasian and Afrotropical Regions are given by Crosskey (1967, 1969, respectively). Comprehensive keys for the Oriental Region are lacking, but the keys by Takaoka and Davies (1995), Takaoka (2003), and Chen (2016) provide a helpful starting point.

The giant polytene chromosomes (usually $n = 3$), which are best developed in the larval silk glands, provide a highly useful tool for discovering and identifying species. Studies of these giant chromosomes in the Simuliidae have generated the largest literature on the genetics of natural populations for any group of organisms (Adler et al., 2010). Giant chromosomes, particularly their banding patterns, reveal that many black flies regarded as single species are actually complexes of two or more species known as cryptic species or sibling species, each of which is biologically unique. The existence of cryptic species has far-reaching implications for biological studies and population management of pests and vectors. For example, *Simulium damnosum*, the black fly known for much of the 20th century as a vector of the agent of human onchocerciasis or river blindness, is the largest species complex among all hematophagous arthropods worldwide, consisting of more than 55 cytologically distinct entities (Post et al., 2007). Many of these entities are distinct species, but not all are vectors of the parasite that causes human onchocerciasis.

Cytotaxonomy of black flies has been reviewed for the world fauna (Adler and Crosskey, 2015). Molecular analyses are becoming an increasingly routine addition to species discovery and identification (Colorado-Garzón et al., 2016).

FIGURE 14.2 Larvae of black flies. (A) North American species, *Simulium venustum*, attached to aquatic vegetation, filter feeding. (B) Head of the larva of the Australian black fly *Paracnephia strenua*, showing details of the labral fans used in filter feeding. *(A) Photograph by Stephen A. Marshall. (B) © Douglas A. Craig.*

FIGURE 14.1 Embryonated eggs of the North American black fly *Simulium vittatum*, which deposits eggs in masses on in-stream substrates. *© Jena Johnson.*

MORPHOLOGY

The immature stages of black flies are adapted for aquatic life, although the nonmobile pupa also has terrestrial adaptations that are useful if the water recedes. The egg is roughly oval or triangular with rounded angles (Fig. 14.1). It has a glutinous outer layer and a smooth, pigmented inner shell. A micropyle, consisting of a simple hole in the egg for the entry of sperm, is present in some species but not others; if a micropyle is absent, sperm entry is facilitated enzymatically.

The larva (Fig. 14.2A) hatches with the aid of an egg burster, a small tubercle on the dorsum of the head capsule. The basic larval design consists of a well-sclerotized head capsule bearing an anterior pair of labral fans (Fig. 14.2B) and an elongate body with one thoracic proleg and a terminal abdominal proleg. Rows of tiny hooks on the prolegs enmesh with silk pads spun from a pair of larval silk glands and applied to a substrate. These silk glands extend from the anterior of the head into the posterior portion of the abdomen,

where they enlarge and double back onto themselves. The adhesiveness of the silk is correlated with the velocity of the flowing water to which each species is adapted.

While clinging to a pad by its posterior proleg, the larva extends its body to filter feed. The prominent labral fans, each with about 20–80 individual rays bearing microtrichia (minute hairs) on their inner surface, are used to filter particulate matter from the water current. Larvae of some species (e.g., *Gymnopais* spp.) that live in habitats, such as glacial meltwaters, with little suspended food have lost the labral fans over evolutionary time. These species rely on their mandibles, specialized labrum, and hypostoma to scrape food from the substrate.

Additional features of the head and body are conspicuous and taxonomically important. The antennae, which consist of three articles and a terminal cone sensillum, are elongate, slender, and variously pigmented. A pair of dark eyespots is prominent on each side of the head capsule. Pigmentation patterns of the head capsule and body and the shape of the **postgenal cleft**, an area of weakly sclerotized cuticle on the ventral side of the head capsule, are important for interpreting the taxonomy of the family. The anteroventral portion of the head capsule bears the **hypostoma**, an anteriorly toothed plate used in conjunction with the mandibles to cut strands of silk and to scrape food from the substrate. Mature larvae are recognized by the presence of a prominent, dark gill histoblast on each side of the thorax.

The pupa (Fig. 14.3), which resembles an adult with its appendages held close to the body, is housed in a silk cocoon. Cocoons are shapeless sacs in the evolutionarily older species but are well-formed, slipper- or boot-shaped coverings sometimes bearing anterior processes and lateral windows in the more derived species. The pupa is held firmly in its cocoon by numerous anteriorly directed sets of hooklets. A pair of conspicuous gills arises from the thorax. The gills are among the most taxonomically useful and fascinating structures in any life stage. They vary in arrangement from thick, clublike structures to clusters of two to more than 100 slender filaments.

Adult black flies (Figs. 14.4–14.6) are characterized by a small but robust body; conical or beadlike antennae with seven to nine flagellomeres, in addition to the scape and pedicel; and an arched thorax bearing a pair of wings that typically span 6–10 mm and have thickened veins near the leading margin. Most species are blackish, but orange, yellow, and variously patterned species also exist. Males (Fig. 14.6) of nearly all species are holoptic, with eyes that occupy most of the head and meet at the midline. Male eyes consist of enlarged dorsal facets, in addition to the

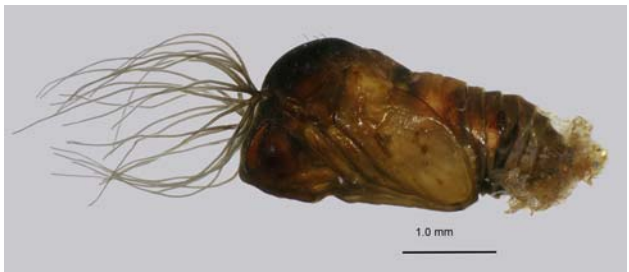


FIGURE 14.3 Pupa of the Australian black fly *Nothogreniera fergusoni*; the cocoon of this species is reduced to a small bit of silk at the posterior end. © *Douglas A. Craig.*

FIGURE 14.4 Female of the North American black fly *Prosimulium mixtum* feeding on a human. *Photograph by Stephen A. Marshall.*

FIGURE 14.5 Female of *Simulium vittatum*, one of the most common and widely distributed black flies in North America. © *Jena Johnson.*

FIGURE 14.6 Male of the North American black fly *Simulium vittatum*, showing the enlarged upper facets of the compound eye. © *Jena Johnson.*

typical-sized ventral facets, an arrangement that enhances the ability of males to locate females entering a mating swarm from overhead. Females are dichoptic, with smaller eyes separated by the frons.

The mouthparts arise ventrally from the head. A conspicuous pair of long maxillary palps attaches near the base of the proboscis. The third palpal segment accommodates the sensory vesicle (Lutz's organ), which has many chemosensilla that detect odors such as carbon dioxide. The labium forms the back of the proboscis and envelops the other mouthparts, including the minutely serrated mandibles and the toothed laciniae, with a pair of large, fleshy lobes called the labella. The mouthparts of the male are similar to those of the female, except the mandibles and laciniae are not adapted for blood-feeding and, therefore, do not bear teeth.

The stout thorax bears a pair of wings, either smoky or hyaline but never patterned. The venation, including the setation, is taxonomically important at the generic level. The color patterns of the legs and thoracic scutum are useful for species identification. The tarsal claws exist in one of three conditions. Species that feed on mammals have either a simple, unarmed claw or a minute tooth at the base of each claw. Bird feeders are endowed with a large thumblike lobe at the base of each claw. The abdomen is weakly sclerotized except the genitalia, which are of the utmost importance in the identification of species. To interpret the taxonomically important characters of the genitalia of both males and females, the abdomens must be treated with a clearing agent such as potassium hydroxide or hot lactic acid and examined in a depression slide with glycerin.

LIFE HISTORY

Immature black flies are found in virtually any water that flows, even if only imperceptibly and temporarily, from the smallest trickles to the largest rivers. Most species occupy specific habitats, and some higher taxa are characteristic of particular environments. For example, members of the genus *Gymnopais* occupy small, icy streams of the Far North, species of *Simulium* (subgenus *Trichodagmia*) live on the lips of waterfalls and in swift rocky flows, members of the *S. noelleri* sp. group are found below impounded waters, and species of *Simulium* (subgenus *Psilozia*) are found in warm, highly productive streams and rivers with open canopies.

Each species of black fly has a specific pattern of seasonal occurrence. Nearly all species in the tribe Prosimuliini are univoltine, completing a single generation annually. The tribe Simuliini contains both univoltine and multivoltine species. Some multivoltine species can complete seven or more generations per year in areas of North America with mild climates. In certain tropical areas of the world, some species (e.g., members of the *S. damnosum* complex) might cycle through more than 20 generations each year.

Eggs typically cannot resist desiccation, although those of some species (e.g., *Austrosimulium pestilens*) can survive in moist soil of dry streambeds for several years, hatching when streams are inundated. During the summer, eggs of multivoltine species (e.g., *S. vittatum*, *S. damnosum*) can hatch in fewer than 4 days. In northern temperate regions, univoltine species (e.g., *Prosimulium* spp.) often spend the warm months as eggs, whereas multivoltine species spend the cold months as eggs. Accordingly, the potential for long-term survival of eggs must be considered in management programs. Eggs of some species (e.g., *S. rostratum*) remain viable in the laboratory just above freezing for up to 2 years.

The larval stage lasts from about a week, or even less, to nearly half a year, depending on species, stream temperature, and food availability. At one extreme, the larvae of some species in the West African *S. damnosum* complex complete development in 4 days. At the other extreme, larvae of many univoltine, temperate species hatch in the fall, develop during the winter, and pupate in the spring. The number of larval instars varies from six to 11, depending on species and environmental conditions, such as food supply.

Final-instar larvae typically move to slower water before pupating in a silk cocoon that is spun on a substrate. Some species (e.g., *Prosimulium magnum*) pupate in masses, but most pupate individually. The duration of the pupal stage depends largely on temperature and species, lasting from several days to a few weeks. When the adult is ready to emerge, it expels air from its respiratory system, thus splitting the pupal cuticle along the dorsal eclosion line.

The newly emerged adult, partially covered in air, rises to the surface of the water with enough force to break the water-air interface. It then seeks a resting site, often streamside, to tan and harden. Adults generally live less than a month, during which time mating, sugar-feeding, host location, blood-feeding, and oviposition must be accomplished. Crosskey (1990) and Adler et al. (2004) provide detailed treatments of simuliid life history and bionomics.

BEHAVIOR AND ECOLOGY

After hatching, early instars often disperse short distances to more suitable sites for development. Larvae lead a largely sessile life attached to silk pads on substrates such as stones, trailing vegetation, sticks, aquatic plants, and leaf packs. The larvae of about 30 species, mostly in tropical Africa and Central Asia, are obligatorily phoretic, anchoring themselves to the bodies of larval mayflies and freshwater crabs and prawns. When disturbed, a larva repositions itself by looping over the substrate in inchworm-like fashion or by releasing itself from the silk

pad and drifting downstream, often on a lifeline of silk. Downstream drift is usually greatest around dusk and during the night; its extent and timing should be considered in management programs.

The majority of larval life is spent feeding, usually by passively filtering suspended matter from the current (Fig. 14.2A) or actively grazing adherent material from the substrate. The larvae of some species are also predaceous, consuming small invertebrates such as chironomid midges. Larvae that filter their food lean with the current and twist their bodies longitudinally 90−180 degrees. In this position, one labral fan receives particulate matter that is resuspended by vortices arising from the substrate, while the other fan receives material from the main flow. Larvae filter particles that are about 0.09−350 μm in diameter, with the majority of ingested particles less than 100 μm in diameter. Larval diet consists of detritus, bacteria, small invertebrates, larval fecal pellets, and algae, with gut contents largely reflecting particle size and composition of available material in the water. Feeding efficiency (i.e., the ability to remove particles from the water column) is low, typically less than 2%. Retention of material in the gut typically varies from 20 min to longer than 2 h, depending mainly on larval age, species, and water temperature. Where larvae achieve extraordinarily high densities, as in the boreal region, their fecal pellets provide significant nutrition for freshwater organisms and might even fertilize river margins, leading ecologists to refer to the larvae as "ecosystem engineers" (Malmqvist et al., 2004a).

Species composition in streams and the distribution patterns of larvae and pupae are associated with a variety of environmental factors, such as oviposition behavior and competition, and abiotic conditions such as water chemistry, substrate availability, and stream size (McCreadie and Adler, 2012). Distributions in a small section of stream or on a specific substrate are customarily referred to as microdistributions. Factors influencing microdistributions are those that vary over a few centimeters or meters, including substrate texture, water depth, hydrodynamics, and interactions with other organisms. Microdistributions can be species specific. For example, last instars of *Simulium truncatum* and *S. rostratum*, two morphologically similar, boreal species often found in the same section of stream, select different microhabitats on the basis of water velocity. The patterns of larval dispersion on a substrate are either spaced (e.g., *S. vittatum*), with a well-defined area surrounding each larva, or clumped (e.g., *S. noelleri*), with each larva occupying only enough space to attach its silk pad. Larvae with spaced patterns vigorously defend their space from other larval black flies.

Macrodistributions encompass a scale of many meters to hundreds of kilometers. The most important factors influencing macrodistributions are stream size, water velocity, temperature, water chemistry, food quality and quantity, and the presence of lake outlets. Within a stretch of stream, species distributions can be predicted by gradients of physical and chemical factors such as temperature and conductivity. Larval densities are usually greatest within a short distance downstream of impoundment outflows. For example, densities as high as 1.2 million larvae/m^2 have been recorded for *S. noelleri* at lake outlets in Europe. Some species rarely are found far from lake outlets and, therefore, species assemblages at these outflows are often distinct from those farther downstream (Adler and McCreadie, 1997). Distributions of species among streams can be predicted by factors such as stream width and elevation. For example, species in genera such as *Twinnia*, *Gymnopais*, *Greniera*, and *Stegopterna*, as well as many species of *Prosimulium* and *Simulium* occur in trickles and small streams. Species such as *Metacnephia lyra*, *Simulium jenningsi*, *S. reptans*, *S. vampirum*, and members of the genus *Ectemina* occupy large streams and rivers. The influence of stream size and impoundment outflows on the distribution of black flies is important throughout most of the world. We know little of the oviposition choices of female black flies, which are a primary determinant of larval distributions. At larger scales, biogeographic factors are useful in predicting species distributions. Streams in one ecoregion (e.g., mountains) tend to be more similar to one another than to streams in a different ecoregion (e.g., coastal plain), with respect to physical, chemical, and riparian characteristics. Simuliid faunas also show significant differences among ecoregions.

Species richness, the number of species in a specified location, varies from one to 13 for any given stream site, but is typically less than eight. At a larger scale, the mean number of species per stream reach in a given region is remarkably consistent, ranging from three to four species (McCreadie et al., 2005). Thus, even though the total number of species in regions varies across North and South America, the mean number of species per stream reach remains relatively invariant (McCreadie et al., 2017).

After emergence, adults of most species of black flies undertake short dispersal flights, usually less than 5 km. Males disperse to find mates and a source of sugar, whereas females of most species have the additional need to find hosts for blood and sites for oviposition. Although exceptions have been reported, black flies are diurnal fliers, generally taking to the wing when temperatures exceed 10°C. Local meteorological conditions can modify or even halt flight, but the primary factors that control daily flight patterns are wind, light, and temperature. Most species show a propensity to be on the wing at particular times of the day, these times varying with species, sex, physiological state, season, and nature of the activity.

Some species of black flies (e.g., *S. vampirum*) undertake far longer flights in search of hosts or breeding sites. These long-range movements are wind assisted, with the direction

of movement being controlled by prevailing winds. Movements of hundreds of kilometers by some species (e.g., members of the *S. damnosum* complex) have implications for control strategies, requiring that breeding grounds remote from problem areas be treated. Long-distance flights typically occur in species that feed on mammals, especially those that inhabit open areas such as savannas or prairies.

The universal energy source used by males and females for flight is sugar. Adults are opportunistic in their choice of carbohydrate sources, using floral or extrafloral nectar, plant sap, and honeydew from aphids and related insects (Burgin and Hunter, 1997). Water markedly increases longevity, and a 10% sugar solution further increases longevity. The sugar meal is stored in the crop and passed to the gut as needed for digestion.

Mating is necessary for all but about 10 parthenogenetic species (e.g., *Prosimulium ursinum*). These parthenogenetic species lack males and are triploid and northern in distribution. In the sexual species, mating occurs shortly after emergence. Males use a variety of strategies to encounter females. The most commonly reported method is the formation of precopulatory swarms. These aerial swarms usually form 2−3 m above ground, either beside or above a marker. Swarm markers tend to be visually apparent aspects of the environment such as a tree branch, rock, waterfall, or host. Females enter the swarm, sometimes immediately after emergence, and are seized by males. Coupled pairs fly out of the swarm or fall to the ground or lower vegetation. Some species (e.g., *S. decorum*) do not form swarms but instead couple on the ground during large, synchronous emergences. Males of some species also perch on vegetation and seize passing females. Visual cues mediate mating, but contact pheromones might also play an important role. Black flies generally are refractive to mating under laboratory conditions, which has impeded attempts to colonize most species and to elucidate details of mating behavior. The long-term successful colonization of *S. vittatum* is a notable exception (Gray and Noblet, 2014).

Copulation lasts from a few seconds (e.g., *S. vittatum*) to 2 h (e.g., *Gymnopais* spp.). During copulation, the male passes a spermathophore (i.e., a package of spermatozoa) to the female. The tip of the spermatophore is opened enzymatically by the female and sperm move into the female's storage structure, the spermatheca. Stored sperm are released to fertilize eggs as they are being deposited.

A bloodmeal is required for the females of more than 90% of the world's simuliid species to mature the eggs. Males do not feed on blood. Females of those species that never take blood have feeble, untoothed mouthparts unable to cut host skin. These females are obligatorily autogenous; that is, they are able to produce eggs without taking blood, relying instead on energy acquired during the larval stage for all of their egg production. Females of species with biting mouthparts are anautogenous (i.e., they mature their eggs with the aid of a bloodmeal). Nonetheless, the females of some species with biting mouthparts can mature the first batch of eggs without a bloodmeal (facultative autogeny) if conditions for larval growth have been optimal. In these facultatively autogenous species, however, each subsequent batch of eggs requires a bloodmeal. Each ovarian or gonotrophic cycle (i.e., maturation of an egg batch) varies from about 2 days to 2 weeks, depending on the species and ambient temperature. Most females probably do not survive long enough to complete more than two or three ovarian cycles. Because the transmission of pathogens is usually horizontal, passing from host to host via the simuliid vector, anautogenous females have a greater potential than facultatively autogenous females to acquire and transmit disease agents.

The majority of simuliid species in the world probably feed on mammals (mammalophily), although those that feed on birds (ornithophily) also are common; no other groups of organisms serve as hosts for black flies. About two-thirds of the blood-feeding species in North America are principally mammalophilic, and the other one-third are mainly ornithophilic (Adler et al., 2004). A number of these species (e.g., *S. johannseni*, *S. venustum*), however, feed on both mammals and birds. Molecular analyses of bloodmeals in wild-caught black flies have proved valuable in determining the hosts of different species of black flies (Malmqvist et al., 2004b). Host specificity varies from highly specific in species such as *Simulium annulus*, which feeds chiefly on loons and cranes, to those such as *S. rugglesi,* which have been recorded feeding on nearly 30 different host species. Most simuliid species attack thinly haired or sparsely feathered regions of the host body and areas that are difficult for the host to groom. Thus, mammals often are attacked along the ventral region of the body and inside the ears. Birds are attacked especially on the neck, bases of the legs, and around the eyes. Humans are bitten wherever flesh is exposed, although specific areas are often attacked, such as along the hairline (e.g., *S. venustum*), the arms and hands (e.g., *S. parnassum*), the upper torso (e.g., members of the *S. ochraceum* and *S. oyapockense* complexes), and the ankles and feet (e.g., members of the *S. damnosum* and *S. metallicum* complexes).

A number of host attractants have been identified. Carbon dioxide released from the host and color, shape, and size of the host provide some of the major cues and attractants that females use to locate an appropriate blood source (Sutcliffe, 1986). Traps used to monitor populations often exploit these cues. For example, sticky silhouettes and carbon dioxide in gaseous or dry-ice form often are used to monitor females.

Biting (Fig. 14.4) and engorging require a series of appropriate cues, especially temperature and various phagostimulants, such as adenosine phosphates, in the host's blood. When the fly begins to bite, the labella are

withdrawn, and small teeth and spines at the apex of the labrum and hypopharynx pull the host skin taut (Sutcliffe and McIver, 1984). The serrated mandibles cut the host flesh, allowing the labrum and hypopharynx to enter the wound, along with the laciniae, which are armed with backwardly directed teeth that anchor the mouthparts. Blood from the wound forms a small pool that is drawn up the food channel formed when the mandibles overlap the labral food canal. Because of their method of feeding from pooled blood, black flies are termed pool feeders or telmophages. Saliva is applied to the host flesh via a salivary groove along the anterior surface of the hypopharynx. Various salivary components promote local anesthesia, enhance vasodilation, inhibit platelet aggregation, and prevent clotting (Cupp and Cupp, 1997). Chemosensilla on the mouthparts help determine that blood will be directed to the midgut.

Female black flies are determined feeders. Once the host skin is penetrated, females typically do not leave until they are satiated. Because most black flies are not nervous, easily interrupted feeders, they make poor mechanical vectors of pathogens. Most species feed for about 3–6 min, taking approximately their own weight in host blood.

Most blood-feeding activity is restricted to outdoor settings (exophily), with females infrequently entering shelters to feed. This behavioral trend has implications for vector control. For example, residual house treatments effective for the control of mosquitoes are of no use for controlling black flies. Nonetheless, some cavity-nesting wild birds can experience severe assaults by ornithophilic black flies that enter through the cavity opening.

Biting activity occurs within certain optimal ranges of temperature, light intensity, wind speed, and humidity, with optima differing for each species. Given the appropriate range of meteorological conditions, many species bite throughout the day. Other species show a particular pattern of biting activity, such as a single peak in the morning (e.g., members of the *S. exiguum* complex) or a bimodal pattern with peaks in the morning and early evening (e.g., those in the *S. damnosum* complex). For all black flies, feeding typically is restricted to the hours of daylight and dusk. A rapid decrease in air pressure, combined with increased cloud cover, produces a sudden flush of biting activity.

Most female black flies can produce a batch of about 100–600 eggs, although the number varies from 25 (some *Gymnopais* spp.) to about 800 (some *Simulium* spp.). Females of some species can produce several of these egg batches in a lifetime, depending on the number of bloodmeals and how long the female lives. Oviposition usually occurs in the late afternoon and early evening. Eggs are deposited freely into the water during flight (e.g., *S. venustum* complex) or attached in strings or masses (Fig. 14.1) to substrates such as rocks and trailing vegetation at the water line (e.g., *S. vittatum*). Some species, however, oviposit in moist fissures in riverbanks (e.g., *S. posticatum*) or in streamside mosses (e.g., some *Prosimulium* spp.). Females of some species participate in communal oviposition, mediated by oviposition pheromones, resulting in masses of thousands of eggs (McGaha et al., 2015).

PUBLIC HEALTH IMPORTANCE

The importance of black flies to humans centers largely around the pestiferous habits of the blood-seeking females and the disease agents they transmit. The human disease agents transmitted by black flies are those that cause onchocerciasis in the tropics of Africa and Central and South America and mansonellosis in northwestern Argentina, southern Panama, and the western Amazon Region. No other human pathogens or parasites are known definitively to be transmitted by black flies, and no endemic simuliid-borne disease of humans has been reported from North America.

The biting and nuisance problems inflicted by black flies have had severe consequences for most outdoor activities including agriculture, forestry, industrial development, military exercises, mining, and tourism. Industrial and recreational development in some regions of Canada and Russia has been impeded or halted by overwhelming attacks from black flies. Yet, these negative effects gain balance through the protection that the attacking black flies afford to sensitive environmental areas that otherwise might suffer from development. Actual monetary losses due to biting and nuisance problems in different sectors of the economy, although significant and sometimes crippling (Sariözkan et al., 2014), are typically poorly documented.

Biting and Nuisance Problems

The black flies that bite humans (i.e., anthropophilic species) constitute 10% or less of the total simuliid fauna in any zoogeographic region (Table 14.1), with some areas of the world being nearly free of biting problems. No black fly is known that feeds exclusively on humans. In North America, where the name "black fly" originated, fewer than 60 species have been recorded to bite humans. Less than one-third of these hold any real status as biting pests, but those that bite regularly can be unrelenting in their attacks. Individual reactions to bites vary from a small red spot at the puncture site, often with initial streaks of oozing blood (Fig. 14.7) to an enlarged swelling the size of a golf ball (Stokes, 1914). Swelling from bites around the eyes can impede vision, and bites on the legs can impair walking.

A general syndrome, sometimes called **black fly fever**, is common in areas such as northeastern North America

TABLE 14.1 Species of Black Flies Regarded as Significant Biting and Nuisance Pests of Humans, Livestock, and Poultry

Species	Geographic Region
Humans	
Austrosimulium australense	New Zealand
Austrosimulium ungulatum	New Zealand
Prosimulium mixtum group	Eastern North America
Simulium amazonicum complex	South America (Amazon Basin)
Simulium arakawae	Japan
Simulium buissoni	Marquesas Islands
Simulium cholodkovskii	Russia
Simulium decimatum	Russia
Simulium jenningsi	Eastern North America
Simulium johannseni	Midwestern North America
Simulium jujuyense	Argentina
Simulium meridionale	Western North America
Simulium nigrogilvum	Thailand
Simulium ochraceum complex	Galapagos Islands
Simulium oyapokense complex	South America (Amazonian Region)
Simulium parnassum	Eastern North America
Simulium penobscotense	Northeastern North America
Simulium pertinax	Brazil
Simulium posticatum	England
Simulium quadrivittatum	Central America
Simulium sanguineum	Northwestern South America
Simulium tescorum	Southwestern United States
Simulium turgaicum	Western Asia
Simulium venustum complex	North America
Simulium vittatum complex	North America
Livestock	
Austrosimulium pestilens	Australia (Queensland)
Cnephia pecuarum	United States (Mississippi River Valley)
Simulium cholodkovskii	Russia
Simulium chutteri	South Africa
Simulium colombaschense	Europe (historical)
Simulium decimatum	Russia
Simulium equinum	Europe, Russia
Simulium erythrocephalum	Europe
Simulium incrustatum	Paraguay
Simulium jenningsi group	Eastern North America
Simulium kurense	Western Asia
Simulium lineatum	Europe
Simulium luggeri	Western Canada
Simulium maculatum	Russia
Simulium ochraceum complex	Galapagos Islands
Simulium ornatum complex	Europe, Russia
Simulium reptans	Europe, Russia
Simulium turgaicum	Russia, western Asia
Simulium vampirum	Western Canada
Simulium vittatum complex	North America
Poultry	
Cnephia ornithophilia	Eastern North America
Simulium meridionale	North America
Simulium rugglesi	North America
Simulium slossonae	Southeastern United States

where biting problems can be intense. It is presumably a response to salivary components from the fly, and is characterized by headache, nausea, fever, and swollen lymph nodes in the neck. Many people experience some itching from the bites, intensified by scratching the wounds. Severe allergic reactions, including asthmatic responses, are infrequent; however, medical treatment, including hospitalization, is sometimes necessary (Gudgel and Grauer, 1954). No human deaths from simuliid bites have been recorded since the beginning of the 20th century, although anecdotal accounts suggest that an unclothed human can be exsanguinated in about 2 h in some areas of Russia. Exposure to fierce attacks of biting and swarming black

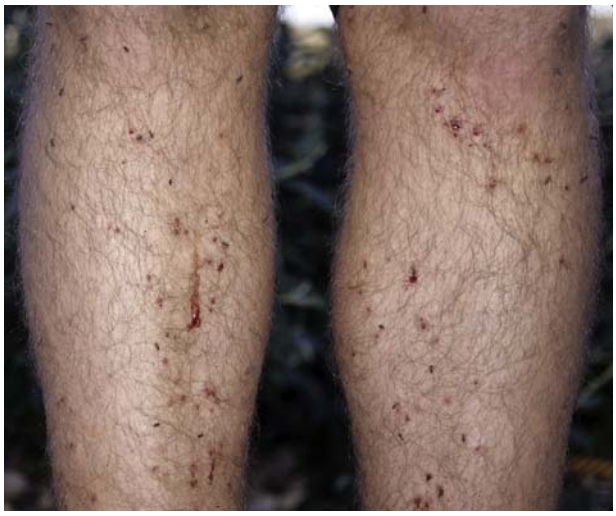


FIGURE 14.7 Bite wounds on legs of a river guide, caused by a North American black fly of the *Simulium venustum* complex. *Photograph by Peter. H. Adler.*

FIGURE 14.8 Overwhelming swarm of blood-seeking flies along Nunavut's Dubawnt River in the Canadian Arctic. *Photograph by Nicolas Perrault; Creative Commons CC0 1.0 Universal Public Domain Dedication.*

flies (Fig. 14.8) can affect a person's emotional state and produce short-term psychological effects that reduce individual efficiency.

Many species of black flies are attracted to humans but do not bite, or they bite infrequently in proportion to the number of flies actually attracted. These species can create enormous nuisance problems. One such species is *Simulium jenningsi*, a major pest in North America. Females of this species sometimes bite humans and occasionally cause allergic reactions, but they are more of a nuisance because of their habit of swarming about the head and entering the eyes, ears, nose, and mouth. Outdoor activities in afflicted areas, such as Pennsylvania (USA), can become unbearable as the females ceaselessly swarm around the head. More than $5 million is spent annually in the management of *S. jenningsi*.

Occasional nuisance problems have been caused by large numbers of flies attracted to incandescent lights and by mating swarms that form over bicycle and foot paths at about the same height as a person walking or riding a bicycle. These kinds of problems usually are caused by members of the North American *S. vittatum* species complex, which breed abundantly in human-altered habitats, such as lake outlets and polluted waters.

Human Onchocerciasis

The greatest public health problem associated with black flies is onchocerciasis, or **river blindness**, a tropical disease caused by the filarial nematode *Onchocerca volvulus*, which is transmitted solely by black flies during blood-feeding. River blindness is the second leading infectious cause of blindness in the world. In the Old World, river blindness is found in 27 countries in the central belt of Africa, with small foci in southern Yemen (Fig. 14.9). In the New World, where the disease possibly was introduced during the slave trade, its current and historical distributions are patchy, with foci in northern Brazil, Colombia, Ecuador, Guatemala, Mexico, and Venezuela (Fig. 14.10). The World Health Organization (1995) conservatively estimated that about 17.7 million people are infected (17.5

FIGURE 14.9 Geographic distribution of human onchocerciasis in Africa and Yemen. *Compiled from various sources.*

FIGURE 14.10 Geographic distribution of human onchocerciasis in the New World, showing foci where the disease has been eliminated and where it is ongoing. *Modification of map; The Carter Center, Atlanta, Georgia, USA.*

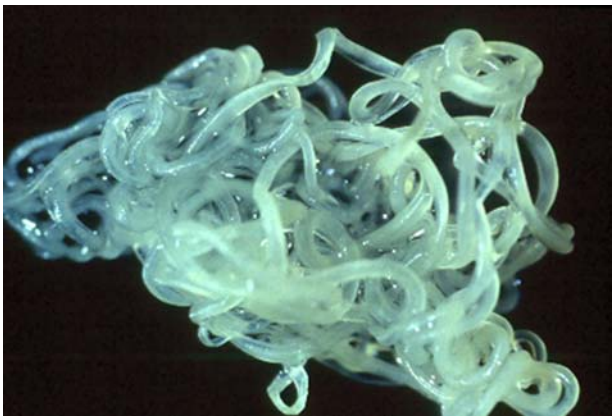


FIGURE 14.11 *Onchocerca volvulus*, after digesting away the nodular human tissue. *Courtesy of Armed Forces Institute of Pathology, USA.*

FIGURE 14.12 Children at a clinic in Guatemala for surgical removal of adults of *Onchocerca volvulus* from subcutaneous nodules on their heads, either waiting (scalps shaved) or returning to be checked following previous surgery (heads bandaged). *Photograph by E. W. Cupp.*

million in Africa and Yemen; 140,500 in tropical America), with approximately 270,000 cases of blindness and another half million individuals with severe visual impairment. Subsequent evidence suggested that the 1995 figures were underestimates and should have been 120 million people at risk, with 37 million heavily infected (Remme et al., 2006). Onchocerciasis is occasionally diagnosed in patients in North America who have traveled from endemic areas. Research on the disease and its vectors has generated a massive body of literature (Muller and Horsburgh, 1987) and numerous reviews (Shelley, 1988b; Crosskey, 1990; World Health Organization, 1995).

Onchocerca volvulus typically is found only in humans (definitive host) and adult flies of the genus *Simulium* (intermediate host). Various strains of *O. volvulus* are recognized, such as forest and savanna strains in West Africa, and these form highly compatible parasite–vector complexes with distinct clinical features. When the female black fly ingests a bloodmeal from an infected human, the **microfilariae** (220–360 μm long) penetrate the gut of the fly and make their way to the thoracic flight muscles. Once in the thoracic muscles, the microfilariae lose their motility and transform to first-stage larvae (L1), which then molt to become second-stage larvae (L2). The final molt in the fly produces the infective third-stage larvae (L3), which migrate to the fly's head and mouthparts. Vector incrimination is based on the presence of L3 worms in the head capsules of female black flies. In West Africa, DNA tests allow animal parasites and the human parasites of savanna and forest to be distinguished. Development in the black fly, which is influenced by ambient temperature, typically requires 6–12 days, but the time between successive bloodmeals taken by the fly is usually 3–5 days. Consequently, the infective larvae will be passed to a human host no earlier than the third bloodmeal when the fly is about 8–10 days old.

In humans, the infective larvae molt to the L4 stage within about a week. One more molt yields juvenile adults, which grow to mature adult worms over the next 12–18 months and begin reproducing. Adult worms (Fig. 14.11) typically become encapsulated in fibrous nodules (onchocercomas) that vary in size from about 0.5 to 10.0 cm and can be subcutaneous over bony prominences (Fig. 14.12) or deep in muscular and connective tissues; they cause no inflammatory response and no great discomfort. Mating between the small male worms (3–5 cm long) and the large females (30–80 cm) occurs in the nodules. Adult female worms can produce microfilariae, potentially millions of them, for up to 14 years. These microfilariae migrate from the nodules to the skin, where they can be acquired by a vector, as well as to the eyes and various other organs (e.g., liver) of the human host. A diagnostic clinical feature of onchocerciasis is the presence of hundreds of microfilariae in skin snips.

River blindness is essentially a rural disease, afflicting those people most vulnerable to both the medical consequences and social stigmas of infection. Symptoms of the

disease depend on factors such as geographical location, microfilarial transmission rates, and frequency of reinfection. Where transmission rates are low, the disease can be asymptomatic. With heavy infections, however, the classical manifestations of the disease appear (i.e., dermal changes, lymphatic reactions, nodules [Fig. 14.12], and ocular disturbances). Other than the nodules in which the adults are enveloped, all symptoms are caused by the microfilariae.

Large numbers of microfilariae migrating throughout the dermis cause horrific itching that can lead to bleeding, secondary bacterial infections, inability to sleep, fever, headache, and even suicide. In addition, to itching, chronic infections in Africa and Yemen can cause dermal lesions, patches of depigmentation ("leopard skin"), fibrosis, loss of elasticity (e.g., "elephant knees"), and dry wrinkling ("lizard skin"). In Yemen, the itching symptoms of the disease, which can result in dark, swollen skin, are known as *aswad* or *sowda*. In Central America, two unique, chronic skin conditions occur: a painful, reddish rash on the face (*erisipela de la costa*) and lesions associated with reddish skin on the trunk and arms (*mal morado*). The lymphatic nodes also can be affected, especially in the groin and thighs; combined with loss of skin elasticity, the result is a condition known as hanging groin. The various skin conditions associated with onchocerciasis can create a social stigma that hinders treatment and exacerbates patient suffering (Tchounkeu et al., 2012).

Migrating microfilariae also enter the eye, resulting in a severe ocular pathology that can involve all tissues of the eye. The discovery of the co-involvement of the symbiotic bacterium *Wolbachia* and its filarial host, *O. volvulus*, in ocular pathology (Pearlman, 2003) opened the possibility of reducing ocular onchocerciasis through antibiotic treatments (Taylor et al., 2014). Ocular problems manifest in many forms, including cataracts, retinal hemorrhages, corneal opacities, secondary glaucoma, sclerosing keratitis, and optic neuritis. Various forms of visual impairment occur, such as night blindness and reduction in peripheral vision, but the most severe consequence is irreversible blindness with complete loss of light perception (Fig. 14.13). Blindness usually takes years to occur; at age 20, for example, it is rare in infected people, but at 50 years of age, half of the infected victims can be blind (Fig. 14.14). The incidence of blindness is highest in the savannas of West Africa, with 15% of some villages experiencing blindness. At these high levels of disease, the village is often abandoned. Outside West Africa, ocular pathology is rare.

At least 26 species of *Simulium* are known vectors of *O. volvulus* (Table 14.2). Most of these vectors are members of species complexes, and considerable taxonomic work is still needed to resolve all of the vector species in areas such as East Africa and the Americas. In West Africa and Yemen, all vectors are members of the *S. damnosum* species complex, and include *S. damnosum sensu stricto* and *S. sirbanum*, the principal vectors associated with the savanna form of the disease and ocular pathology. The vectors in East Africa are members of the *S. damnosum* complex, the *S. neavei* group, and *S. albivirgulatum*. In the

FIGURE 14.13 Human blindness caused by the filarial nematode *Onchocerca volvulus* transmitted by black flies of the *Simulium damnosum* complex in West Africa; note opacity of corneas. © *Eric Poggenpohl.*

FIGURE 14.14 Young boy leading a man blinded by onchocerciasis in Burkina Faso. *Photograph by E. W. Cupp.*

TABLE 14.2 Disease Agents Transmitted by Black Flies

Disease Agent	Vectors[1]	Hosts	Geographic Areas	Select References
Protozoa[2]				
Leucocytozoon cambournaci	*Helodon decemarticulatus, Cnephia ornithophilia, Simulium aureum* complex, *S. vernum* group	Sparrows	North America	Adler et al., 2004
Leucocytozoon dubreuili	*H. decemarticulatus, C. ornithophilia, S. aureum* complex, *S. vernum* group	Thrushes	North America	Adler et al., 2004
Leucocytozoon icteris	*H. decemarticulatus, C. ornithophilia, S. anatinum, S. annulus, S. aureum* complex	Blackbirds	North America	Adler et al., 2004
Leucocytozoon lovati	*S. aureum* complex, *S. vernum* group	Grouse	North America	Adler et al., 2004
Leucocytozoon neavei	*Simulium* spp., especially *S. adersi*	Guinea fowl	Eastern Africa	Fallis et al., 1974
Leucocytozoon sakharoffi	*H. decemarticulatus, S. aureum* complex, *S. angustitarse*	Corvids	North America, England	Adler et al., 2004 Fallis et al., 1974
Leucocytozoon schoutedeni	*Simulium* spp., especially *S. adersi*	Chickens	Eastern Africa	Fallis et al., 1973
Leucocytozoon simondi	*Cnephia ornithophilia, S. anatinum, S. fallisi, S. rendalense, S. rugglesi, S. usovae, S. venustum* complex	Ducks, geese	North America, Norway	Adler et al., 2004
Leucocytozoon smithi	*S. aureum* complex, *S. congareenarum, S. jenningsi* group, *S. meridionale, S. slossonae*, possibly *S. ruficorne* group	Turkeys	North America, introduced to Africa	Adler et al., 2004
Leucocytozoon tawaki	*Austrosimulium ungulatum*	Penguins	New Zealand	Allison et al., 1978
Leucocytozoon toddi	*H. decemarticulatus, S. aureum* complex, *S. vernum* group	Hawks	North America	Adler et al., 2004
Leucocytozoon ziemanni	*H. decemarticulatus, S. aureum* complex, *S. vernum* group	Owls	North America	Adler et al., 2004
Trypanosoma avium	*Metacnephia lyra, S. aureum* complex, *S. latipes, S. vernum*	Grouse, raptors	Europe	Votýpka et al., 2002; Reeves et al., 2007
Trypanosoma confusum	*H. decemarticulatus, Simulium* spp.	Birds	North America	Bennett, 1961
Trypanosoma corvi	*S. latipes*	Kestrels	England	Dirie et al., 1990
Trypanosoma numidae	*Simulium* spp., especially *S. adersi*	Chickens, guinea fowl	Eastern Africa	Fallis et al., 1973
Zelonia australiensis	*S. dycei*	Macropods	Australia	Barratt et al., 2017
Filarial Nematodes				
Dirofilaria ursi	*S. venustum* complex	Bears	North America	Addison, 1980
Mansonella ozzardi	*S. amazonicum* complex, *S. argentiscutum*, possibly *S. exiguum* complex, *S. oyapockense* complex, *S. sanguineum*	Humans	Northern South America, northwestern Argentina, Panama	Shelley, 1988a; Shelley and Coscarón, 2001

Continued

TABLE 14.2 Disease Agents Transmitted by Black Flies—cont'd

Disease Agent	Vectors[1]	Hosts	Geographic Areas	Select References
Onchocerca cervipedis	*Prosimulium impostor, S. decorum, S. venustum* complex	Deer, moose	North America	Pledger et al., 1980
Onchocerca dewittei	*S. bidentatum*	Wild boar	Japan	Uni et al., 2015
Onchocerca dukei	*S. bovis*	Cattle	Africa	Wahl and Renz, 1991
Onchocerca gutturosa	*S. erythrocephalum, S. bidentatum*	Cattle	Japan, Ukraine	Crosskey, 1990; Takaoka, 1999
Onchocerca lienalis	*S. erythrocephalum, S. jenningsi, S. ornatum* complex, *S. reptans, S. arakawae, S. daisense, S. kyushuense*	Cattle	North America, Russia, western Europe, Japan	Lok et al., 1983; Crosskey, 1990; Takaoka, 1999
Onchocerca lupi	*S. vittatum*	Dogs	Southwestern North America	Hassan et al., 2015
Onchocerca ochengi	*S. damnosum* complex	Cattle	West Africa	Wahl et al., 1998
Onchocerca ramachandrini	*S. damnosum* complex	Wart hogs	West Africa	Wahl, 1996
Onchocerca skrjabini	*S. arakawae, S. bidentatum, S. daisense, S. oitanum*	Japanese deer, serows	Japan	Takaoka, 1999
Onchocerca takaokai	*S. bidentatum*	Wild boar	Japan	Uni et al., 2015
Onchocerca tarsicola	*P. tomosvaryi, S. ornatum* complex	Deer, reindeer	Western Europe	Crosskey, 1990
Onchocerca volvulus	Africa: *S. albivirgulatum, S. damnosum, S. dieguerense, S. ethiopiense, S. kilibanum, S. konkourense, S. leonense, S. mengense, S. neavei, S. sanctipauli, S. soubrense, S. squamosum, S. thyolense, S. woodi, S. yahense,* Americas: *S. callidum, S. exiguum* complex, *S. guianense* complex, *S. incrustatum, S. limbatum, S. metallicum* complex, *S. ochraceum* complex, *S. oyapockense* complex, *S. quadrivittatum,* Yemen: *S. rasyani*	Humans	Africa, Central America, South America	Shelley, 1988b; Crosskey, 1990; World Health Organization, 1995; Post et al., 2008
Splendidofilaria fallisensis	*S. anatinum, S. rugglesi*	Ducks	North America	Anderson, 1968
Viruses				
Vesicular stomatitis virus	*S. notatum, S. vittatum*	Cattle, horses, pigs; occasionally goats, llamas, sheep	Americas	

[1]*Vector species also have the potential to be pests, depending on host specificity and population size.*
[2]*Many* Leucocytozoon *lineages have been found in female black flies, but transmission has not been demonstrated (Murdock et al., 2015; Lotta et al., 2016).*

Americas, at least nine species have been incriminated as vectors, the most important of which are members of the *S. exiguum*, *S. guianense*, *S. metallicum*, *S. ochraceum*, and *S. oyapockense* complexes; their importance varies with location and the eradication status of the disease.

An understanding of the unique life history and behavior of each vector species is key to the control of onchocerciasis. Breeding sites of the vectors represent the ideal targets for control. The immature stages of the *S. damnosum* complex primarily inhabit swift sections of medium to large rivers, from dry savannas to forest highlands, depending on the species. Larvae and pupae of species in the *S. neavei* group live primarily in perennial, shaded forest streams where they have an obligatory phoretic relationship with river crabs. In Latin America, members of the *S. metallicum* and *S. ochraceum* complexes breed in large and small streams, respectively, that drain forested mountain slopes, whereas members of the *S. exiguum* and *S. oyapockense* complexes breed in large rivers of the rain forest, and members of the *S. guianense* complex inhabit the rocky shoals of large rivers.

Although each species breeds in a specific habitat, the adults of some species can travel great distances beyond their natal waterways. The adults of *S. sirbanum* and *S. damnosum sensu stricto*, for example, can travel more than 500 km, assisted by seasonally changing winds. In the wet season, moist monsoon winds from the southwest move flies in a northeastwardly direction. In the dry season, winds from the northeast assist flies in their reverse, southwestwardly flights. Continual reinvasions by vectors, therefore, occur with each season in both the northern and southern parts of West Africa, and must be considered in control efforts.

Mansonellosis

The filarial nematode *Mansonella ozzardi* is the causal agent of mansonellosis, a questionably pathogenic disease of humans. It is transmitted by at least five species of black flies in the Neotropical rain forests of Brazil, Colombia, Guyana, Venezuela, and southern Panama, as well as in northwestern Argentina (Table 14.2). The disease is now spreading into previously uninfected areas (Adami et al., 2014). Black flies first were incriminated as vectors of *M. ozzardi* in 1959 and subsequently confirmed experimentally as vectors in 1980. Mansonellosis also is found in the Caribbean Islands, where only ceratopogonid midges (*Culicoides* spp.) are known to transmit the causal agent (Shelley, 1988a).

Adult nematodes of *M. ozzardi* occur in the subcutaneous tissues of humans, and the microfilariae are found principally in the peripheral blood where they are acquired by blood-feeding flies. The life cycle of the nematode in black flies is similar to that of *O. volvulus*. A number of mammals and some birds and amphibians can be infected, but humans are the only significant reservoirs. In some highly endemic areas (e.g., Colombia Amazon), up to 85% of the human population can be infected. Mansonellosis generally is viewed as causing little or no pathology, but some reports have indicated that joint pains, headaches, hives, and pulmonary symptoms are associated with infections (Adami et al., 2014). Treatment with ivermectin can reduce microfilaremia. The discovery of *Wolbachia* bacteria in *M. ozzardi* (Casiraghi et al., 2001) opens the possibility for antibiotic treatment.

Other Diseases Related to Black Flies

Because black flies that feed on humans also feed on other hosts, the potential exists for certain disease-causing agents of domestic and wild animals to be transferred to humans. As examples, about 16 cases of zoonotic onchocerciasis are known in humans (Fukuda et al., 2011). Black flies also have been implicated as mechanical vectors of the bacterial agent of **tularemia** in the United States and Russia, suggesting that occasional cases of transmission of this pathogen to humans might occur. Similarly, **Eastern equine encephalitis** virus in the United States and **Venezuelan equine encephalitis** virus in Colombia have been isolated from several species of black flies, suggesting at least the potential for transmission of these pathogens to humans. Black flies in the Marquesas Islands have been implicated in the indirect transmission of **Hepatitis B** virus by causing numerous, itching lesions on the skin (Chanteau et al., 1993). Direct transmission of the virus by black flies also is theoretically possible.

Several additional diseases might be related to biting black flies. One such disease is **endemic pemphigus foliaceus** or **fogo selvagem**, a potentially lethal, autoimmune, blistering skin affliction. The disease is centered among poor, outdoor laborers in certain regions of Brazil (Eaton et al., 1998). Further work is needed to determine if black flies are the causal agents of the disease. Another affliction possibly associated with black flies in the New World is **thrombocytopenic purpura**, a disorder in which the platelet count is reduced. Again, more data are needed before black flies can be linked to the cause. A mysterious disease with a possible link to black flies is **nodding syndrome**, characterized by epileptic seizures in children of East Africa. Nodding syndrome has been known since the 1960s, but its incidence has increased in the past 10 years and is strongly associated with infection by the simuliid-borne filarial nematode *O. volvulus*, suggesting an autoimmune response (Johnson et al., 2017).

VETERINARY IMPORTANCE

The veterinary importance of black flies is manifested through pathogen transmission, biting, and nuisance swarming. Filarial nematodes, protozoans, and possibly several viruses are transmitted to animals. The most insidious parasites are those that cause leucocytozoonosis in domestic ducks, geese, and turkeys.

Deaths of birds and livestock have resulted from attacks by large numbers of black flies. Livestock under persistent attack sometimes stampede, trampling young animals, crashing into structures, and tumbling from precipices. Suffocation has been blamed for some deaths, with so many flies clogging the respiratory passages that breathing can become severely impaired. Deaths also have been attributed to respiratory tract infections caused by inhalation of flies. If enough blood is withdrawn, it may become too thick to transport oxygen efficiently, thereby killing the animal via **exsanguination**. Perhaps the most common cause of mortality can be attributed to the actual bites of the flies or, more specifically, to toxemia and acute shock caused by the various salivary components that are injected during blood-feeding.

More difficult to assess in economic terms, but equally harmful to the livestock and poultry industries, are the effects of harassment through biting and swarming (Fig. 14.15, Table 14.1). Biting is often aimed at weakly protected areas of the body, such as the ears, neck, and ventral midline (Fig. 14.16). Persistent attacks by black flies can cause unruly host behavior, weight loss, reduced egg and milk production, malnutrition in young animals, dermatitis and epidermal necrosis, impotence in bulls, delayed pregnancies, abortions, and possibly stress-related diseases such as pneumonia.

Actual monetary losses from black flies are not well documented but can be significant. The beef and dairy industries of Saskatchewan, for example, lost more than $3 million in 1978 from attacks by *Simulium luggeri* (Fredeen, 1985). In spring 1993, the ostrich and emu industry lost about $1.5 million along the Trinity River in eastern Texas as a result of attacks by black flies (Sanford et al., 1993). Effects of black flies on pets have rarely been documented, although *Cnephia pecuarum* has caused hospitalizations and deaths as recently as the end of the 20th century (Atwood, 1996). The death of even a single exotic bird, such as a parrot, from blood-feeding by black flies can reflect a loss of thousands of dollars (Mock and Adler, 2002).

Wildlife also succumb to withering attacks from black flies (Fig. 14.17), especially when the animals are stressed, as in years of lean food supply. Nestling birds are particularly vulnerable, including raptors and songbirds. Nestling bluebirds and tree swallows, for example, have been killed by *S. meridionale*, a species that routinely enters nest boxes

FIGURE 14.15 Cattle under attack by *Simulium vampirum* on the prairie of Alberta, Canada. *Photograph by Joseph A. Shemanchuk, Department of Agriculture and Agri-Food, Government of Canada.*

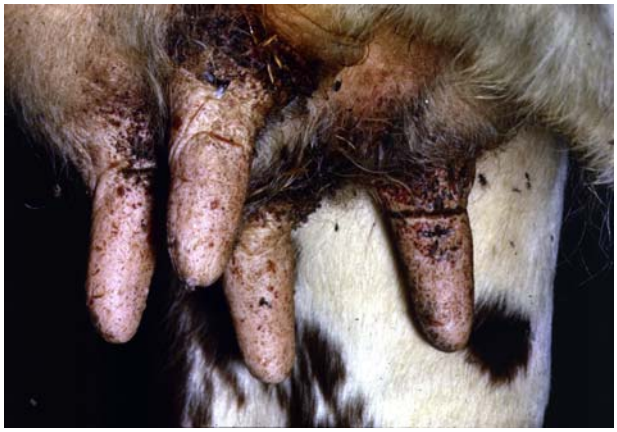


FIGURE 14.16 Damage to cow udder caused by black flies on the Canadian prairie. *Photograph by Joseph A. Shemanchuk, Department of Agriculture and Agri-Food, Government of Canada.*

FIGURE 14.17 Endangered whooping crane attacked by *Simulium annulus* in Wisconsin. *Photograph by Richard P. Urbanek, U.S. Fish and Wildlife Service.*

FIGURE 14.18 *Simulium annulus* and *Simulium johannseni* attracted to eggs of the whooping crane by odors from the bird's uropygial gland. *Photograph by Richard P. Urbanek, U.S. Fish and Wildlife Service.*

(Adler et al., 2004). Attacks on endangered species are of particular concern. The endangered Attwater's prairie chicken suffers attacks from the black fly *Cnephia ornithophilia*, which also carries a *Leucocytozoon* blood parasite (Adler et al., 2007). Endangered whooping cranes consistently abandon their nests during severe attacks from *Simulium annulus* and *Simulium johannseni* (Urbanek et al., 2010) (Figs. 14.17 and 14.18).

An odd, but indirect, nuisance problem mediated occasionally by black flies involves the Neotropical human bot fly *Dermatobia hominis*. Female bot flies capture hematophagous arthropods, including black flies, to which they glue their eggs. Once the carrier has landed on a host, the larvae of the bot fly hatch and bore into the host skin, causing myiasis. At least one species of black fly (*Simulium nigrimanum*) that feeds on domestic animals is known to be used as a carrier.

Bovine Onchocerciasis

At least 11 species of filarial nematodes in the genus *Onchocerca* are transmitted by simuliids to domesticated and wild animals (Table 14.2). Black flies transmit at least four species of filarial nematodes (genus *Onchocerca*) to cattle in the Afrotropical, Nearctic, and Palearctic Regions (Table 14.2). *Onchocerca lienalis* is the most widespread of these filarial parasites. *Simulium jenningsi* is its primary vector in the United States, whereas the *S. ornatum* complex is a principal vector in the Old World. The microfilariae of *O. lienalis* are concentrated in the umbilical region of the host. They are ingested during blood-feeding and transmitted to a new host after they have developed to the infective third stage in the simuliid vector. The percentage of infected cattle is often quite high, but symptoms and general effect on the host are usually not overt. Infected animals sometimes show dermatitis and inflammation of the skin and connective ligament.

A Palearctic species often confused with *O. lienalis* is *Ochocerca gutturosa*. Its microfilariae occur in the skin of the neck and back of the host. It has been confirmed from Japan and perhaps the Ukraine, where *S. bidentatum* and *S. erythrocephalum*, respectively, have been implicated in its transmission. Elsewhere, ceratopogonid midges (*Culicoides* spp.) are vectors. In West Africa, *O. ochengi* is transmitted to cattle by members of the *S. damnosum* complex, and *O. dukei* is transmitted by *S. bovis*. Both of these *Onchocerca* spp. can create nodules, either dermal (*O. ochengi*) or subcutaneous (*O. dukei*), in the inguinal region of the host. Economic losses resulting from bovine onchocerciasis rarely have been assessed, although a few reports have indicated that the quality of hides can be reduced.

At least four to seven additional species of *Onchocerca* are transmitted by black flies to nonbovine hosts (Table 14.2). *Onchocerca cervipedis* in North America and *O. tarsicola* in Europe infect the subcutaneous connective tissues, mainly in the legs, of deer, moose, and reindeer; consequently, they are sometimes called **legworms**. More than 60% of a host population can be infected. *Onchocerca ramachandrini* is a parasite of warthogs and is transmitted by members of the *S. damnosum* complex in West Africa. *Onchocerca lupi* parasitizes dogs in the southwestern United States. *Onchocerca skrjabini* parasitizes Japanese deer, and *Onchocerca takaoka* parasitizes wild boars in Japan.

Leucocytozoonosis

Species of protozoans in the genus *Leucocytozoon* are transmitted to birds by black flies, causing a malaria-like disease termed leucocytozoonosis (Table 14.2). More than 100 species of *Leucocytozoon* have been described, but the vectors—presumed to be primarily simuliids—have been demonstrated for only a fraction of these species. Molecular work has uncovered additional diversity of *Leucocytozoon* parasites in black flies (Murdock et al., 2015; Lotta et al., 2016). Most bird species and ornithophilic simuliid species are probably hosts of *Leucocytozoon* spp. The disease is known colloquially as **turkey malaria**, **duck malaria**, or **gnat fever**. The taxonomy of the genus *Leucocytozoon* is being revised using molecular techniques, and the parasite−vector−bird associations are expected to be reworked significantly. Two species of the parasite are of major economic concern, and both occur in North America. *Leucocytozoon simondi* is specific to ducks and geese, and its primary vectors are *S. anatinum* and *S. rugglesi*. *Leucocytozoon smithi* is specific to turkeys and is transmitted primarily by *S. meridionale* and *S. slossonae*.

Leucocytozoon species undergo a complex malaria-like life cycle. Gametocytes in the blood of an avian host are acquired by a female black fly. The parasite then undergoes

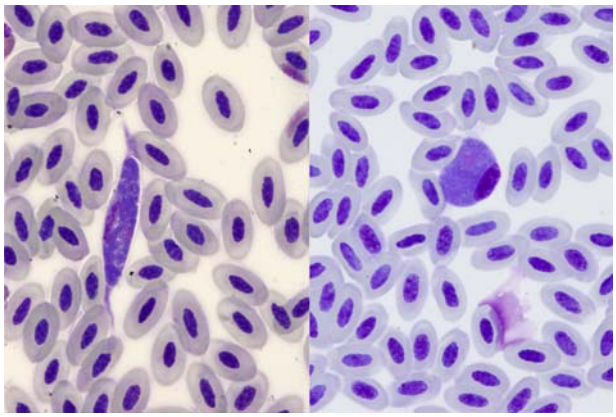


FIGURE 14.19 Mature macrogametocytes of *Leucocytozoon* cf. *toddi* from a buzzard (*Buteo buteo*), showing remarkable polymorphism. *Photograph by Jan Votýpka.*

both asexual and sexual development over a period of 3–4 days in the fly. During a subsequent bloodmeal, the fly transmits the parasites, as sporozoites, to another bird, which serves as a host for asexual development and gametocyte production (Fig. 14.19).

Leucocytozoonosis can be fatal in poultry, but its effects on wild hosts, with the exception of some populations of Canada geese (Herman et al., 1975), generally are less apparent or difficult to separate from the effects of blood-feeding (Rohner et al., 2000). Birds with chronic infections have weakened immune systems and reduced reproduction. Severe infections produce emaciation, dehydration, and convulsions that lead to death. Internally, the liver and spleen of moribund hosts are enlarged, the heart muscle is pale, and the lungs are congested.

The disease had devastating effects on the poultry industry throughout much of North America when birds were held in outdoor arenas (Noblet et al., 1975). Entire flocks were killed and production facilities shut down in areas such as Nebraska, South Carolina, and Manitoba. The U.S. Agricultural Research Service estimated an annual average loss of nearly $750,000 in the United States from 1942 to 1951 as a result of leucocytozoonosis in domestic turkeys. The last major outbreaks of the disease in domestic turkeys were in the 1970s. Turkeys now are raised primarily in poultry houses, reducing the incidence of disease because the vectors generally do not venture inside shelters.

Other Parasites and Pathogens of Veterinary Importance

Black flies transmit additional parasites to wild animals (Table 14.2). The protozoan *Trypanosoma confusum* is specific to birds in North America and is transmitted when infected fecal droplets from the black fly contaminate the bite. Birds of numerous families serve as hosts. Other species of bird trypanosomes (e.g., *Trypanosoma corvi*) are believed to cause infections when the birds consume infected black flies or eat other birds that have been infected (Votýpka and Svobodová, 2004). The filarial nematodes *Splendidofilaria fallisensis* and *Dirofilaria ursi* are transmitted to ducks and black bears, respectively. The effects of these protozoan and filarial parasites on their wild hosts are poorly known.

Several North American species of simuliids, such as *S. notatum* and *S. vittatum*, serve as biological and mechanical vectors of vesicular stomatitis virus to livestock, primarily cattle, horses, and pigs (Smith et al., 2009). The virus causes lesions in various epithelial tissues, especially in the mouth. Millions of dollars can be lost during epizootics. Laboratory experiments have shown that a viremic host is not necessary for a female black fly to become infected; flies can become infected by feeding on the same host with an infected black fly (Mead et al., 2000).

Additional parasites of wildlife have been associated with black flies. Minute nematodes of the family Robertdollfusidae in the guts of African black flies might be transmitted to wildlife (Bain and Renz, 1993). **Bunyaviruses**, **Eastern equine encephalitis virus**, and **snowshoe hare virus** have been isolated from several North American black flies. Minimal mechanical transmission has been demonstrated for **Whataroa virus** in laboratory mice in New Zealand (Austin, 1967) and for **myxomatosis** in rabbits in Australia (Mykytowycz, 1957). These examples suggest that much is yet to be learned about the vector potential of black flies among wildlife. *Chlamydia* infections in sheep and **Rift Valley fever virus** are suspected of being simuliid-borne in South Africa (Palmer, 1995).

Simuliotoxicosis

Attacks by black flies have, at times, been so massive and virulent that livestock have been killed. Many of the deaths probably result from acute toxemia and anaphylactic shock caused by toxins introduced with the saliva as black flies are feeding. The diseased condition, either temporary or terminal, that results from the bites of black flies is known as simuliotoxicosis, a term first used to describe the toxic effects of simuliid bites on reindeer (Wilhelm et al., 1982). Cattle, especially calves, are vulnerable to simuliotoxicosis, but goats, horses, mules, pigs, and sheep also have been affected. Susceptible animals succumb in less than 2 h. Some immunity is apparent in animals living in afflicted areas. The biochemical nature of simuliotoxicosis requires more investigation.

Most of the species responsible for simuliotoxicosis breed in large rivers from which the adults emerge in astronomical numbers. They include *Austrosimulium pestilens* in Queensland (Australia), *Cnephia pecuarum* in the Mississippi River Valley (USA), *S. colombaschense* along

the Danube River in central Europe, *S. vampirum* on the Canadian prairies, and *S. erythrocephalum*, the *S. ornatum* complex, and *S. reptans* in central Europe.

One of the worst attacks in recorded history killed about 22,000 animals in 1923 along Europe's Danube River in the southern Carpathian Mountains (Ciurea and Dinulescu, 1924). Prodigious attacks in this region during the 1700s prompted Empress Maria Theresa of the old Austro-Hungarian Empire to order one of the first biological studies of black flies, which eventually was published in 1795. On the Canadian prairies, thousands of livestock were killed from about 1886 into the 1970s by *S. vampirum*, a member of the *S. arcticum* complex (Fredeen, 1977). Massive mortality due to attacks by *Cnephia pecuarum* occurred in the United States during and immediately after the Civil War when the levees of the Mississippi River deteriorated, allowing the river to overflow and create extensive breeding areas for this species (Riley, 1887).

Simuliotoxicosis on a large scale is now rare, mainly because the former breeding sites of most of the responsible species have been altered by pollution, impoundment, and land development. Some of these species, however, still create nuisance problems for livestock, and occasional outbreaks cause deaths in localized areas of their ranges (Werner and Adler, 2005).

PREVENTION AND CONTROL

Management of black flies typically is aimed at the larval stage, in large part because in this life stage the pest species are concentrated in easily identifiable, specific habitats. Although adulticiding has sometimes offered temporary relief, it is typically more costly and has been used less frequently than larviciding. It usually has involved both aerial and ground fogging with DDT or permethrin products. Current efforts to manage black flies in the adult stage are restricted primarily to the application of repellents and pour-on insecticides.

The use of **chemical insecticides** in managing black flies dates to the dawn of the 20th century, reaching a peak from the mid-1940s into the 1970s when DDT was the principal means of control against both larvae and adults. The development of resistance and the undesirable effects on nontarget organisms led to the abandonment of DDT and the search for surrogate compounds, the most prominent of which were methoxychlor (chlorinated hydrocarbon) and temephos (organophosphate). These compounds, as well as insect growth regulators, were not selective and, therefore, had negative effects on nontarget organisms. The use of chemical insecticides to manage black flies became infrequent toward the end of the 20th century, although compounds such as methoxychlor and temephos continued to be used in a few areas of the world.

Black flies worldwide are managed primarily through the use of the entomopathogenic bacterium *Bacillus thuringiensis* var. *israelensis* (*Bti*, serotype H14), which is aimed at the larval stage. The actual killing agent is an endotoxin in the parasporal inclusions that disrupts the cells of the highly alkaline larval midgut. The efficacy and environmental safety of *Bti* are so superb that most other means of population suppression and management have disappeared since the commercial *Bti* product entered the scene in the early 1980s (Molloy, 1990; Gray et al., 1999). *Bti* can be applied by hand or aircraft. North America's largest suppression program for black flies is operated by the state of Pennsylvania (USA), which treats waterways for *S. jenningsi* in about half of its counties. Because of the intensive use of *Bti* for more than 30 years, target populations should be monitored for resistance.

The potential for exploiting **natural enemies** for simuliid control is enormous. Nearly 200 species of symbiotic organisms, in addition, to many species of bacteria, have been documented from the larval stage, and many have a parasitic relationship with their black fly hosts (McCreadie et al., 2011). Adult simuliids have many of the same symbiotes as the larvae, plus additional symbiotic species. Although natural enemies exert some control in most populations of black flies, attempts to mass produce them have not been made since the 1970s (Laird, 1981). Commonly encountered parasites include mermithid nematodes, microsporidia, the chytrid fungus *Coelomycidium simulii*, and several viruses. The prevalence of infection with these parasites and pathogens is usually less than 10% of a population. Infections typically slow development, however, so that parasitized larvae become relatively more frequent in a population over time as healthy individuals pupate first.

Mermithid nematodes probably hold the greatest promise for biological control of black flies. However, until more can be learned about their taxonomy and host specificity and how to cultivate them economically for mass release, they are unlikely to be useful in integrated pest management programs. Preparasitic mermithid nematodes crawl on stream substrates and use a protrusible stylet to penetrate the host body. As the mermithids mature, they can be seen through the host integument, coiled within the abdomen. Mermithids either exit and kill the host larva or pass into the adult, exiting shortly thereafter. Postparasitic worms molt to adults, mate, and deposit eggs in the streambed.

Patent infections with microsporidia are recognized by the large, irregular cysts that distort the larval host abdomen. Life cycles of microsporidia that attack black flies are poorly known, although transovarial transmission has been documented. Larvae with patent infections of the fungus *Coelomycidium simulii* are packed with minute, spherical thalli throughout their bodies. Thalli produce spores that are released into the water column after death of the host. Two common viruses that infect larvae are

iridescent virus, which imparts an overall blue or violet cast, and cytoplasmic polyhedrosis virus, which creates white bands around the midgut. A significant number of bacterial species living in and on black flies, from parasites to mutualists, offer potential opportunities for control (Tang et al., 2012). Many predators consume black flies; most are typically opportunistic.

Physical control of the breeding habitat is occasionally effective in reducing pest populations, usually when the pest species is concentrated in a restricted area, such as directly downstream of an impoundment. In these situations, attachment sites (e.g., trailing vegetation) can be removed, or water levels can be altered to strand larvae above the water line.

Personal protection for humans involves primarily the use of **repellents**, both natural and synthetic, that are applied directly to the skin or impregnated in clothing. Among the more effective repellents are those with *N*,*N*-diethyl-*meta*-toluamide (DEET) as the active ingredient. Wearing light-colored clothing and minimizing openings in the clothing, such as button holes, through which black flies can gain access to skin, is standard practice when entering areas where black flies are a problem. Fine-mesh head nets are effective in areas where pest populations are intolerable. Fishermen often smoke cigars to ward off the flies, perhaps aided by the nicotine. Many additional means of protection can be found in the annals of folklore, but the utility of most remains suspect.

Various techniques have been devised to protect livestock and other animals, ranging from the use of smudges (i.e., smoldering fires that produce dense smoke) to the application of repellent substances and the use of shelters. Repellent products for livestock historically involved oils and greases, often laced with turpentine or other plant-derived products. Among the more commonly used repellents in recent times are permethrin solutions and ***eartags*** containing ivermectin. Various pour-on and spray formulations of insecticides and repellents are available commercially. White petroleum jelly can be applied inside the ears of horses to reduce biting problems. Providing shelters is an effective means of protecting livestock and poultry because many of the pest species of black flies infrequently enter enclosures. Providing the entries of shelters with self-application devices for repellents provides an added dose of protection. Black flies that enter houses of song birds can be managed by eliminating the vents or surrounding the holes with adhesives.

Onchocerciasis Control

The largest management program in the world for black flies was the World Health Organization's **Onchocerciasis Control Programme** (OCP) in West Africa from 1975 to 2002. Its history, as briefly summarized below, has been written by numerous authors (e.g., Davies, 1994; World Health Organization, 1995; Bump, 2004). The initial foundations for the program were laid in 1968, and in 1975 the program launched its first aerial treatments for the control of onchocerciasis. The goal of the OCP was to eliminate onchocerciasis as a major public health threat in seven West African countries: Benin, Burkina Faso, Ghana, Ivory Coast, Mali, Niger, and Togo. The program later was expanded to include the countries of Guinea, Guinea-Bissau, Senegal, and Sierra Leone, thus covering a total of 11 countries and 50,000 km of rivers. It was directed at the vectors of onchocerciasis, namely members of the *S. damnosum* species complex.

The primary strategy of the OCP was a massive aerial larviciding program aimed at reducing adult vector populations, thus interrupting transmission. Maintaining vectors at a sufficiently low number for a sufficiently long time prevents new cases of transmission while worms in the human reservoir die out, breaking the disease cycle. Given the longevity of adult worms, control programs in endemic areas must be maintained for approximately 15 years to eliminate the worm from the human reservoir (Remme et al., 1990; Plaisier et al., 1991). Prior to the OCP, aerial application of DDT was the main means of control, but by 1970 resistance had begun to develop. From 1975 into the 1980s, the OCP applied primarily temephos to the rivers. The first appearance (1980) of resistance to this compound by the vectors in the OCP area (Guillet et al., 1980) eventually led to the rotation of six insecticides, including *Bti*.

Vector control was integrated with an ivermectin chemotherapy program for the human reservoir in 1988. Ivermectin, originally developed for veterinary purposes, reduces the number of microfilariae in the skin, so that ingestion of sufficient microfilariae by the vectors becomes difficult. This microfilarialcidal drug, however, does not kill the adult worms. A single oral dose of Mectizan (the formulation of ivermectin for humans) every 6−12 months is not only nontoxic at levels higher than prescribed dosages but also sufficient to kill microfilariae in the skin and eyes and reverse progression of the disease. Dying microfilariae, however, can cause temporary adverse reactions in patients. Mass distribution of ivermectin has been possible through the humanitarian efforts of numerous organizations, including Merck and Co., which decided in 1987 to donate ivermectin tablets for the worldwide treatment of onchocerciasis for as long as necessary. In 1995, the **African Programme for Onchocerciasis Control** (APOC), which includes 19 African countries, was formed, with the goal of eliminating onchocerciasis from the continent. Its focus has been the mass distribution of ivermectin. Ten years later, more than 117,000 communities and 350,000 volunteers in 15 African countries were

participating in the ivermectin distribution program (World Health Organization, 2007).

By 1995, vector control had interrupted transmission in about 90% of the original OCP area, protecting more than 30 million people from infection and sparing 100,000 from blindness at a cost of about $360 million. The combined use of ivermectin and weekly insecticide treatments of larval breeding sites was predicted to free the OCP area of onchocerciasis by 2002. Accordingly, the OCP terminated on December 31, 2002, but with approximately 46,000 new cases of onchocerciasis-related blindness in Africa each year, APOC was slated to continue until 2010. From 1995 to 2010, APOC was estimated to have prevented the loss of about 8.2 million years of life at a cost of $257 million (Coffeng et al., 2013). With new cases of onchocerciasis-related blindness in Africa, APOC was extended to 2015. The specter of resistance to ivermectin by *O. volvulus* (Osei-Atweneboana et al., 2011), however, also has threatened to compromise one of the pillars of onchocerciasis control. Civil wars, weakened infrastructure, poor surveillance, irregular financing, and other social disruptions also present formidable obstacles to achieving an onchocerciasis-free continent.

The Carter Center's **Onchocerciasis Elimination Program for the Americas** (OEPA), which was initiated in 1993, includes Brazil, Colombia, Ecuador, Guatemala, Mexico, and Venezuela (Blanks et al., 1998). It, too, relies on mass treatment with ivermectin donated by Merck, and tremendous progress has been made in fighting the disease. Transmission has been interrupted or eliminated in 11 of the 13 New World foci. Colombia was declared onchocerciasis-free in 2013, Ecuador in 2014, Mexico in 2015, and Guatemala in 2016 (World Health Organization, 2016). In Latin America, about 29,500 Yanomami people require continual treatment in the last-remaining foci, which are in the Amazonian region on the border of Brazil and Venezuela (World Health Organization, 2016; Botto et al., 2016). The *S. guianense* complex is the vector in these foci.

REFERENCES AND FURTHER READING

Adami, Y. L., Rodrigues, G., Alves, M. C., Moraes, M. A. P., Banic, D. M., & Maia-Herzog, M. (2014). New records of *Mansonella ozzardi*: A parasite that is spreading from the state of Amazonas to previously uninfected areas of the state of Acre in the Purus river region. *Memórias do Instituto Oswaldo Cruz, 109*, 87–92.

Addison, E. M. (1980). Transmission of *Dirofilaria ursi* Yamaguti, 1941 (Nematoda: Onchocercidae) of black bears (*Ursus americanus*) by blackflies (Simuliidae). *Canadian Journal of Zoology, 58*, 1913–1922.

Adler, P. H., & Crosskey, R. W. (2015). Cytotaxonomy of the Simuliidae (Diptera): A systematic and bibliographic conspectus. *Zootaxa, 3975*, 1–139.

Adler, P. H., & Crosskey, R. W. (2018). *World blackflies (Diptera: Simuliidae): A comprehensive revision of the taxonomic and geographical inventory [2018]*. http://entweb.clemson.edu/biomia/pdfs/blackflyinventory.pdf.

Adler, P. H., & McCreadie, J. W. (1997). The hidden ecology of black flies: Sibling species and ecological scale. *American Entomologist, 43*, 153–161.

Adler, P. H., Currie, D. C., & Wood, D. M. (2004). *The black flies (Simuliidae) of North America*. Ithaca, New York: Cornell University Press. xv + 941 pp. + 24 color plates.

Adler, P. H., Roach, D., Reeves, W. K., Flanagan, J. P., Morrow, M. E., & Toepfer, J. E. (2007). Attacks on the endangered Attwater's prairie-chicken (*Tympanuchus cupido attwateri*) by black flies (Diptera: Simuliidae) infected with an avian blood parasite. *Journal of Vector Ecology, 32*, 309–312.

Adler, P. H., Cheke, R. A., & Post, R. J. (2010). Evolution, epidemiology, and population genetics of black flies (Diptera: Simuliidae). *Infection, Genetics and Evolution, 10*, 846–865.

Adler, P. H., Kúdelová, T., Kúdela, M., Seitz, G., & Ignjatović-Ćupina, A. (2016). Cryptic biodiversity and the origins of pest status revealed in the macrogenome of *Simulium colombaschense* (Diptera: Simuliidae), history's most destructive black fly. *PLoS One, 11*(1), e0147673.

Allison, F. R., Desser, S. S., & Whitten, L. K. (1978). Further observations on the life cycle and vectors of the haemosporidian *Leucocytozoon tawaki* and its transmission to the Fjordland crested penguin. *New Zealand Journal of Zoology, 5*, 663–665.

Anderson, R. C. (1968). The simuliid vectors of *Splendidofilaria fallisensis* of ducks. *Canadian Journal of Zoology, 46*, 610–611.

Atwood, D. W. (1996). *Distribution, abundance, control and field observations of the southern buffalo gnat, Cnephia pecuarum (Diptera: Simuliidae), in Arkansas and Texas* (Ph.D. thesis). Fayetteville, Arkansas: University of Arkansas, 127 pp.

Austin, F. J. (1967). The arbovirus vector potential of a simuliid. *Annals of Tropical Medicine and Parasitology, 61*, 189–199.

Bain, O., & Renz, A. (1993). Infective larvae of a new species of Robertdollfusidae (Adenophorea, Nematoda) in the gut of *Simulium damnosum* in Cameroon. *Annales de Parasitologie Humaine et Comparee, 68*, 182–184.

Barratt, J., Kaufer, A., Peters, B., Craig, D., Lawrence, A., Roberts, T., et al. (2017). Isolation of novel trypanosomatid, *Zelonia australiensis* sp. nov. (Kinetoplastida: Trypanosomatidae) provides support for a Gondwanan origin of dixenous parasitism in the Leishmaniinae. *PLoS Neglected Tropical Diseases, 11*(1), e0005215.

Blanks, J., Richards, F., Beltrán, F., Collins, R., Álvarez, E., Zea Flores, G., et al. (1998). The onchocerciasis elimination program for the Americas: A history of partnership. *Revista Panamericana de Salud Pública, 3*, 367–374.

Bennett, G. F. (1961). On the specificity and transmission of some avian trypanosomes. *Canadian Journal of Zoology, 39*, 17–33.

Botto, C., Basáñez, M. G., Escalona, M., Vivas-Martínez, S., Villamizar, N., Noya-Alarcón, O., et al. (2016). Evidence of suppression of onchocerciasis transmission in the Venezuelan Amazonian focus. *Parasites & Vectors, 9*(1), 1.

Bump, J. B. (2004). *The lion's gaze: African river blindness from tropical curiosity to international development* (Ph.D. thesis). Baltimore, Maryland: Johns Hopkins University, 421 pp.

Burgin, S. G., & Hunter, F. F. (1997). Nectar versus honeydew as sources of sugar for male and female black flies (Diptera: Simuliidae). *Journal of Medical Entomology, 34*, 605–608.

Casiraghi, M., Favia, G., Cancrini, G., Bartoloni, A., & Bandi, C. (2001). Molecular identification of *Wolbachia* from the filarial nematode *Mansonella ozzardi*. *Parasitology Research, 87*, 417–420.

Chanteau, S., Sechan, Y., Moulia-Pelat, J.-P., Luquiaud, P., Spiegel, A., Boutin, J.-P., et al. (1993). The blackfly *Simulium buissoni* and infection by hepatitis B virus on a holoendemic island of the Marquesas Archipelago in French Polynesia. *The American Journal of Tropical Medicine and Hygiene, 48*, 763–770.

Chen, H.-B. (2016). *Chinese black flies*. Guiyang, China: Guizhou Science and Technology Publishing House Co., Ltd., 673 pp.

Ciurea, I., & Dinulescu, G. (1924). Ravages causés par la mouche de Goloubatz en Roumanie; des attaques contre les animaux et contre l'homme. *Annals of Tropical Medicine and Parasitology, 18*, 323–342.

Coffeng, L. E., Stolk, W. A., Zouré, H. G. M., Veerman, J. L., Agblewonu, K. B., Murdoch, M. E., et al. (2013). African programme for onchocerciasis control 1995–2015: Model-estimated health impact and cost. *PLoS Neglected Tropical Disease, 7*(1), e2032.

Colorado-Garzón, F., Adler, P. H., García, L. F., Muñoz de Hoyos, P., Bueno, M., & Matta, N. E. (2016). Estimating diversity in the *Simulium ignescens* and *Simulium tunja* complexes in Colombia: Chromosomal rearrangements as the core of integrative taxonomy. *Journal of Heredity, 108*, 12–24.

Coscarón, S., & Coscarón Arias, C. L. (2007). Neotropical Simuliidae (Diptera: Insecta). In J. Adis, J. R. Arias, G. Rueda-Delgado, & K. M. Wantzen (Eds.), *Vol. 3. Aquatic biodiversity in Latin America*. Bulgaria: Pensoft, Sofia, 685 pp.

Crosskey, R. W. (1967). The classification of *Simulium* Latreille (Diptera: Simuliidae) from Australia, New Guinea and the western Pacific. *Journal of Natural History, 1*, 23–51.

Crosskey, R. W. (1969). A re-classification of the Simuliidae (Diptera) of Africa and its islands. In *Bulletin of the British Museurm (Natural History). Entomology Supplement 14* (pp. 1–195).

Crosskey, R. W. (1990). *The natural history of blackflies*. Chichester, England: John Wiley & Sons Ltd., 711 pp.

Crosskey, R. W., & Howard, T. M. (1997). *A new taxonomic and geographical inventory of world blackflies (Diptera: Simuliidae)*. London: The Natural History Museum, 144 pp.

Cupp, E. W., & Cupp, M. S. (1997). Black fly (Diptera: Simuliidae) salivary secretions: Importance in vector competence and disease. *Journal of Medical Entomology, 34*, 87–94.

Currie, D. C., & Adler, P. H. (2008). Global diversity of black flies (Diptera: Simuliidae) in freshwater. *Hydrobiologia, 595*, 469–475.

Davies, J. B. (1994). Sixty years of onchocerciasis vector control: A chronological summary with comments on eradication, reinvasion, and insecticide resistance. *Annual Review of Entomology, 39*, 23–45.

Dirie, M. F., Ashford, R. W., Mungomba, L. M., Molyneux, D. H., & Green, E. E. (1990). Avian trypanosomes in *Simulium* and sparrowhawks (*Accipiter nisus*). *Parasitology, 101*, 243–247.

Eaton, D. P., Diaz, L. A., Hans-Filho, G., dos Santos, V., Aoki, V., Friedman, H., et al., the Cooperative Group on Fogo Selvagem Research. (1998). Comparison of black fly species (Diptera: Simuliidae) on an Amerindian reservation with a high prevalence of fogo selvagem to neighboring disease-free sites in the state of Mato Grosso do Sul, Brazil. *Journal of Medical Entomology, 35*, 120–131.

Fallis, A. M., Jacobson, R. L., & Raybould, J. N. (1973). Experimental transmission of *Trypanosoma numidae* Wenyon to Guinea fowl and chickens in Tanzania. *Journal of Protozoology, 20*, 436–437.

Fallis, A. M., Desser, S. S., & Khan, R. A. (1974). On species of *Leucocytozoon*. *Advances in Parasitology, 12*, 1–67.

Fredeen, F. J. H. (1977). A review of the economic importance of black flies (Simuliidae) in Canada. *Quaestiones Entomologicae, 13*, 219–229.

Fredeen, F. J. H. (1985). Some economic effects of outbreaks of black flies (*Simulium luggeri* Nicholson & Mickel) in Saskatchewan. *Quaestiones Entomologicae, 21*, 175–208.

Fukuda, M., Otsuka, Y., Uni, S., Boda, T., Daisaku, H., Hasegawa, H., et al. (2011). Zoonotic onchocerciasis in Hiroshima, Japan, and molecular analysis of a paraffin section of the agent for a reliable identification. *Parasite, 18*, 185–188.

Gray, E. W., & Noblet, R. (2014). Black fly rearing and use in laboratory bioassays. In K. Maramorosch, & F. Mahmood (Eds.), *Rearing animal and plant pathogen vectors* (pp. 42–72). Boca Raton, Florida: Science Publishers, CRC Press.

Gray, E. W., Adler, P. H., Coscarón-Arias, C., Coscarón, S., & Noblet, R. (1999). Development of the first black fly (Diptera: Simuliidae) management program in Argentina and comparison with other programs. *Journal of the American Mosquito Control Association, 15*, 400–406.

Gudgel, E. F., & Grauer, F. H. (1954). Acute and chronic reactions to black fly bites (*Simulium* fly). *Archives of Dermatology and Syphilology, 70*, 609–615.

Guillet, P., Escaffre, H., Ouédraogo, M., & Quillévéré, D. (1980). Mise en évidence d'une résistance au téméphos dans le complexe *S. damnosum* (*S. sanctipauli* et *S. soubrense*) en Côte d'Ivoire (zone du programme de lutte contre l'onchocercose dans la région du bassin de la Volta). *Cahiers ORSTOM Séries Entomologie Médicale et Parasitologie, 23*, 291–299.

Hassan, H. K., Bolcen, S., Kubofcik, J., Nutman, T. B., Eberhard, M. L., Middleton, K., et al. (2015). Isolation of *Onchocerca lupi* in dogs and black flies, California, USA. *Journal of Emerging Infectious Diseases, 21*, 789–796.

Herman, C. M., Barrow, J. H., & Tarshis, I. B. (1975). Leucocytozoonosis in Canada geese at the Seney National Wildlife Refuge. *Journal of Wildlife Diseases, 11*, 404–411.

Johnson, T. P., Tyag, R., Lee, P. R., Lee, M.-H., Johnson, K. R., Kowalak, J., et al. (2017). Nodding syndrome may be an autoimmune reaction to the parasitic worm *Onchocerca volvulus*. *Science Translational Medicine, 9*. eaaf6953.

Laird, M. (1981). *Blackflies: The future for biological methods in integrated control*. New York: Academic Press.

Lok, J. B., Cupp, E. W., & Bernardo, M. J. (1983). *Simulium jenningsi* Malloch (Diptera: Simuliidae): A vector of *Onchocerca lienalis* Stiles (Nematoda: Filarioidea) in New York. *American Journal of Veterinary Research, 44*, 2355–2358.

Lotta, I. A., Pacheco, M. A., Escalante, A. A., González, A. D., Mantilla, J. S., Moncada, L. I., et al. (2016). *Leucocytozoon* diversity and possible vectors in the Neotropical highlands of Colombia. *Protist, 167*, 185–204.

Malmqvist, B., Adler, P. H., Kuusela, K., Merritt, R. W., & Wotton, R. S. (2004a). Black flies in the boreal biome, key organisms in both terrestrial and aquatic environments: A review. *Écoscience, 11*, 187–200.

Malmqvist, B., Strasevicius, D., Hellberg, O., Adler, P. H., & Bensch, S. (2004b). Vertebrate host specificity of wild-caught blackflies revealed by mitochondrial DNA in blood. *Proceedings of the Royal Society of London B (Supplement), Biology Letters, 271*, S152–S155.

McCreadie, J. W., Adler, P. H., & Hamada, N. (2005). Patterns of species richness for blackflies (Diptera: Simuliidae) in the Nearctic and Neotropical regions. *Ecological Entomology, 30*, 201–209.

McCreadie, J. W., Adler, P. H., & Beard, C. E. (2011). Ecology of symbiotes of larval black flies (Diptera: Simuliidae): Distribution, diversity, and scale. *Environmental Entomology, 40*, 289−302.

McCreadie, J. W., & Adler, P. H. (2012). The roles of abiotic factors, dispersal, and species interactions in structuring stream assemblages of black flies (Diptera: Simuliidae). *Aquatic Biosystems, 8*(14), 1−11.

McCreadie, J. W., Hamada, N., Grillet, M. E., & Adler, P. H. (2017). Alpha richness and niche breadth of a widespread group of aquatic insects in Nearctic and Neotropical streams. *Freshwater Biology, 62*, 329−339.

McGaha, T. W., Jr., Young, R. M., Burkett-Cadena, N. D., Iburg, J. P., Beau, J. M., Hassan, S., et al. (2015). Identification of communal oviposition pheromones from the black fly *Simulium vittatum*. *PLoS One, 10*, e0118904.

Mead, D. G., Ramberg, F. B., Besslesen, D. G., & Máre, C. J. (2000). Transmission of vesicular stomatitis virus from infected to noninfected black flies co-feeding on nonviremic deer mice. *Science, 287*, 485−487.

Mock, D. E., & Adler, P. H. (2002). Black flies (Diptera: Simuliidae) of Kansas: Review, new records, and pest status. *Journal of the Kansas Entomological Society, 75*, 203−213.

Molloy, D. (1990). Progress in the biological control of black flies with *Bacillus thuringiensis israelensis,* with emphasis on temperate climates. In H. de Barjac, & D. J. Sutherland (Eds.), *Bacterial control of mosquitoes and black flies: Biochemistry, genetics, and applications of Bacillus thuringiensis israelensis and Bacillus sphaericus* (pp. 161−186). New Brunswick, New Jersey: Rutgers University Press.

Muller, R., & Horsburgh, R. C. R. (1987). *Bibliography of onchocerciasis (1841−1985).* C. A. B. International Institute of Parasitology.

Murdock, C. C., Adler, P. H., Frank, J., & Perkins, S. L. (2015). Molecular analyses on host-seeking black flies (Diptera: Simuliidae) reveal a diverse assemblage of *Leucocytozoon* (Apicomplexa: Haemospororida) parasites in an alpine ecosystem. *Parasites & Vectors, 8*(343), 1−7.

Mykytowycz, R. (1957). The transmission of myxomatosis by *Simulium melatum* Wharton (Diptera: Simuliidae). *CSIRO Wildlife Research, 2*, 1−4.

Noblet, R., Kissam, J. B., & Adkins, T. R., Jr. (1975). *Leucocytozoon smithi:* Incidence of transmission by black flies in South Carolina (Diptera: Simuliidae). *Journal of Medical Entomology, 12*, 111−114.

O'Roke, E. C. (1934). A malaria-like disease of ducks caused by *Leucocytozoon anatis* Wickware. *University of Michigan School of Forestry Conservation Bulletin, 4*, 1−44.

Osei-Atweneboana, M. Y., Awadzi, K., Attah, S. K., Boakye, D. A., Gyapong, J. O., & Prichard, R. K. (2011). Phenotypic evidence of emerging ivermectin resistance in *Onchocerca volvulus*. *PLoS Neglected Tropical Diseases, 5*, e998.

Palmer, R. W. (1995). *Biological and chemical control of blackflies (Diptera: Simuliidae) in the Orange River*. Report to the Water Research Commission by the Onderstepoort Veterinary Institute. WRC Report 343/1/95 (pp. 1−10).

Pearlman, E. (2003). Immunopathogenesis of *Onchocerca volvulus* keratitis (river blindness): A novel role for endosymbiotic *Wolbachia* bacteria. *Medical Microbiology and Immunology, 192*, 57−60.

Plaisier, A. P., Van Oortmarssen, G. J., Remme, J. H. F., & Habbema, J. D. F. (1991). The reproduction lifespan of *Onchocerca volvulus* in West Africa savanna. *Acta Tropica, 48*, 271−284.

Pledger, D. J., Samuel, W. M., & Craig, D. A. (1980). *Black flies (Diptera: Simuliidae) as possible vectors of legworm (Onchocerca cervipedis) in moose of central Alberta*. Proceedings of the North American Moose Conference Workshop. 16 (pp. 171−202).

Post, R. J., Mustapha, M., & Krueger, A. (2007). Taxonomy and inventory of the cytospecies and cytotypes of the *Simulium damnosum* complex (Diptera: Simuliidae) in relation to onchocerciasis. *Tropical Medicine and International Health, 12*, 1342−1353.

Reeves, W. K., Adler, P. H., Rätti, O., Malmqvist, B., & Strasevicius, D. (2007). Molecular detection of *Trypanosoma* (Kinetoplastida: Trypanosomatidae) in black flies (Diptera: Simuliidae). *Comparative Parasitology, 74*, 171−175.

Remme, J. H. F., De Sole, G., & Van Oortmarssen, G. J. (1990). The predicted and observed decline in the prevalence and intensity of onchocerciasis infection during 14 years of successful vector control. *Bulletin of the World Health Organization, 68*, 331−339.

Remme, J. H. F., Feenstra, P., Lever, P. R., Medici, A. C., Morel, C. M., Noma, M., et al. (2006). Tropical diseases targeted for elimination: Chagas disease, lymphatic filariasis, onchocerciasis, and leprosy. In D. T. Jamison, J. G. Breman, A. R. Measham, G. Alleyne, M. Claeson, D. B. Evans, et al. (Eds.), *Disease control priorities in developing countries* (2nd ed., pp. 433−449). Washington, DC: World Bank.

Riley, C. V. (1887). *Report of the entomologist*. United States Department of Agriculture Report 1886 (pp. 459−492), 11 plates.

Rohner, C., Krebs, C. J., Hunter, D. B., & Currie, D. C. (2000). Roost site selection of great horned owls in relation to black fly activity: An anti-parasite behavior? *The Condor: Ornithological Applications, 102*, 950−955.

Rubtsov, I. A. (1956). Blackflies (fam. Simuliidae). In *"Fauna of the USSR". New series No. 64, insects, Diptera, 6 (6)*. Moscow: Akademii Nauk SSSR [In Russian; English translation: 1990. Blackflies (Simuliidae). Second Edition. "Fauna of the USSR". Diptera, 6 (6). E. J. Brill, Leiden.].

Sanford, D., Eikenhorst, B., Lamb, T., Cates, J. E., Robinson, J., Olsen, J., et al. (1993). Black flies cause costly losses in East Texas ostriches and emus. *Texas Agricultural Extension Service Veterinary Quarterly Review, 9*(2), 1−2.

Sariözkan, S., Inci, A., Yildirim, A., Düzlü, O., Gray, E. W., & Adler, P. H. (2014). Economic losses during an outbreak of *Simulium* (*Wilhelmia*) species (Diptera: Simuliidae) in the Cappadocia region of Turkey. *Turkiye Parazitolojii Dergisi, 38*, 116−119.

Shelley, A. J. (1988a). Biosystematics and medical importance of the *Simulium amazonicum* group and the *S. exiguum* complex in Latin America. In M. W. Service (Ed.), *Biosystematics of haematophagus insects* (pp. 203−220). Oxford: Clarendon Press.

Shelley, A. J. (1988b). Vector aspects of the epidemiology of onchocerciasis in Latin America. *Annual Review of Entomology, 30*, 337−366.

Shelley, A. J., & Coscarón, S. (2001). Simuliid blackflies (Diptera: Simuliidae) and ceratopogonid midges (Diptera: Ceratopogonidae) as vectors of *Mansonella ozzardi* (Nematoda: Onchocercidae) in northern Argentina. *Memórias do Instituto Oswaldo Cruz, 96*, 451−458.

Shelley, A. J., Hernández, L. M., Maia-Herzog, M., Luna Dias, A. P. A., & Garritano, P. R. (2010). The blackflies (Diptera: Simuliidae) of Brazil. In J. R. Arias, S. Golovach, K. M. Wantzen, & E. Domínguez (Eds.), *Vol. 6. Aquatic biodiversity in Latin America*. Bulgaria: Pensoft, Sofia, 821 pp.

Smith, P. F., Howerth, E. W., Carter, D., Gray, E. W., Noblet, R., & Mead, D. G. (2009). Mechanical transmission of vesicular stomatitis New

Jersey virus by *Simulium vittatum* (Diptera: Simuliidae) to domestic swine (*Sus scrofa*). *Journal of Medical Entomology, 46*, 1537–1540.

Stokes, J. H. (1914). A clinical, pathological and experimental study of the lesions produced by the bite of the "black fly" (*Simulium venustum*). *Journal of Cutaneous Diseases, 32*(751–769), 830–856.

Sutcliffe, J. F. (1986). Black fly host location: A review. *Canadian Journal of Zoology, 64*, 1041–1053.

Sutcliffe, J. F., & McIver, S. B. (1984). Mechanics of blood-feeding in black flies (Diptera, Simuliidae). *Journal of Morphology, 180*, 125–144.

Takaoka, H. (1999). Review on zoonotic *Onchocerca* species and their insect vectors in Japan. *Medical Entomology & Zoology, 50*, 1–8 (In Japanese, with English summary).

Takaoka, H. (2003). *The black flies (Diptera: Simuliidae) of Sulawesi, Maluku and Irian Jaya*. Fukuoka, Japan: Kyushu University Press.

Takaoka, H. (2017). Speciation, faunal affinities and geographical dispersal of black flies (Diptera: Simuliidae) in the Oriental Region. *Acta Tropica, 166*, 234–240.

Takaoka, H., & Davies, D. M. (1995). *The black flies (Diptera: Simuliidae) of West Malaysia*. Japan: Kyushu University Press.

Tang, X., Adler, P. H., Vogel, H., & Ping, L. (2012). Gender-specific bacterial composition of black flies (Diptera: Simuliidae). *FEMS Microbiology Ecology, 80*, 659–670.

Taylor, M. J., Hoerauf, A., Townson, S., Slatko, B. E., & Ward, S. A. (2014). Anti-*Wolbachia* drug discovery and development: Safe macrofilaricides for onchocerciasis and lymphatic filariasis. *Parasitology, 141*, 119–127.

Tchounkeu, Y. F. L., Onyeneho, N. G., Wanji, S., Kabali, A. T., Manianga, C., Amazigo, U. V., et al. (2012). Changes in stigma and discrimination of onchocerciasis in Africa. *Transactions of the Royal Society of Tropical Medicine and Hygiene, 106*, 340–347.

Uni, S., Fukuda, M., Agatsuma, T., Bain, O., Otsuka, Y., Nakatani, J., et al. (2015). *Onchocerca takaokai* n. sp. (Nematoda: Filarioidea) in Japanese wild boars (*Sus scrofa leucomystax*): Description and molecular identification of intradermal females. *Parasitology International, 64*, 493–502.

Urbanek, R. P., Zimorski, S. E., Fasoli, A. M., & Szyszkoski, E. K. (2010). Nest desertion in a reintroduced population of migratory whooping cranes. *Proceedings of the North American Crane Workshop, 11*, 133–141.

Votýpka, J., & Svobodová, M. (2004). *Trypanosoma avium:* Experimental transmission from black flies to canaries. *Parasitology Research, 92*, 147–151.

Votýpka, J., Oborník, M., Volf, P., Svobodová, M., & Lukeš, J. (2002). *Trypanosoma avium* of raptors (Falconiformes): Phylogeny and identification of vectors. *Parasitology, 125*, 253–263.

Wahl, G. (1996). Identification of a common filarial larva in *Simulium damnosum* s. l. (Type D, Duke, 1967) as *Onchocerca ramachandrini* from the wart hog. *The Journal of Parasitology, 82*, 520–524.

Wahl, G., & Renz, A. (1991). Transmission of *Onchocerca dukei* by *Simulium bovis* in north-Cameroon. *Tropical Medicine & Parasitology, 42*, 368–370.

Wahl, G., Ekale, D., & Schmitz, A. (1998). *Onchocerca ochengi:* Assessment of the *Simulium* vectors in north Cameroon. *Parasitology, 116*, 327–336.

Werner, D., & Adler, P. H. (2005). A faunistic review of the black flies (Simuliidae, Diptera) of the federal state of Sachsen-Anhalt, Germany. *Abhandlungen und Berichte für Naturkunde, 27*, 205–245.

Wilhelm, A., Betke, P., & Jacob, K. (1982). Simuliotoxikose beim ren (*Rangifer tarandus*). In R. Ippen, & H. D. Schräder (Eds.), *Verhandlungbericht des XXIV Internationalen Symposium über die Erkrankungen der Zootiere Erkrankungen der Zootiere* (pp. 357–360). Berlin: Akademie-Verlag.

World Health Organization. (1995). Onchocerciasis and its control: Report of a WHO expert committee on onchocerciasis control. *WHO Technical Report Series, 852*, 1–10.

World Health Organization. (2007). *Onchocerciasis control in the WHO African region: Current situation and way forward. AFR/RC57/5*, 7 pp.

World Health Organization. (2016). Progress toward eliminating onchocerciasis in the WHO region of the Americas: Verification of elimination of transmission in Guatemala. *Weekly Epidemiological Record, 91*(43), 501–516.

World Health Organization. (2017). *Onchocerciasis. Fact Sheet 374*. http://www.who.int/mediacentre/factsheets/fs374/en/.

This page intentionally left blank

Chapter 15

Mosquitoes (Culicidae)

Woodbridge A. Foster[1] **and Edward D. Walker**[2]

[1]*Department of Evolution, Ecology, and Organismal Biology, and Department of Entomology, The Ohio State University, Columbus, OH, United States;* [2]*Department of Microbiology and Molecular Genetics, and Department of Entomology, Michigan State University, East Lansing, MI, United States*

Since ancient times, mosquito bites or habitats have been associated with human disease, and, in 1878, mosquitoes were the first arthropods formally incriminated as intermediate hosts of vertebrate parasites. During the past century it has become established that mosquitoes are the most important arthropods affecting human health. They attain their greatest impact as vectors for the organisms causing such well-known human diseases as malaria, filariasis, encephalitis, yellow fever, and dengue. These afflictions are especially severe in developing regions of the tropics. They cause early death and chronic debilitation that can strain the resources of health services and reduce human productivity, thereby perpetuating economic hardship.

Mosquito-borne diseases also persist in industrialized temperate countries. Yet, human discomfort from bites is often the chief concern. In the United States, hundreds of millions of dollars are spent annually to control them for this reason alone. Additionally, large populations of mosquitoes can cause intense irritation and extensive blood loss to livestock and wildlife, resulting in reduced productivity and even death.

Mosquitoes occur in practically every region of every continent in the world except Antarctica. They develop in an extremely broad range of biotic communities: arctic tundra, boreal forests, high mountains, plains, deserts, tropical forests, salt marshes, and ocean tidal zones. The greatest species diversity occurs in tropical forests, but extremely high densities of mosquitoes are common even in the species-poor biomes, such as the tundra. Many species have benefited from human alteration of the environment, and a few have become domesticated. Because of their immense importance, mosquitoes have been the subject of many major books. Among the more important books that deal exclusively with mosquito biology are texts by Christophers (1960), Clements (1992, 1999), Forattini (1962, 1965), Gillett (1971), Bock and Cardew (1996), Horsfall (1955), Lounibos et al. (1985), Mattingly (1969), and Service (1990, 1993a). *Journal of the American Mosquito Control Association* is devoted mainly to studies of mosquitoes. A substantial proportion of the scientific articles in the *Journal of Medical Entomology* and *Medical and Veterinary Entomology* also report mosquito research, as other scientific journals increasingly do. *Wing Beats* is a trade magazine dedicated to mosquitoes.

TAXONOMY

The family **Culicidae**, derived from *culex*, the Latin name for "gnat," is a member of one of the main stocks of Nematocera, the infraorder Culicomorpha. It consists of two superfamilies that include all of the piercing/sucking nematocerans, both predators and blood-feeding biters. The superfamily Chironomoidea comprises the families Chironomidae and Thaumaleidae, which have nonpiercing mouthparts, and Simuliidae and Ceratopogonidae, which pierce either vertebrates or invertebrates. The superfamily Culicoidea comprises the Dixidae, Corethrellidae, Chaoboridae, and Culicidae, the second and fourth of which feed on vertebrate blood. Several of these families are superficially similar. However, among all the culicomorphs, the long proboscis of mosquitoes is distinctive. It is considered the most specialized of biting mouthparts among Nematocera and indicates a long and close association of mosquitoes with vertebrate animals. Wood and Borkent (1989) provide an overview of nematoceran phylogeny and classification.

Culicidae consists of about 3,500 recognized species. The largest number remaining to be discovered probably inhabits tropical rainforests, where faunas are more diverse but less well surveyed than temperate regions. Species that have been studied intensively often reveal that they consist of complexes of closely related species, indicating that many reproductively isolated and niche-specific forms remain to be identified or are undergoing speciation.

Medical and Veterinary Entomology. https://doi.org/10.1016/B978-0-12-814043-7.00015-7

TABLE 15.1 Classification of Culicidae

Subfamily	Tribe	Genera
Anophelinae		*Anopheles* (*An.*), *Bironella* (*Bi.*), *Chagasia* (*Ch.*)
Culicinae	Aedeomyiini	*Aedeomyia* (*Ad.*)
	Aedini	*Aedes* (*Ae.*), *Armigeres* (*Ar.*), *Eretmapodites* (*Er.*),
		Haemagogus (*Hg.*), *Heizmannia* (*Hz.*), *Opifex* (*Op.*), *Psorophora* (*Ps.*),
		Udaya (*Ud.*), *Verrallina* (*Ve.*), *Zeugnomyia* (*Ze.*)
	Culicini	*Culex* (*Cx.*), *Deinocerites* (*De.*), *Galindomyia* (*Ga.*), *Lutzia* (*Lu.*)
	Culisetini	*Culiseta* (*Cs.*)
	Ficalbiini	*Ficalbia* (*Fi.*), *Mimomyia* (*Mi.*)
	Hodgesiini	*Hodgesia* (*Ho.*)
	Mansoniini	*Coquillettidia* (*Cq.*), *Mansonia* (*Ma.*)
	Orthopodomyiini	*Orthopodomyia* (*Or.*)
	Sabethini	*Isostomyia* (*Is.*), *Johnbelkinia* (*Jb.*), *Kimia* (*Km.*), *Limatus* (*Li.*), *Malaya* (*Ml.*), *Maorigoeldia* (*Mg.*), *Onirion* (*On.*), *Runchomyia* (*Ru.*), *Sabethes* (*Sa.*), *Shannoniana* (*Sh.*), *Topomyia* (*To.*), *Trichoprosopon* (*Tr.*), *Tripteroides* (*Tp.*), *Wyeomyia* (*Wy.*).
	Toxorhynchitini	*Toxorhynchites* (*Tx.*)
	Uranotaeniini	*Uranotaenia* (*Ur.*)

The classification of all mosquitoes into two subfamilies, 11 tribes of Culicinae, and 41 genera is based on Knight and Stone (1977) and modified according to updates to the online Systematic Catalog of the Culicidae, Walter Reed Biosystematics Unit website. In parentheses are the two-letter generic abbreviations recognized by the American Mosquito Control Association and used in several journals and books.

Current culicid classification (Table 15.1) recognizes two subfamilies: **Anophelinae** and **Culicinae**. Anophelinae is considered to be a primitive group. The former subfamily Toxorhynchitinae has been reduced to tribe status on the basis of cladistic analysis of morphological and nucleotide-sequence data (Harbach and Kitching, 1998). Anopheline eggs bear characteristic floats, their larvae lack air tubes, and adults have elongate palps in both sexes. Typical culicine larvae have air tubes, and adult females have short palps.

There are 41 genera of mosquitoes, 38 of which are in the subfamily Culicinae. Culicines are organized into 11 tribes, the most diverse of which are Aedini and Sabethini in terms of numbers of genera and species worldwide. The 13 genera in North America north of Mexico, and the number of species in each, are *Anopheles* (23), *Aedes* (83), *Psorophora* (17), *Haemagogus* (1), *Culex* (37), *Deinocerites* (3), *Culiseta* (8), *Coquillettidia* (1), *Mansonia* (2), *Orthopodomyia* (3), *Wyeomyia* (5), *Uranotaenia* (3), and *Toxorhynchites* (2) (Darsie and Ward, 1981, 2004; online-updated classification of Knight and Stone, 1977, in Systematic Catalog of Culicidae, Walter Reed Biosystematics Unit). A proposed elevation of many subgenera to the rank of genus led to some confusion in the mosquito nomenclature used by researchers during 2000–2015. A classification of the tribe Aedini, one that takes into account both the need for stability of names and the currently understood evolutionary relationships of taxa, has been presented by Wilkerson et al. (2015) and is used in this chapter.

Three important species groups of mosquitoes worldwide are the *Anopheles gambiae* and *Culex pipiens* complexes and the *Aedes* subgenus *Stegomyia*. The ***Anopheles gambiae* complex** of Africa consists of eight recognized species. Two of these, *An. gambiae* (Fig. 15.33) and *An. arabiensis*, are widespread and important vectors of malaria and lymphatic filariasis. *Anopheles arabiensis* tends to occur in somewhat drier regions than does *An. gambiae*. Both prefer to bite humans, but *An. gambiae* is more anthropophilic, endophilic, and endophagic, and therefore it is the more important vector. Another species in that complex, *Anopheles coluzzii*, is a regionally important vector. Other important anopheline complexes, such as the Asian *An. dirus* complex and the African *An. funestus* group, also are recognized.

The *Culex pipiens* species assemblage is a ubiquitous group of two closely related domestic and peridomestic species distributed worldwide: 1) the temperate species *Cx. pipiens*, the northern house mosquito (including the facultatively autogenous *Culex pipiens*, form molestus, often breeding in subterranean habitats); and 2) the tropical and subtropical *Cx. quinquefasciatus* (formerly called *Culex*

fatigans), the southern house mosquito (Fig. 15.29). Their ranges overlap in the central latitudes of the United States, where they commonly hybridize. They are vectors of several human pathogens, such as St. Louis encephalitis virus, West Nile virus, and lymphatic filariasis, as well as bird malaria. Two possible members of the species assemblage, *Cx. australicus* and *Cx. globocoxitus*, inhabit Australia. A form occurring in temperate China and Japan, *Cx. pallens*, has no formal status as a species. A full examination of this problematic assemblage is presented in a series of papers introduced and summarized by Linthicum (2012).

Several brightly marked *Aedes* spp. in the large **subgenus *Stegomyia*** are medically important, including *Ae. aegypti* and *Ae. albopictus*. *Aedes aegypti*, the yellow fever mosquito (Fig. 15.23), has a worldwide distribution in the tropics and subtropics. It is the primary vector of dengue, urban yellow fever, Chikungunya, and Zika viruses. It exists in at least two forms, *aegypti* and *formosus*, considered to be either subspecies or separate species. *Aedes aegypti formosus* is the original feral form and is found in large parts of interior Africa. It has a black body, develops in tree holes, feeds on a wide variety of animals, and rarely enters houses. It has adapted to some domestic situations in Africa, where it develops in rain-filled containers. *Aedes aegypti aegypti* is a paler, brownish-black domestic form. It occurs mainly in coastal regions of Africa and is distributed throughout much of southern Asia and most warmer parts of the New World, including the southern United States. In Africa it has become independent of rain, developing in hand-filled water jars without regard to season. On other continents, where it does not compete with *Ae. a. formosus*, it uses both rain-filled and hand-filled containers. Some authorities recognize a still paler and more domestic type of *Ae. a. aegypti* as the subspecies *Ae. a. queenslandensis*, but this is probably only a localized variant.

Aedes albopictus, the Asian tiger mosquito (Fig. 15.26), similarly occupies water-filled containers and also transmits dengue, Chikungunya, and Zika viruses. It was largely confined to Asia, where it occurs in tropical and subtropical rural settings. It readily oviposits in tree holes. A cold-hardy, egg-diapausing strain of this mosquito has been carried from northern Japan to other parts of the world via the trade in used automobile and truck tires. The first established population was detected in Texas (USA) in 1985. It has since spread through much of the southern, central, and eastern United States, including foci in the upper Midwest, much farther north than the nondiapausing *Ae. aegypti*. It also has gained a foothold in several other parts of the world. In most of its range in the southern United States, *Ae. albopictus* has replaced *Ae. aegypti* as the predominant mosquito in artificial containers in suburban and rural environments.

Other important members of the subgenus *Stegomyia* include *Ae. africanus*, *Ae. bromeliae*, and *Ae. luteocephalus*, which transmit yellow fever and dengue viruses in parts of Africa; and *Ae. polynesiensis* and *Ae. pseudoscutellaris*, which transmit lymphatic filariasis in South Pacific islands.

Keys to the mosquito genera worldwide were provided by Mattingly (1971). Keys for the identification of species of restricted geographical regions are available for most states in the United States, for Canadian provinces, and for many countries throughout the world. These include handbooks that also present biological and medical information on individual species. Good examples of statewide handbooks are written for New Jersey (Headlee, 1945), California (Bohart and Washino, 1978), Indiana (Siverly, 1972), Minnesota (Barr, 1958), New York (Means, 1979), Florida (Darsie and Morris, 1998), and Alaska (Gjullin et al., 1961). The US regional handbooks include the southeastern United States (King et al., 1960; Burkett-Cadena, 2013) and the northwestern states (Stage et al., 1952). Some handbooks include keys to pupae, and the handbook for Illinois (Ross and Horsfall, 1965) is noteworthy in particular for its egg keys.

The most recent comprehensive treatments of North American species are Wood et al. (1979), which contains keys to larvae and adults of Canada, plates of taxonomic structures for each species, distribution maps, and biological information; and Darsie and Ward (1981), updated in 2004, which covers all of North America north of Mexico and has illustrated keys and distribution maps. These works were preceded by Carpenter and LaCasse (1955), which contains formal descriptions, biology, and meticulously crafted full-page plates of adults of each species. A thorough treatment of North American genera was presented by Stone (1981).

Other parts of the world covered by notable works include the South Pacific (Belkin, 1962), United Kingdom (Marshall, 1938), the Neotropical Region (Lane, 1953), and Japan and Korea (LaCasse and Yamaguti, 1950).

For details on morphological terminology and anatomical features of mosquitoes, the work of Harbach and Knight (1980) is recommended. Members of species complexes often are indistinguishable morphologically. Specialists have overcome some of these problems by using chromosome banding patterns, isozyme profiles, DNA probes and DNA restriction-fragment patterns, or nucleotide sequences of various genes to distinguish such species from one another. These molecular methods for identification are widely available for routine field work.

All known mosquito species in the world are listed in *A Catalog of the Mosquitoes of the World* (Knight and Stone, 1977), plus its four supplements (Knight, 1978; Ward, 1984; Gaffigan and Ward, 1985; Ward, 1992). Updates to the catalog are presented online by the Walter Reed Biosystematics Unit. This work provides the taxonomic history

and current standing of all recognized species, their distributions by country, and references to the general literature and to taxonomic works for all regions of the world. The most recent large treatment of a major geographic area is the 12-volume catalog on the Australasian Region, edited by Debenham, Hicks, Lee, and others (1980–1989).

Original systematic studies of mosquitoes are published in several scientific journals, but the one devoted solely to this subject, *Mosquito Systematics*, was subsumed under the *Journal of the American Mosquito Control Association* in 1995. Two long series of valuable papers of international scope on mosquito taxonomy and distribution have been published in the journal *Contributions of the American Entomological Institute: Contributions to the Mosquito Fauna of Southeast Asia* and *Mosquito Studies*. Preferred common names of mosquito species are listed by the Entomological Society of America (Bosik, 1997) and by Pittaway (1992). An internationally accepted set of two-letter abbreviations for all mosquito genera is shown in Table 15.1. These abbreviations appear in most mosquito publications and are used in this chapter.

MORPHOLOGY

The **eggs** of most mosquitoes are elongate, ovoid, or spindle-shaped; others are spherical or rhomboid. The outermost layer of the egg shell, or **chorion** (Fig. 15.1), often has intricate surface structures and patterns diagnostic of the particular species. The chorions of *Anopheles* species have unique, transparent air-filled compartments flanking the egg that serve as **floats** (Fig. 15.1A). Eggs of *Anopheles*, *Toxorhynchites*, *Wyeomyia*, *Aedes*, *Psorophora*, and *Haemagogus* spp. are laid individually, whereas in *Culex*, *Culiseta*, *Coquillettidia*, and *Mansonia* spp., they are attached together in a single clump, forming a floating egg raft (Fig. 15.2A) or submerged cluster (Fig. 15.2B). *Culex* eggs have a cup-shaped corolla at one end (Fig. 15.1B), allowing them to sit vertically on the water surface in a raft (Fig. 15.2A); the upper ends have apical droplets with a chemical thought to maintain the raft upright.

Mosquito larvae, commonly known as **wigglers** or **wrigglers**, pass through four instars, which closely resemble one another except for their size. Larvae are rich in taxonomic characters that are easy to see on slide-mounted specimens (Fig. 15.3). The head is defined by a distinct capsule bearing a pair of "eyes" composed of clusters of lateral ocelli, a pair of antennae of varying shape and length, and chewing mouthparts bearing a variety of brushes, combs, and sweepers used in feeding (Fig. 15.4). The lateral **palatal brushes** on the labrum create water currents that draw floating or suspended particles toward the mouth. Sweepers and brushes on the mandibles, and brushes on the maxillae, are thought to collect and pack particles to create a bolus of food in the pharynx. In

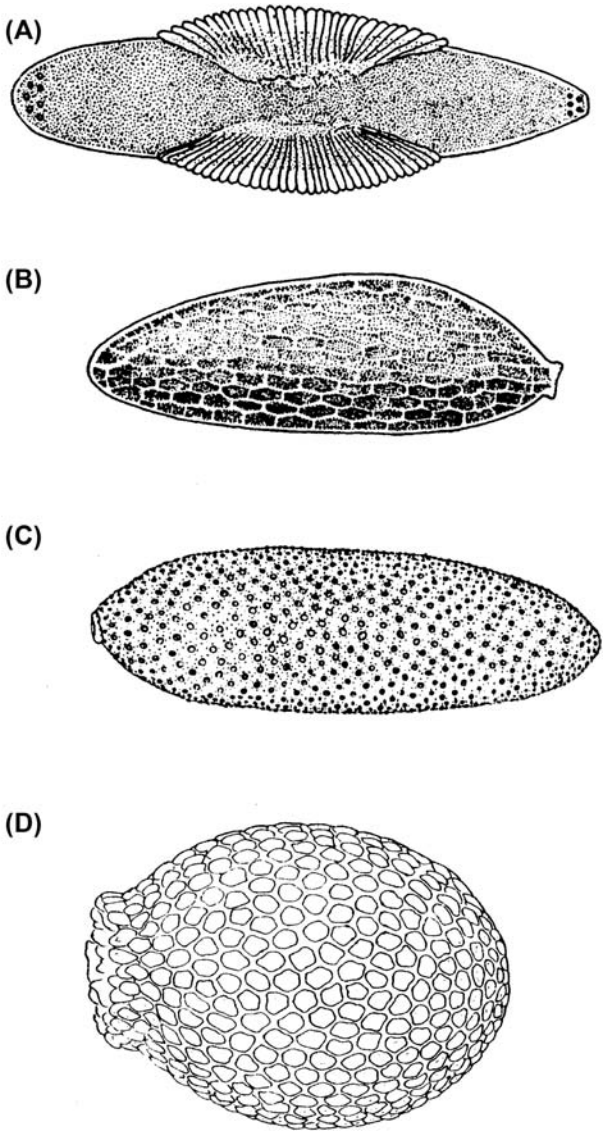


FIGURE 15.1 Eggs of mosquitoes, showing variations in shape and chorionic sculpturing. (A) *Anopheles*. (B) *Culex*. (C) *Aedes aegypti*. (D) *Toxorhynchites brevipalpis*. *A and B from Ross, 1947; C and D from Harbach and Knight, 1980.*

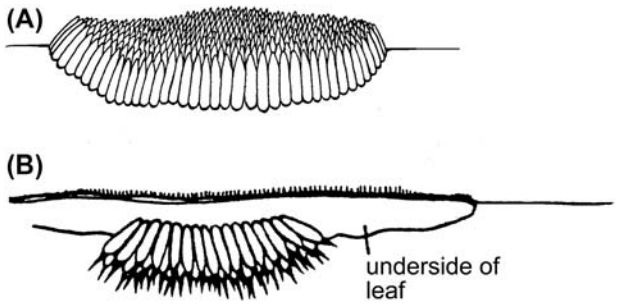


FIGURE 15.2 Mosquito egg rafts and clusters. (A) Floating egg raft of *Culex restuans* (Ross, 1947). (B) submerged egg cluster of *Mansonia*, attached to underside of floating leaf. (Gordon and Lavoipierre, 1962).

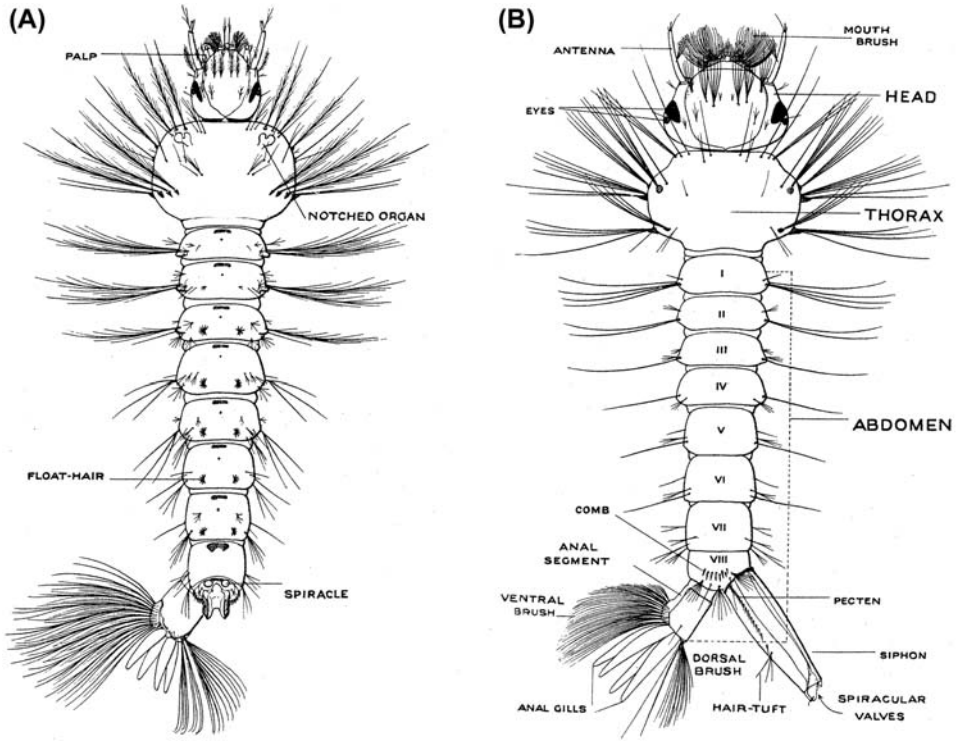


FIGURE 15.3 External anatomy of mosquito larvae, dorsal view, with anal segment and siphon at posterior end rotated to provide better view. (A) Anopheline form (*Anopheles maculipennis*). (B) Culicine form (*Aedes cinereus*). (Marshall, 1938).

predatory larvae, the mandibles and/or maxillae are heavy and sharply toothed for seizing or holding prey. The thorax is wide, with three indistinct, legless segments.

The larval abdomen is narrower than the thorax, cylindrical, and composed of eight apparent segments, the second-to-last being a composite of segments 8 and 9. A pair of spiracles opens on the dorsal side of this segment. In culicines the spiracles open at the end of the **respiratory siphon**, an elongate air tube extending dorsally. The siphon of *Coquillettidia* and *Mansonia* is short, ending in a heavily sclerotized point with a dorsal sawlike edge used to pierce and remain lodged in plant tissue. In anophelines the siphon

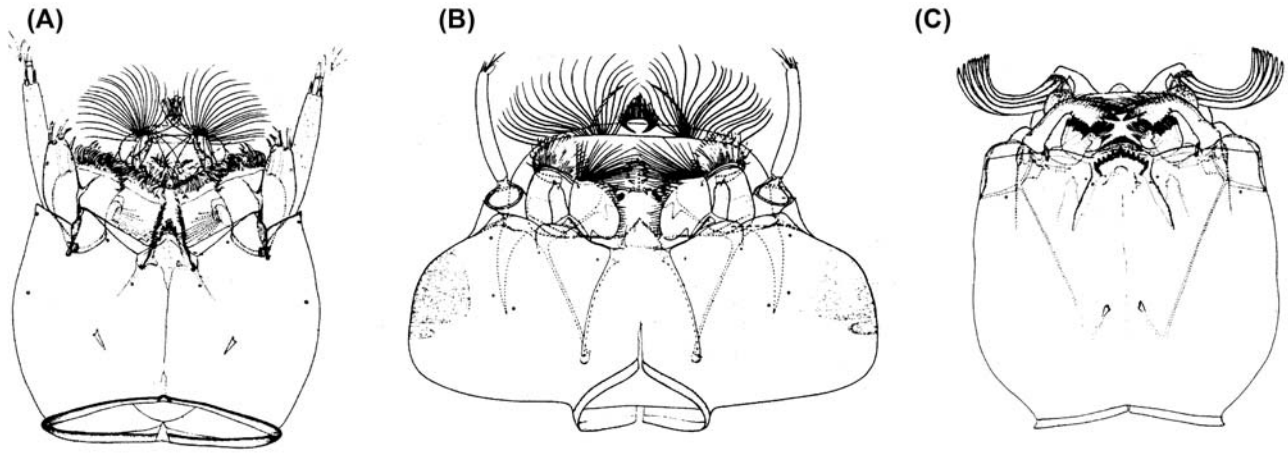


FIGURE 15.4 Heads of mosquito larvae, postero-ventral view. (A) Anopheline form (*Anopheles quadrimaculatus*). (B) typical culicine form (*Aedes fulvus pallens*). (C) toxorhynchitine form (*Toxorhynchites brevipalpus*). The lateral palatal brushes of most larvae are used to generate water currents for filter feeding; in *Toxorhynchites* they are modified for seizing prey. (Harbach and Knight, 1980).

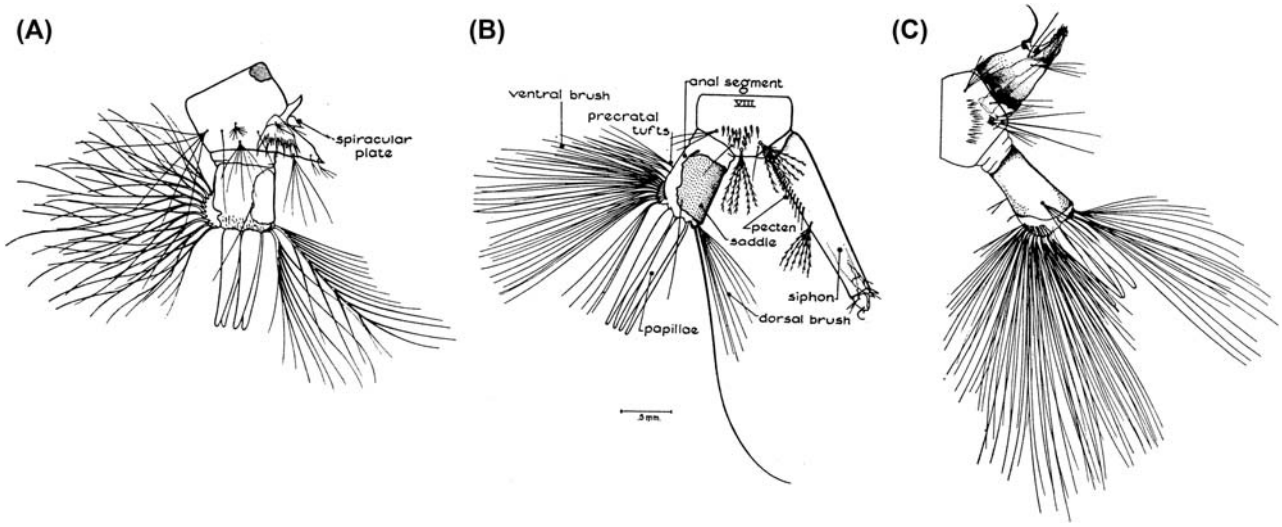


FIGURE 15.5 Terminal segments of mosquito larvae. (A) Anopheline form (*Anopheles earlei*), lacking a siphon. (B) typical culicine form (*Aedes fitchii*), showing elongate siphon. (C) specialized culicine form (*Coquillettidia perturbans*), showing short, stout siphon suited for piercing and clinging to plants. (Barr, 1958).

is lacking, and the spiracles are borne on a short spiracular plate. Segment 10, the anal segment, extends ventrally at an angle from the rest of the abdomen. It typically bears four anal papillae used primarily in osmoregulation. The terminal region of the larva bears several structures useful in identification (Fig. 15.5). They include comb scales on segment 8, pecten spines on the siphon, a saddle sclerite encircling the anal segment, and various tufts and brushes of setae. Some of these terminal structures apparently are used to groom the mouthparts when the larva bends its body around to form a loop.

Larval internal anatomy conforms to the general insect plan. The alimentary tract is almost straight, the only notable features being eight large gastric caeca at the junction of the foregut and midgut in the thorax and five Malpighian tubules at the midgut–hindgut junction. Because most of the cuticle is semitransparent, two large tracheal trunks are obvious, extending forward from the spiracles to the thorax.

Mosquito pupae, commonly known as **tumblers**, are comma-shaped, with the head and thorax fused to form a cephalothorax and the abdomen curled beneath it (Fig. 15.6). Projecting from the dorsal mesothorax is a pair of respiratory tubes, or **air trumpets**, through which the pupa obtains oxygen at the water surface. Within the cephalothorax, the developing appendages of the adult head and thorax usually can be seen coiled ventrally; they envelop an air pocket, the ventral air space that provides buoyancy to help maintain the pupa at the water surface when resting. At the end of the abdomen two broad paddles are attached to the eighth segment. The pupa can flex its abdominal segments, causing the paddles to flap downward, propelling it through the water when it is disturbed.

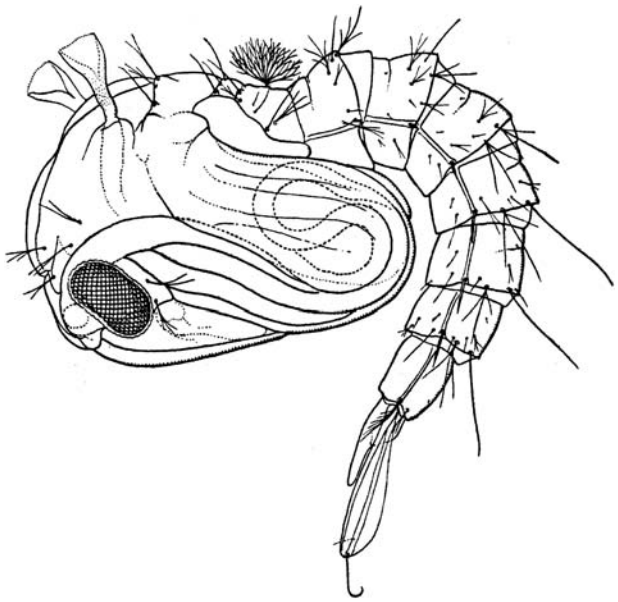


FIGURE 15.6 Mosquito pupa. Lateral view of *Anopheles gambiae* in resting position at water surface; presence of adult structures visible within pupal cuticle. (Smart, 1948).

Adult mosquitoes are slender, with thin legs and narrow, elongate wings (Fig. 15.7). The body surface is covered with scales, setae, and fine pile, creating the characteristic markings and colors of each species. The two compound eyes, each represented by 350–900 ommatidial lenses, wrap around the front and sides of the head. The antennae arise between the eyes, are long and filamentous, and are usually sexually dimorphic. In species in which sound is used to locate females in flight, the flagellum of the male antenna has whorls of much longer fibrillae,

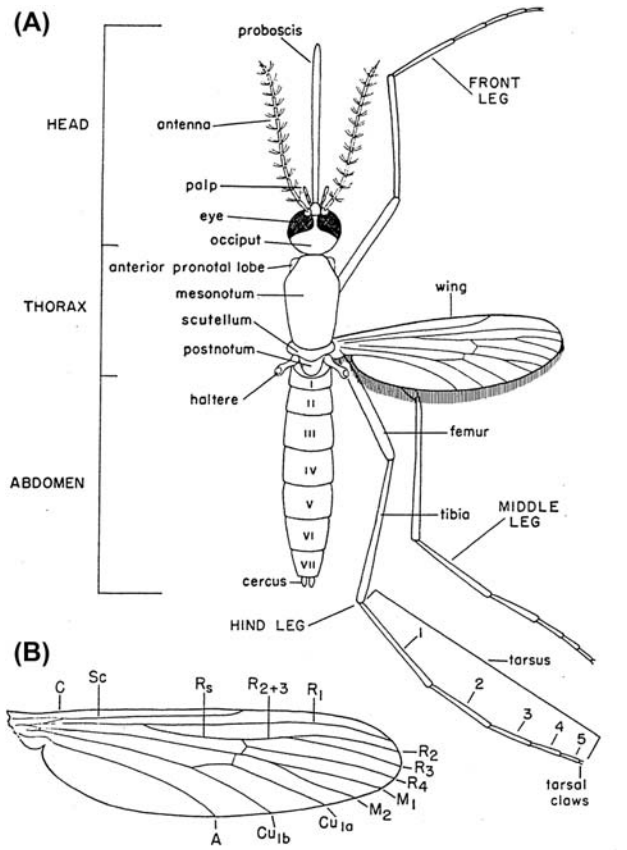


FIGURE 15.7 External anatomy of mosquito adult. (A) Generalized adult, dorsal view. (B) wing, showing typical venation and vein nomenclature. (Ross and Horsfall, 1965).

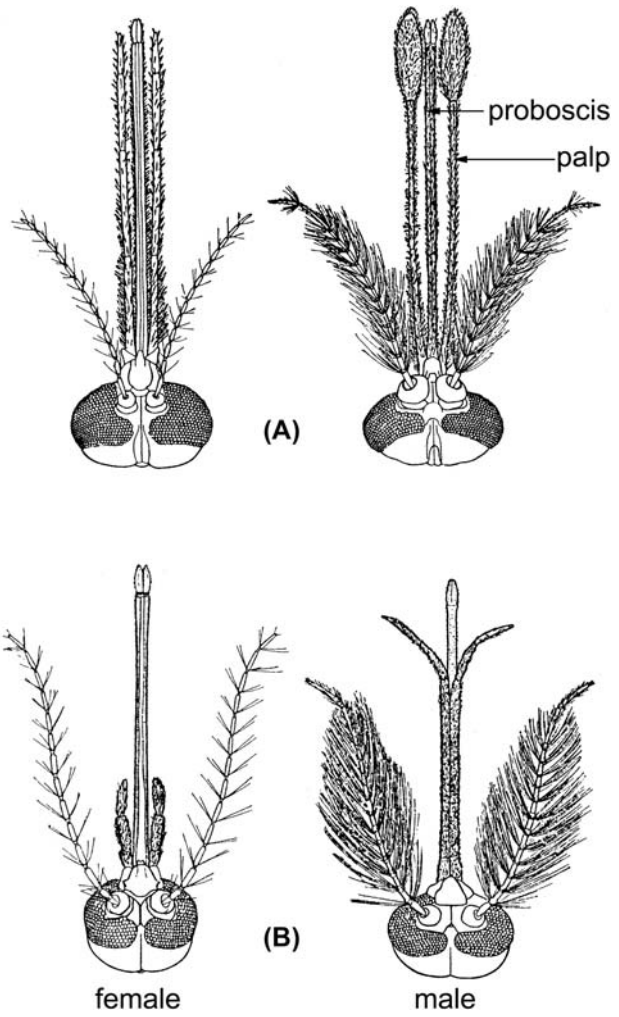


FIGURE 15.8 Heads of anopheline and culicine mosquitoes, females (left), males (right); males typically with plumose antennae. (A) *Anopheles* (anopheline), both males and females with palps about as long as the proboscis; male with plumose antennae and tips of palps broadened. (B) *Culex* (culicine), females typically with short palps and males with long, curved or brushlike palps. (Gordon and Lavoipierre, 1962).

giving it a plumose appearance (Fig. 15.8). The pedicel at the base of the antenna is a large globular structure that contains **Johnston's organ**, a mass of radially arranged mechanoreceptors that respond to vibrations of the flagellum induced by sound. In addition, to the long fibrillae, the antenna has a variety of sensory structures, including those for detecting host odors.

The mosquito proboscis is prominent, projecting anteriorly at least two-thirds the length of the abdomen. It consists of the basic complement of insect mouthparts: the labrum, paired mandibles, hypopharynx, paired maxillae, and labium. The first four structures have evolved into fine stylets, forming a tightly fitting fascicle that in females is used to penetrate host skin (Fig. 15.9). The fascicle is cradled within the groove of the large and conspicuous labium (Fig. 15.10), which comprises the bulk of the proboscis. The tip of the labium bears a pair of small taste-sensitive labella and a short, pointed ligula between them. The latter allows water and sugar solutions to make contact with the inner surfaces of the labella. Of the fascicle of stylets, the hypopharynx and mandibles are narrowly pointed at their tips, whereas the maxillae end in serrated blades. Both mandibles and maxillae puncture the skin and advance the fascicle into the host's tissue. A salivary channel runs the length of the hypopharynx, delivering saliva to the tissue during probing. The labrum is curled laterally to form a food canal for drawing the host's blood or a sugar solution up the proboscis. In males, and in females of non–blood-feeding species, the mandibles and maxillae have atrophied, so they cannot pierce skin. In both sexes of *Toxorhynchites*, the nonpiercing proboscis is curved downward (Fig. 15.11). Maxillary palps arise at the base of the proboscis and bear several kinds of sensilla. Though there are many exceptions, palps usually are short in female culicines but longer than the proboscis in most male culicines and in both sexes of anophelines (Fig. 15.8).

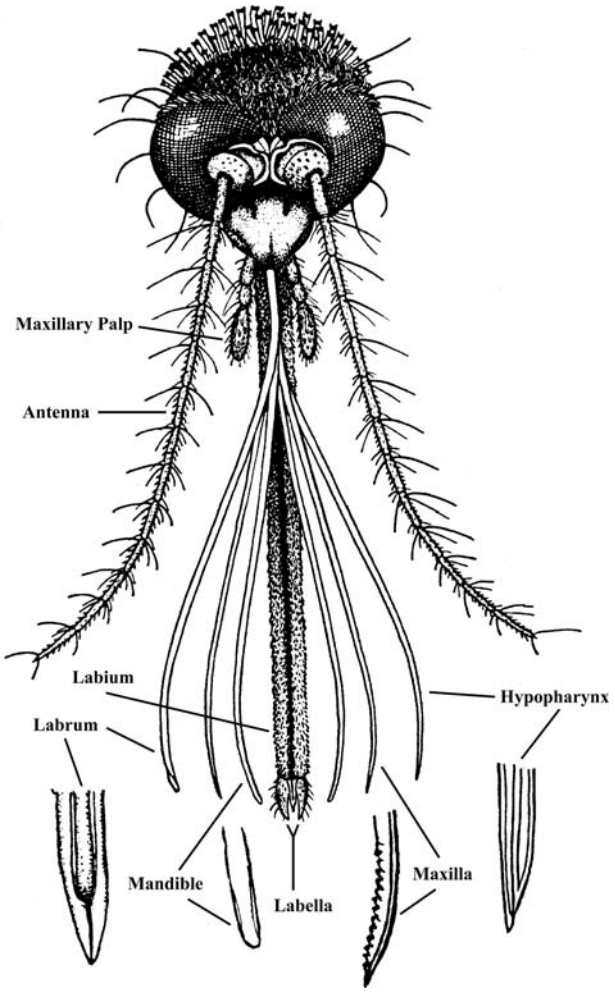


FIGURE 15.9 Mouthparts of adult female mosquito, showing labium, splayed stylets, and variations in structure of their tips. (Matheson, 1944).

The mosquito thorax forms a single, relatively rigid muscle-filled locomotor unit with obscured segmentation. The mesothorax and metathorax each has a pair of lateral spiracles. The slender legs are attached close together on the underside of the thorax by elongate, downward-projecting coxae; the tarsi are tipped with two claws and a central pad, the empodium. The wings are narrow, have a distinctive pattern of veins, and bear scales along the veins and the hind margin, the latter forming a fringe. The **halteres**, tiny modified hindwings used in flight control, are located right behind the insertions of the wings.

The abdomen is clearly segmented and capable of extensive expansion and some movement, owing to the membranous areas between each set of tergites and sternites. This allows for expansion of the abdominal wall to accommodate large blood and sugar meals and developing clutches of eggs. Abdominal segments 5−8 are progressively smaller so that the abdomen tapers toward the posterior end. Segment 9 is quite small and bears the cerci, the postgenital lobe of the female, and the claspers and other genitalic structures, or **terminalia**, of the male (Fig. 15.12). At emergence, the male genitalia are inverted. During the first hours of adulthood, segments 8 and 9 of males together rotate 180 degrees to reach the mature position. The complex and varied male genitalia provide a useful source of characters for species identification.

Located in the thorax are a pair of three-lobed salivary glands, whose ducts join anteriorly to form a common salivary duct that enters the hypopharynx (Fig. 15.13). In males, these glands produce saliva used only in sugar-feeding; in females, some portions are devoted to sugar-feeding and others to blood-feeding. The foregut, which begins in the head with the muscular cibarium and pharynx, pumps food up the labral food canal. The tubular esophagus extends

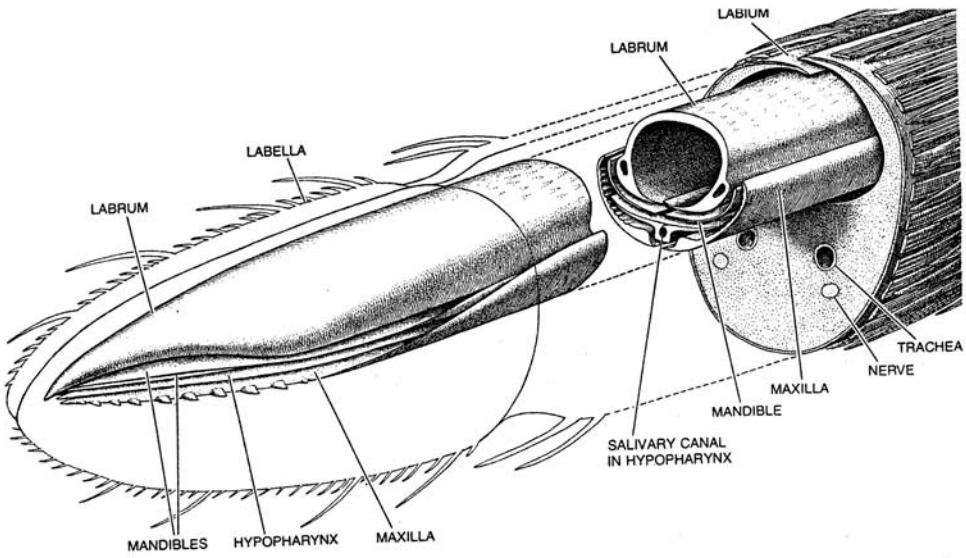


FIGURE 15.10 Fascicle of stylets of adult female mosquito. Mouthparts near tip of proboscis, showing natural arrangement of stylets in a single bundle, or fascicle, within a groove in the labium, which forms a sheath. *Jones, 1978; illustration by Tom Prentiss.*

FIGURE 15.11 *Toxorhynchites amboinensis*, adult male. The form of the palps and antennae in females and males is similar to that of other culicines; however, the proboscis of both sexes is bent downward at an angle of 90° or more. *Photograph by Woodbridge A. Foster.*

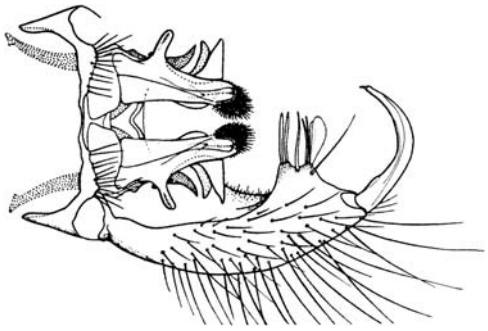


FIGURE 15.12 Male genitalia of *Culex quinquefasciatus*, showing principal copulatory structures used in male taxonomic identification. Gonocoxite and gonostylus on left (upper) side have been omitted. (Ross and Roberts, 1943).

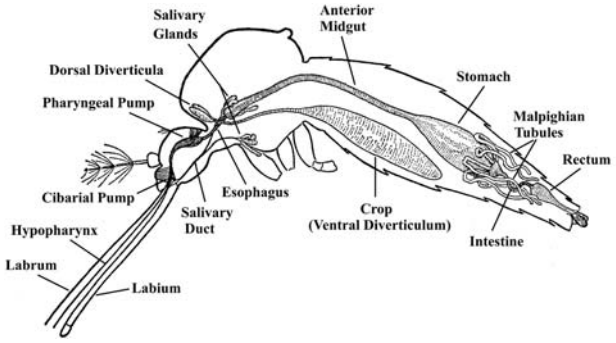


FIGURE 15.13 Digestive system of the adult mosquito. Semi-diagrammatic view of major structures, including the salivary glands, foregut-midgut junction, and rectum. (Snodgrass, 1959).

through the cervix, or neck, into the thorax. There it is modified to form three diverticula, including two small dorsal outpocketings and a large ventral crop; the crop extends through the thorax and expands to form a large sac in the abdomen. Imbibed sugar solutions are stored in these diverticula and pass, a little at a time, through the proventricular valve into the midgut. A bloodmeal, on the other hand, passes directly into the widened posterior midgut, or stomach (Fig. 15.13). There it becomes surrounded by a semipermeable, saclike **peritrophic membrane** secreted by the midgut epithelium. The resulting blood bolus then is digested and absorbed. The pyloric valve separates the midgut from hindgut; five Malpighian tubules empty into the hindgut just beyond the valve in the pyloric chamber. The anterior portion of the hindgut is tubular and loosely coiled; the posterior part is enlarged to form a bulbous rectum with large papillae projecting into it. The papillae probably are involved in resorption of salt ions.

Paired gonads are located in the posterior third of the abdomen. The testes of males contain packets of sperm in various stages of maturation. A duct extending posteriorly from each testis widens to form a seminal vesicle, which stores mature sperm. The two seminal vesicles lie together and unite posteriorly to form the ejaculatory duct. Two large accessory glands open into this duct, which leads to the aedeagus. In females, the reproductive system consists of a pair of ovaries and accessory structures (Fig. 15.14). Each ovary includes a few dozen to over 200 polytrophic ovarioles, the egg-forming units. A duct from each ovary extends posteriorly, and the two unite to form a common oviduct, which connects to the gonopore via a genital chamber. Opening into the genital chamber by tiny ducts are one (in anophelines) to three (in most culicines) sperm-storing spermathecae, a small accessory gland, and the seminal bursa (lacking in most *Anopheles*), which receives semen from the male during mating.

LIFE HISTORY

The holometabolous life cycle of mosquitoes is completed in two different environments, one aquatic, the other terrestrial. The larvae and pupae develop in a wide range of aquatic habitats. These include temporary surface water (e.g., tidal pools in salt marshes, rain pools, and flood water), permanent surface water (e.g., pools, streams, swamps, and lakes), and diverse natural and artificial water-holding containers (e.g., tree holes, leaf axils, fruit husks, mollusk shells, drinking water pots, and discarded tires). An extensive analysis and classification of larval habitats have been presented by Bates (1949), Mattingly (1969), Laird (1988), and Service (1993). The only absolute requisite of all development sites is that they maintain at least a film of water for the duration of the larval and pupal periods. However, individual species tend to oviposit, and therefore develop, in sites with specific structural and chemical properties. Adults are much more mobile than the immatures, but they also tend to occupy characteristic resting, foraging, and overwintering habitats.

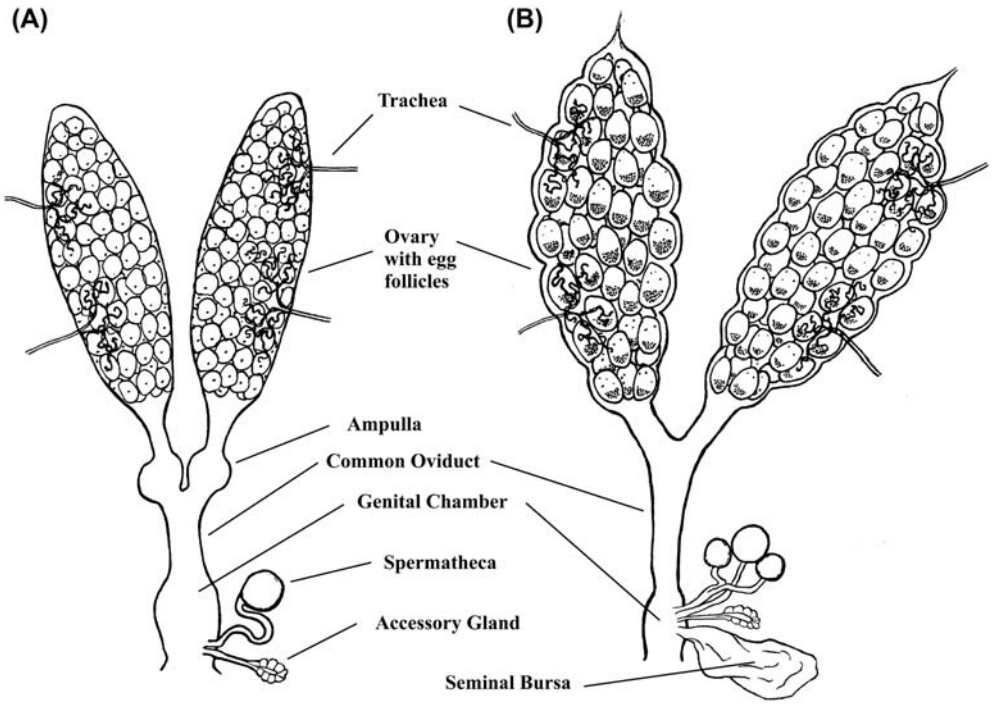


FIGURE 15.14 Female reproductive system of the mosquito. (A) Anopheline form, based on *Anopheles gambiae*. (B) Culicine form, based on *Aedes aegypti*. *Original by Woodbridge A. Foster.*

Mosquito eggs are laid either on or in water, or on solid substrates that are likely to become indundated. Females in the subfamilies Anophelinae and most of the Culicinae in the tribe Sabethini scatter their eggs individually on the water surface, whereas those in the tribe Aedini (e.g., *Aedes*, *Psorophora*, and *Haemagogus*) attach their eggs individually on a substrate that later will become inundated with water. Clumped eggs are laid in boat-like rafts on the water surface by several genera of Culicinae (e.g., *Culex*, *Culiseta*, *Coquillettidia*, *Uranotaenia*, *Armigeres*, and *Trichoprosopon*) or in a radial cluster attached underwater to vegetation in the case of *Mansonia*.

At first the eggs are white, but most turn dark within hours as the chorion tans. In lowland tropics, subtropics, and summers of some temperate regions, eggs usually complete embryonic development within 2−3 days after being laid, but may take up to a week or longer in cool climates. Larvae hatch soon after embryonation in species that lay their eggs directly in water, including all Anophelinae and most tribes of Culicinae. The best known exceptions are members of the genera *Aedes*, and other Aedini that typically develop in temporary water. Their eggs are laid on solid substrates out of water, and the larvae within them remain quiescent until inundated. The eggs can tolerate periods of cold and desiccation and may remain viable for years. Hatching usually occurs at warm temperatures after the eggs have been submerged and microbial activity has caused the oxygen level in the water to drop.

Depending on the species and particular conditions of the water, most mosquito **larvae** spend most of their time either at the water surface or at the bottom of the water column, coming to the surface for air only occasionally, or not at all. At ideal conditions of food and temperature (26−28°C), the entire larval phase of *Aedes aegypti*, a tropical and subtropical mosquito, may last as few as 5−6 days. The first three instars are completed in about 1 day each and the fourth lasts about 3 days. In males these periods are slightly shorter, so the males pupate about 1 day earlier than females. Larvae of many species grow even faster, as when the water is heated by direct sunlight, whereas others develop slowly. *Toxorhynchites* and *Wyeomyia* spp. usually take 2−3 weeks even under ideal conditions. At cooler temperatures, or when food is scarce, growth becomes slower and can practically cease, with larvae remaining alive for months. Larvae of some species that inhabit high latitudes or high altitudes, or that develop in the early spring in temperate regions, have growth thresholds close to freezing and can tolerate even temporary entrapment in solid ice. This is typical of the snow pool *Aedes* spp. and of mosquitoes that overwinter as larvae, such as *Wyeomyia smithii* in pitcher plants and *Orthopodomyia alba* in tree holes.

The **pupa** spends nearly all of its time at the water surface. By the time it has molted to form a pharate adult within the pupal cuticle, it is very dark. In warm water the entire pupal stage typically lasts about 2 days in both sexes.

In some mosquitoes, such as *Toxorhynchites* and *Wyeomyia* spp., the shortest pupal periods may be 5−6 days. In all species the pupal period lasts longer at lower temperatures.

Adult males tend to emerge earlier than females, because of their shorter larval growth periods. As **adult emergence** approaches, the pupa remains stationary at the water surface, and the abdomen gradually straightens over 10−15 min. The adult emerges from the pupal cuticle by ingesting air, causing the cephalothorax to split and the adult to rise up out of the cuticle and stand on the water surface. The entire process takes only a few minutes. The newly emerged adult is capable of short flights a few minutes later but cannot sustain long flights for many hours, until after the cuticle becomes fully sclerotized. Lipids and glycogen, carried over from larval reserves, provide sufficient energy for a few days of flight and survival.

It is typically during the first 3−5 days of adult life that both sexes obtain **sugar** from plant **nectar** or **honeydew**, become sexually mature, and then mate. In some species (e.g., *Culiseta inornata*, *Wyeomyia smithii*, and *Deinocerites cancer*) sexual maturation is complete at the time of emergence or only a few hours later, and mating occurs almost immediately. Mosquitoes typically first feed on sugar to obtain enough energy for sexual maturation and for the flight necessary for mating, dispersal, and finding vertebrate blood. Natural sugar is taken repeatedly throughout adult life by both sexes of most species. Females typically mate only once. Males can inseminate several females before their supplies of mature sperm and accessory gland secretion become depleted. The semen supply is replenished in a few days.

Amorphous masses of fat body line the inner walls of the abdomen. The fat body synthesizes and stores both glycogen for flight and lipids for maintenance, using the digestive products of sugar and bloodmeals. Glycogen is stored also in the fibrillar flight muscles of the thorax, serving as a source of energy for immediate flight if the sugars in the crop and hemolymph have been exhausted.

Only females feed on vertebrate blood. In most mosquitoes, ingestion and digestion of a bloodmeal initiate **egg development** by stimulating a cascade of hormones from the brain and ovaries. The large amount of protein contained in hemoglobin and the blood serum provides the amino acids for synthesizing **vitellogenin**, the proteinaceous precursor of egg yolk. The protein also serves as the substrate for building lipid and glycogen, which contribute both to egg yolk and to the maternal energy reserves used for survival and flight. A bloodmeal will stimulate egg development only if it is sufficiently large and if the female's ovarian follicles have reached the resting stage, at which point they are considered to be **gonoactive**. If a female has had poor larval nutrition, the follicles may not have reached the resting stage, and she will be unable to develop any eggs until having ingested sugar or a preliminary bloodmeal. Such a **gonoinactive** female, needing food to bring the ovarian follicles to the resting stage, is sometimes said to be "pre-gravid." Details of the hormonal control of these processes are discussed by Brown and Lea (1990), Hagedorn (1994, 1996), and Klowden (1996).

In most species, females are **anautogenous**, the egg follicles remaining in the resting stage until a bloodmeal is taken. Following each bloodmeal, the female develops one mature clutch of eggs, exhibiting what is known as gonotrophic concordance. However, females of **autogenous** species or populations can develop eggs without a bloodmeal; among these there are obligate and facultative types. A facultatively autogenous female typically develops only the first clutch of eggs without blood; she does so only if she emerges with sufficient reserves and cannot readily find blood. Thereafter, a bloodmeal is required for each gonotrophic cycle. Species that are obligately autogenous have atrophied feeding stylets, never take blood, and subsist entirely on their larval reserves and plant sugar. Autogeny has been reviewed by O'Meara (1985). At the other extreme, there are some anautogenous species that take blood not only at the beginning of each gonotrophic cycle but also once or twice during egg development. These supplementary bloodmeals can provide extra energy, acting as a substitute for sugar (e.g., domestic *Aedes aegypti*, *Anopheles gambiae*).

Ordinarily, all eggs develop synchronously and become mature in 2−5 days after blood-feeding at favorable temperatures. During this time, the most advanced follicle within each ovariole passes through a series of five easily observed **physiological stages** (Fig. 15.15), originally described by Christophers (1911) and concisely summarized by Clements (1992). These stages comprise four physiological phases. The follicles develop synchronously, beginning with the **previtellogenic phase** (stages G through II). The follicle typically stops growing at stage IIa or IIb, the **resting stage**, until the female takes a bloodmeal. Within hours of blood-feeding, the follicle enters the **initiation phase** (stage IIIa), when yolk (vitellogenin) synthesis by the fat body begins, followed by the **trophic phase** (stages IIIb and IV), the main period of yolk incorporation and follicle growth. In the **post-trophic phase**, the egg has reached its mature shape, and the **chorion**, or egg shell, is formed. The eggs are then ready to be oviposited.

By this time, the next follicle in each ovariole either has progressed to stage I or already has reached the resting stage (stage II). In the first case, it awaits oviposition before developing to the resting stage. Once oviposition has occurred and these new follicles are in the resting stage, the next bloodmeal is necessary for subsequent follicle development. The entire cycle of egg production, from bloodmeal through oviposition, is the **gonotrophic cycle**. A female typically undergoes many such cycles, each starting

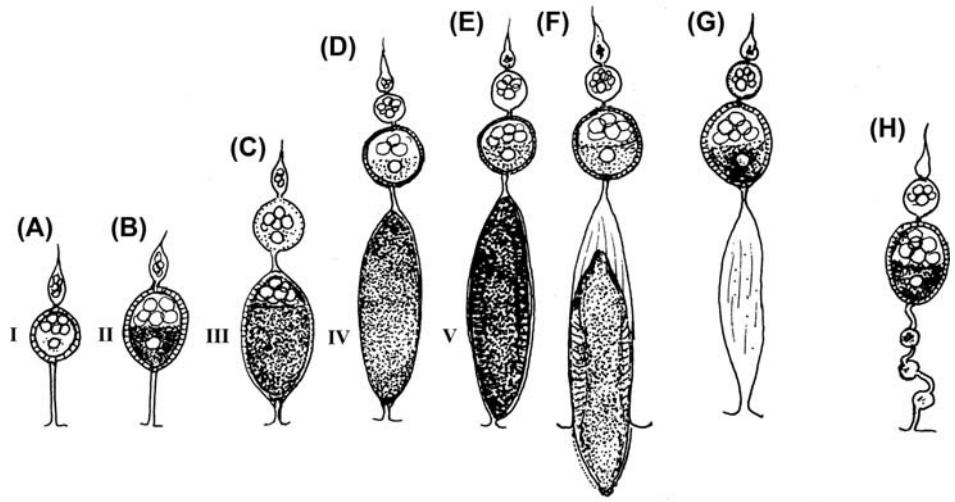


FIGURE 15.15 Egg follicle development in mosquitoes, showing stages in the development of an egg follicle within a single ovariole of the ovary. (A)–(E), Stages I–V of Christophers in an *Anopheles* female. After oviposition (F), the ovariole stalk remains swollen for awhile, called the "sac stage" (G). Note: formerly, the sac was thought to shrink to a dilatation. Now it is believed that the dilatations (three of them shown in H) generally form from follicles that fail to develop into eggs after a bloodmeal and are then resorbed. (World Health Organization, 1975).

with a bloodmeal, an important fact when considering how most mosquito-borne pathogens are transmitted.

The number of ovarioles that produce mature eggs depends on the sizes of the female body, energy reserves, and bloodmeal. The follicles that do not pass beyond stage II degenerate. When most or all the ovarioles contain mature eggs, the ovaries may occupy nearly the entire volume of the distended abdomen. The gravid female fertilizes one egg at a time, as each passes down the oviduct to be oviposited on the water or a damp substrate.

When all eggs have been fertilized and expelled during oviposition, the ovaries return to their pretrophic size, but the tracheae on the ovaries, which had been tightly coiled as **tracheal skeins** before the eggs developed, become stretched and straightened. In *Anopheles* spp., a swelling at the base of each lateral oviduct, the **ampulla**, becomes permanently stretched during the first oviposition. These signs serve to distinguish **parous** females, those that have completed at least one gonotrophic cycle, from **nulliparous** females, those that have not.

The number of completed **gonotrophic cycles** also can be determined. According to current interpretations, each ovariole ovulating a mature egg is left with an egg sac, which becomes reduced to a zone of granules in the calyx, the ovariole's connection to the lateral oviduct. Furthermore, a dilatation is formed in the stalk of each ovariole where a follicle has degenerated after a bloodmeal, instead of developing into an egg (Fig. 15.15). Thus, a count of the maximum numbers, per ovariole, of dilatations in the stalk and zones of granules in the calyx yields an estimate of the number of gonotrophic cycles completed. This **physiological age grading** can provide the medical entomologist with valuable information on the age of individuals and the age structure of a mosquito population. Details of these processes and their interpretation and application are given by Detinova (1962), Sokolova (1994), Fox and Brust (1994), and Hoc (1996).

Univoltine mosquito species complete only one generation per year. This occurs either if the developmental time is slow in relation to the season favorable for development or if the life cycle includes an obligate form of diapause, a compulsory phase of arrested development. **Bivoltine** and **multivoltine** species can complete two or more generations, respectively, during each breeding season, but the number actually completed may depend on temperature, available larval habitats, or available hosts. Mosquitoes pass through the winter or dry season as eggs, larvae, or adults, depending on the species and the climate. In cold climates, overwintering takes place in a state of diapause.

BEHAVIOR AND ECOLOGY

Eggs that are laid on or in water generally are not resistant to desiccation and hatch shortly after embryonation, provided that they are wet and not too cold. This is typical of *Anopheles*, *Culex*, *Culiseta*, and *Toxorhynchites* spp. *Aedes*, *Psorophora*, and *Haemagogus* eggs, on the other hand, typically are laid on damp substrates, display great resistance to desiccation, and remain quiescent for months or years after embyrogenesis until they receive a hatching stimulus. Sometimes moisture by itself is sufficient to induce hatching. Usually, however, the requisite stimulus is a reduction of dissolved oxygen in the water caused by microbial activity and decomposition of organic matter.

Among quiescent eggs that are eventually submerged, only a portion of a single egg clutch may hatch during any one inundation, resulting in **installment hatching**. This apparently is the combined result of intrinsic variations among eggs in their hatching-stimulus thresholds and of local variations in microbial activity, causing differences in oxygen tension around the eggs. Even during a single inundation, hatching may not occur all at once but over a period of many days. In both quiescent and non-quiescent types of eggs, physical agitation of the water sometimes serves as a hatching stimulus.

When an egg is ready to hatch, the first-instar larva uses a dorsal hatching spine on its head, the egg breaker or egg burster, to apply pressure to a preformed weakness in the chorion. This causes the chorion to pop open at one end, and the larva wriggles free. Because the eggs of *Culex*, *Culiseta*, and *Coquillettidia* usually stand vertically on the water surface in rafts, the larvae develop inside them with their anterior end oriented downward and hatch directly into the water.

Mosquito larvae are not buoyant and must, at rest, be suspended at the surface by special hairs and spiracular structures that cling to the surface tension while obtaining oxygen directly from the air. Culicinae typically migrate up and down in the water column, so they occur both at the surface and at the bottom of a body of water, depending on the availability of food. At the surface the tip of the siphon opens above the surface film, as the larvae hang diagonally downward most of the time (Fig. 15.16B). *Mansonia*, *Coquillettidia*, and some *Mimomyia* spp. are unusual in remaining submerged throughout larval and pupal development, with their siphons embedded in the tissues of aquatic plants from which they derive some oxygen (Fig. 15.16C). Mosquitoes that live in water-filled leaf axils (e.g., *Wyeomyia* spp.) are adept at flattening themselves against vertical surfaces and maneuvering in narrow spaces. Anopheline larvae spend most of their time at the water surface, often close to vegetation or floating material. They are able to remain suspended horizontally at the surface (Fig. 15.16A) due to pairs of dorsal palmate setae (float hairs) on several abdominal segments (Fig. 15.3A).

Larvae propel themselves by a back-and-forth lashing movement of the abdomen. Anopheline larvae usually swim horizontally at the surface film. When larvae of typical culicine mosquitoes are feeding below the surface, they periodically swim actively back to the surface to obtain oxygen. However, in many microenvironments dissolved oxygen also is absorbed from the water through the cuticle, requiring only infrequent trips to the surface by some species.

Mosquito larvae feed on a variety of organic detritus, suspended material, and small organisms in their aquatic habitats. The organisms include bacteria, protists, fungi, algae, microinvertebrates, and small macroinvertebrates in the case of predatory species. The organic detritus usually consists of dead plant material and dead macroinvertebrates.

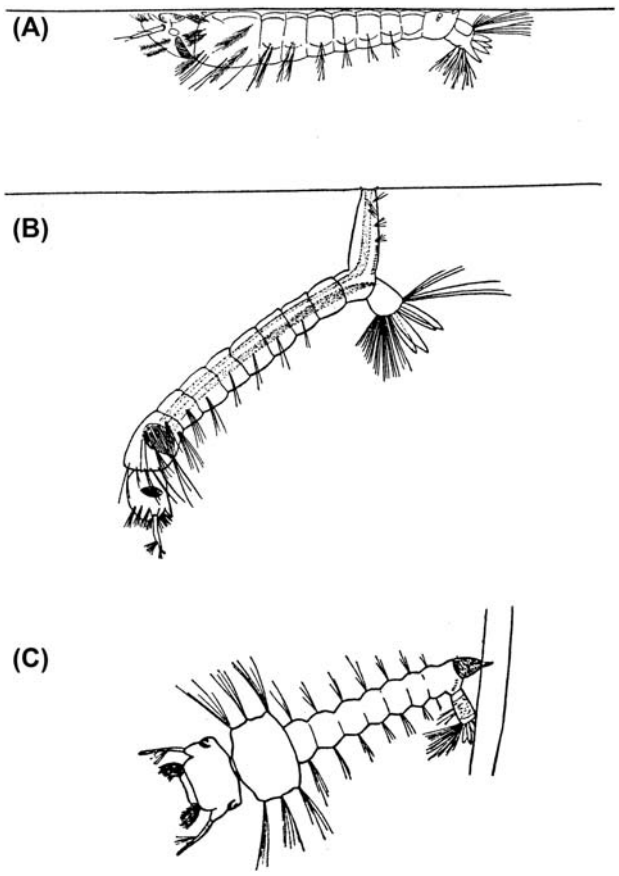


FIGURE 15.16 Resting and feeding positions of mosquito larvae. (A) *Anopheles*, showing horizontal position at the water surface, dorsal-side up; larva has rotated its head 180° so that the ventral side of the head is uppermost and the mouthparts are applied to the water's surface for filter feeding on floating detritus. (B) *Culex*, showing typical diagonal position while suspended from water surface. (C) *Mansonia*, attached to submerged part of aquatic plant (stem or root) by its siphon. *A and B from Ross, 1947; C from Gordon and Lavoipierre, 1962.*

They collect these food items in five basic ways: filtering, gathering, scraping, shredding, and preying. **Filterers** generate water currents with their lateral palatal brushes on the labrum, drawing suspended particles though fine combs where they are collected and directed to the mouth. **Gatherers** use their mouthparts in a similar manner, but only after stirring up the particles from solid surfaces. **Scrapers** obtain food by scraping it off solid surfaces, whereas **shredders** gnaw, chew, and bite off pieces of organic matter. **Predators** grasp insects and other small, mobile prey in their large and sharp mandibles or maxillae (e.g., some *Psorophora* spp.) or with long, curved palatal brushes (e.g., *Toxorhynchites*) (Figs. 15.4C and 15.17).

Most species use more than one of the above techniques. *Anopheles* primarily filter-feed at the water surface

FIGURE 15.17 *Toxorhynchites amboinensis* larva feeding on larva of *Culex pipiens*. This predaceous species has been used in biological control trials and naturally exerts a damping effect on populations of pest and vector mosquitoes. *Photograph by Woodbridge A. Foster.*

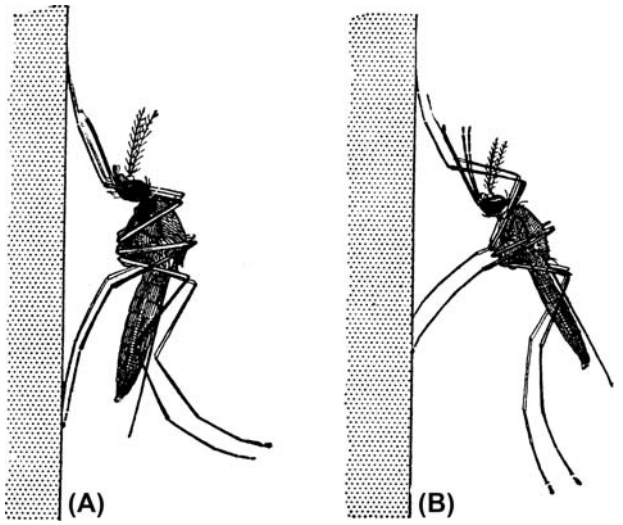


FIGURE 15.18 Comparison of typical positions of mosquito adults resting on a vertical surface. (A) Culicine. (B) anopheline. Culicines typically hold the abdomen parallel to the substrate or pointed toward it, and the proboscis and abdomen form an angle. Anophelines characteristically tilt the body at a sharp angle to the substrate, with the abdomen pointing away from it, and with the proboscis and abdomen in line. (Marshall, 1938).

by rotating their heads 180 degrees so that the oral opening becomes dorsal. Many *Aedes* and *Culex*, on the other hand, filter-feed near the surface but also gather, scrape, or shred organic matter at the bottom, depending on food availability. *Coquillettidia* and *Mansonia*, which are anchored to submerged vegetation, use a combination of filter-feeding, gathering, and scraping techniques within their immediate surroundings. Larval feeding has been reviewed by Merritt et al. (1992).

Mosquito pupae normally remain motionless at the water surface with the tips of their thoracic air trumpets in contact with the air. Like larvae, they dive when disturbed, propelling themselves with their caudal paddles by extending the abdomen, then snapping it back inward toward the cephalothorax. Pupae of most species are buoyant, due to the ventral air space beneath the cephalothorax, and rise to the surface without swimming. They remain submerged by repeatedly swimming downward or by wedging or lodging themselves under debris. After sufficient submergence time, they lose their buoyancy as their air supply dwindles, and they must swim actively to the surface. Pupae of a few mosquitoes (e.g., *Limatus* spp.) are never buoyant and can keep from sinking only by clinging to the surface film, much as most mosquito larvae do. The plant-piercing pupae of *Mansonia* and *Coquillettidia* spp. do not rise to the water surface until they release their attachment to plants when ready for adult emergence.

On **adult emergence** from the pupal stage, adults typically seek shelter in vegetation, cavities, and other resting sites, where they remain except during periods of activity. When resting, they typically position themselves head-up on vertical surfaces, with forelegs and midlegs on the substrate and hindlegs raised (Fig. 15.18). Culicines hold the abdomen in various positions, but the proboscis is always at an angle to it (Fig. 15.18A). Anophelines, on the other hand, hold the proboscis and abdomen in line, oblique to the substrate (Fig. 15.18B). This distinctive position also is apparent during feeding. While at rest, adults perform various stereotyped grooming movements and frequently wave their hindlegs.

Dispersal and foraging may occur only a few dozen meters from their larval habitats. Most species fly less than 2 km in a lifetime. Such ranges are typical of domestic species and result from random elements in their repeated foraging flights for mates, sugar sources, hosts, resting sites, and oviposition sites. Other species enter a specific dispersal mode that is wind-assisted or light-directed and carries them dozens or even hundreds of kilometers from their origins. These flights are most obvious following massive adult emergences of species with quiescent eggs, such as the salt-marsh mosquito *Aedes taeniorhynchus* after high tides, and floodwater mosquitoes such as *Aedes vexans* in bottomlands after heavy rain. The average dispersal distance of such broods is difficult to determine accurately,

because efforts to recapture marked specimens must be made over vast areas, and relatively few are caught. Some salt-marsh species and African anophelines must make extended, round-trip flights to complete their gonotrophic cycles when development sites and blood-feeding sites are many kilometers apart.

Average flight speed also is difficult to determine under natural conditions. *Aedes aegypti* can fly upwind at air speeds up to 5.4 km/hr, while other species in a dispersing mode are estimated to fly much faster. However, their ground speed drops nearly to zero as headwind velocities approach their maximum air speed. Thus mosquitoes tend to avoid flight under windy conditions, except when assisted by a tailwind. Mosquito dispersal in its various forms has been reviewed by Service (1997).

Each mosquito species has a characteristic pattern of adult **diel activity**, under the control of an endogenous circadian rhythm that is entrained by the daily light-dark cycle. Generally one or two **flight periods** occur each 24 hours, characterized as being diurnal, nocturnal, or crepuscular (dawn and dusk). During these periods, both sexes will take flight without external cues other than ambient light. Mosquitoes likely engage in a generalized search pattern during **foraging flights**, then respond to specific stimuli associated with either mating sites, sugar sources, hosts, or oviposition sites as they encounter them, depending on their needs.

Mosquito species vary in the habitats where they forage for mates and food. Some fly over varied terrain; others tend to be active in either wooded or open areas; still others perform all activities close to larval and resting sites. There is some evidence that adult females become familiar with local habitats that have provided either food or oviposition sites in the past and tend to return there, based on experience.

Field studies and experiments indicate that some species are guided in their foraging flights by specific visual features along the horizon, or they fly along the edges of tree stands bordering open terrain. Others apparently simply fly crosswind or downwind, depending on wind speed. When they can see likely sites that supply needed resources, they alter their flight paths to move directly toward the object. In the absence of visual information, if they detect salient odors at a distance, they fly upwind within a downwind-drifting odor plume and then use those and other cues at close range until they arrive at their source. Drinking water, imbibed from the surface of moist substrates, presumably is located similarly, with elevated humidity as an indicator of its proximity. Nectar meals and blood meals also provide water.

Mating usually takes place a few days after adult emergence. The males typically form flight swarms at particular times at swarm markers (prominent objects or other contrasting features of the environment). Each male follows a looping flight path over the marker. In species such as *Aedes aegypti* and *Ae. albopictus*, the females' preferred host serves as the swarm marker. Females probably use the same visual or host-chemical swarm markers that males do, to locate their specific meeting place, but there is some evidence that swarming males also may release a volatile pheromone that attracts females from a distance. When a female enters a swarm, males detect the characteristic frequency of her wing beat and her position with their plumose antennae and Johnston's organs and fly toward her. Her fundamental flight tone varies from about 150 to 600 Hz, depending on the temperature and her size and species. It is about 100−250 Hz lower than the males' flight sound. Each sex can detect the other's audible flight tone or one of the ultrasonic harmonics of it, and at close range adjust these frequencies so that they converge. These duets and their mutual sonic convergence appear to be necessary for successful mating. Swarms usually are species-specific, but mixed swarms do occur; hybrid matings are rare. Interspecific pairings may be avoided either by the lack of frequency-matching or by the recognition of species-specific contact pheromones.

Successful copulation typically occurs only after the duetting and close-range maneuvering, after which the male orients his body with respect to her, venter-to-venter, clasping her genitalia with his. Even then, the female may reject the male by using her genitalia to make full copulation and intromission impossible. If there is mutual acceptance, the couple drifts or flies from the swarm, often shifting to an end-to-end position, with the female flying forward and the male facing backward, clinging to her only by his genitalia. Mating may be completed in the air or on vegetation. Copulation lasts from 12 seconds to several minutes.

There are many exceptions to this standard method of mating. Males of *Deinocerites* and *Opifex* guard pupae at the water surface and mate with the females as they emerge. Males of *Culiseta inornata* remain at the emergence site for long periods and locate newly emerged females at random while crawling about, recognizing them by a specific contact pheromone on their legs. A male *Eretmapodites chrysogaster* will follow a female in tandem flight to a host, wait beside her while she takes a bloodmeal, and then will copulate when she is finished. Males of the sabethine genera *Sabethes*, *Limatus*, *Wyeomyia*, *Runchomyia*, *and Topomyia* locate females at rest on vines, sticks, and tree trunks, and most perform a variety of leg-waving and genitalic courtship rituals on the substrate before insemination. Several aspects of mosquito mating behavior have been reviewed by Downes (1969). Lees et al. (2014), a lead review followed by a series of papers in a special issue of Acta Tropica, together provide a comprehensive summary of male biology and behavior, with emphasis on their relevance to genetic control.

During **copulation** the male deposits a mixture of sperm and accessory gland secretion in the female's seminal bursa or genital chamber. The semen often produces a distinct seminal mass in the bursa or genital chamber, sometimes called a **mating plug**. It disappears in 1−2 days and therefore does not, by itself, prevent subsequent insemination. Within an hour the sperm move into the spermathecae where they are stored. At least in culicine mosquitoes, the swollen bursa itself and, later, substances in the male accessory gland collectively called **matrone**, or MAGS, cause the female to become unreceptive to males. Substances in the accessory fluid also can affect feeding behavior and promote egg development and oviposition. A single insemination usually is sufficient for the life of a female. Most evidence indicates that females only rarely receive sperm from more than one male.

Adult mosquitoes of both sexes of most species regularly feed on plant sugar throughout life, but only females feed on vertebrate blood. Water presumably is taken from the surface of moist substrates as well as in sugar meals and bloodmeals. Field studies and experiments indicate that some species are guided in their foraging flights by specific visual features along the horizon, or they fly along the edges of tree stands bordering open terrain. Others apparently simply fly crosswind or downwind, depending on wind speed. When they can see likely sources of sugar or blood, they alter their flight paths to move directly toward the object. If they detect odors of flowers or hosts in the absence of visual information about a food source, they turn to fly upwind within the downwind drifting odor plume, eventually arriving at the odor source.

Sugar-feeding starts soon after emergence, usually before females begin responding to host stimuli. Sugar is taken frequently by both sexes throughout adult life, both between and during gonotrophic cycles. Females of some domestic species may take sugar infrequently or never (e.g., *Aedes aegypti* and *Anopheles gambiae* in some localities); they typically live in close association with their hosts and utilize blood for both energy and reproduction. **Sugar sources** include floral and extrafloral nectaries, homopteran honeydew, spoiled or damaged fruit, tree sap, and damaged or even undamaged plant leaves and stems. *Malaya* spp. solicit regurgitated nectar and honeydew from ants.

Nectar and honeydew are the most important sugar sources. They contain not only sugar but also amino acids; these are insufficient to initiate egg development but probably promote longevity. Most mosquitoes obtain sugar from a variety of plant species that provide accessible nectar; others seem to be fairly specific in their choices, and a few are important pollinators of particular plant species whose flowers they visit. Mosquitoes generally feed from fragrant, light-colored, clustered flowers with short corollas that allow easy access to the nectar.

Details of sugar-feeding biology have been reviewed by Foster (1995). The fragrances serving as mosquito attractants commonly include blends of several forms of terpenoids and benzenoids, among other compounds (Nyasembe and Torto, 2013).

Blood-feeding by female mosquitoes rarely begins until at least 1−3 days after adult emergence, and often not until after mating and sugar-feeding. Their hosts include all classes of vertebrates: mammals, birds, reptiles, amphibians, and even amphibious fishes, earthworms, and leeches. They have been reported to take hemolymph from other insects, but perhaps this occurs only when the insects have been contaminated with vertebrate odors.

Host specificity and **host preference** vary widely. Some species feed almost entirely on members of one genus of animal, others opportunistically attack members of two or three vertebrate classes. Bloodmeal identification methods, used to determine a mosquito's host, have been reviewed by Washino and Tempelis (1983) and Clements (1999). The immunological methods have been replaced largely by DNA profiling techniques, reviewed by Kent (2009). Host specificity is a function of both the mosquito's innate host preference and the hosts available to the mosquito when and where it is active. Some species forage over a broad range of habitats. Others are active principally in either wooded or open areas or remain close to sites of larval development or adult resting. Still other species attack their hosts in rather narrowly defined zones within a habitat. For example, within tropical forests, different species feed in different vertical strata: ground level, intermediate levels, or just below the leaf canopy.

Host-finding behavior in mosquitoes usually involves the use of volatile chemicals to locate vertebrate hosts. Carbon dioxide, lactic acid, and octenol are among the most widely documented host attractants. Other skin emanations also are known to be important, because odors from live hosts are always more attractive than any combination of those three chemicals in a warm, humid airstream. Several fatty acids produced by the normal bacterial flora of the skin are particularly effective in attracting *Anopheles gambiae* to human feet. Mixtures of these fatty acids and other compounds probably play a major role in attracting most mosquitoes (see Smallegange and Takken, 2010). Subtle differences in these odors of different host species and even different individuals undoubtedly play a role in host preference. These odors commonly have a combined effective range of 7−30 m, but up to 60 m for some species.

Vision also is important in orienting to hosts, particularly for diurnal species and especially in an open environment and at intermediate or close ranges. Dark, contrasting, and moving objects are particularly attractive. As the female approaches to within one to 2 m of a potential host, chemical and visual cues are still important, but convective heat and humidity surrounding the body also

come into play. Odors, carbon dioxide, heat, and humidity all are detected by sensilla on the antennae and palps. In the special case of some frog-feeding species, the calls of frogs serve as auditory attractants.

Host-finding behavior in mosquitoes was reviewed by Bowen (1991) and Cardé and Gibson (2010). Specific behavioral and physiological aspects of attraction have been reviewed by several authors (Anon., 1994). Takken and Verhulst (2013) have reviewed the entire subject of mosquito host preferences.

If the suite of **host stimuli** is acceptable, the female attempts to land on the host animal, often preferring certain body parts, such as the head or legs. On landing, she proceeds through four phases of feeding behavior: exploration, penetration and vessel-seeking, imbibing, and withdrawal. She typically remains motionless for a few seconds, then begins exploratory movements, including contacting the skin surface with her proboscis in probing motions. If the host is not suitable, she may wander for a considerable time and leave without feeding. Even on a suitable animal she usually explores at least briefly before selecting a spot that is likely to be well vascularized. Probing activity is stimulated by heat and moisture, and probably also by chemicals on the surface of the skin. As in the case of airborne attraction, these stimuli are detected by antennal and palpal receptors, but receptors on the proboscis, tarsi, and elsewhere on the legs apparently also are important.

FIGURE 15.19 Female mosquito (*Haemagogus lucifer*, a vector of jungle yellow fever), showing position of mouthparts during blood-feeding. The fascicle is exsheathed from the labium along part of its length while the labium buckles backward, allowing the fascicle to enter the skin in search of a blood vessel. *Photograph by Woodbridge A. Foster.*

Adult blood-feeding has been studied extensively. Mosquitoes can feed from a variety of skin surfaces, including the moist skin of frogs and the scaly legs of reptiles and birds. They can penetrate mucus, matted hair, light layers of feathers, and heavy cloth such as denim, provided it is not thicker than the length of the proboscis. Once a feeding site is selected, the fascicle of stylets pierces the skin while the labium serves as its guide and is bent backwards without penetrating (Fig. 15.19). The maxillae and mandibles on each side of the fascicle alternately slide by each other in quick stabbing/puncturing movements. While doing so the tissue is gripped with the backward-directed maxillary teeth as the stylets penetrate epidermal and subepidermal tissue.

Saliva flows from the tip of the hypopharynx as the flexible end of the fascicle bends at sharp angles, probing in various directions within the subepidermal tissue in search of a small arteriole or venule. The saliva contains an antihemostatic enzyme, apyrase, which inhibits platelet aggregation and causes randomly punctured vessels to bleed freely into the surrounding tissue spaces. This makes it easier for the mosquito to locate a vessel and shortens the total time on the host. The saliva also contains anticoagulants, which facilitate vessel location and blood ingestion by preventing the blood from clotting. Sensilla on the labrum and in the cibarium apparently detect plasma and cellular factors, including adenyl nucleotides such as ATP, which help the mosquito to locate a blood vessel and stimulate ingestion. Upon finding a vessel, the female slips the tip of her fascicle into the lumen and draws blood up through the food canal by pumps in the cibarium and pharynx. The blood accumulates in the midgut, allowing the mosquito to engorge fully in 1–4 min.

During this time, the female begins to extract water from the bloodmeal and may deposit small droplets of urine on the host's skin. In some *Anopheles* spp., this fluid is copious, to a degree coming directly from the midgut, rather than being processed through the malpighian tubules. It includes some blood cells from the accumulating meal and appears red. When abdominal stretch receptors signal the presence of sufficient blood in the midgut, the female pushes with her forelegs to withdraw her stylets and flies away. Usually she is too heavy to fly far until a substantial amount of water and salt in the bloodmeal has been excreted in the urine, after 1–2 h.

While digesting the meal and developing eggs, females locate species-characteristic **resting sites** and may remain there until the eggs are mature. However, females of many species are known to leave their resting sites during each daily activity period throughout the gonotrophic cycle. These flights allow them to obtain sugar meals or supplementary bloodmeals, to relocate closer to an oviposition site, or perhaps simply to find a more suitable resting site. In at least some species, a hormone from the ovaries in the trophic phase inhibits the mosquito's responsiveness to host attractants, by blocking host-odor receptors on the antennae, provided she has substantial energy reserves (see Klowden, 1996, for a concise review).

The interval of time between bloodmeals, an important component of **pathogen transmission**, is determined by two things: the occurrence of supplementary bloodmeals within the gonotrophic cycle and the duration of the cycle. The latter depends not only on how fast the blood is digested and eggs matured but also on how soon the female lays her entire batch of eggs. In addition, feeding success and frequency are modified by pathogens that appear to manipulate the mosquito to enhance its likelihood of transmission between vertebrate hosts. For example, the malaria parasite *Plasmodium* interferes with salivation, causing the mosquito to pierce one or more hosts and inject the parasite multiple times within a single night. Furthermore, infected mosquitoes are more strongly attracted to hosts, and infected hosts are more attractive to mosquitoes than are uninfected hosts. Some of these interactions have been reviewed by Cator et al. (2012).

Oviposition generally occurs during the same part of the day as mating and feeding. Gravid females locate and evaluate suitable sites by using chemical and visual cues, including organic chemicals, salts, high humidity, dark cavities, and reflective surfaces. The organic chemical cues are derived from decaying organic matter, microorganisms, the chemical byproducts of larvae or pupae that have previously developed there, and the presence of mosquito eggs that have been deposited by other females. The apical droplets on the eggs of *Culex quinquefasciatus* contain an oviposition-attractant pheromone.

Within each genus of mosquito, there is considerable variation among species in their **oviposition site preferencs**, and therefore their larval habitats. In general, *Anopheles* spp. occur in permanent or semipermanent water, such as the edges of lakes, ponds, streams, and pools; others develop in temporary rain puddles, leaf axils, and tree holes. *Culex* typically lay eggs in permanent or semipermanent pools, ponds, and water containers. Several medically important species of both *Anopheles* and *Culex* develop in water with large surface areas, taking advantage of irrigated fields and reservoirs created by dams. *Culiseta* are found in several kinds of permanent surface pools; some species have very narrow requirements. *Coquillettidia* and *Mansonia* oviposit in permanent bodies of water that contain floating or emergent aquatic plants to which the submerged immatures can attach.

Aedes, *Psorophora*, and *Haemagogus* spp. lay their eggs on damp surfaces where they will be inundated by temporary water or a rising water level. *Aedes* spp. and ecologically similar mosquitoes form two general categories, according to typical habitat: (1) **floodwater mosquitoes**, which include floodplain species, saltmarsh species, and snow pool and spring species—some floodplain species have become prolific in rice fields and other forms of irrigation; (2) **container mosquitoes**, including leaf-axil species, tree-hole species, and artificial-container species. Several medically important species use both tree holes and artificial containers. Among genera of minor importance, *Toxorhynchites* lay their eggs only in natural and artificial containers in wooded areas; *Wyeomyia* oviposits primarily in leaf axils, liana crevices, flower bracts, or pitcher plants, depending on the species, and *Deinocerites* oviposits exclusively in crab holes in tidal mudflats and mangrove swamps.

The distribution of mosquito eggs reflects the availability, size, and stability of the larval habitats used by a species. Though mosquito life histories are highly variable, the oviposition behavior of mosquitoes tends to follow along taxonomic lines. Most mosquitoes fall into one of three behavioral categories:

1. **Eggs laid out of water**. Species in this group may distribute eggs of a single clutch individually among several widely scattered potential development sites, particularly if those sites are common but small. Container species such as *Aedes aegypti*, *Ae. albopictus*, and *Haemagogus* species deposit the eggs at varying distances above the water line, and at least *Ae. aegypti* lays only a portion of the clutch in each water container, sometimes called skip oviposition. Floodwater species such as *Ae. vexans*, *Ae. dorsalis*, and the saltmarsh-inhabiting *Ae. taeniorhychus* generally scatter their eggs widely over areas where water will accumulate, inserting them into crevices of drying mud or plant debris in low ground.
2. **Eggs placed on or in water**. Mosquitoes in this category lay the entire clutch in a clump at one site while standing on the water surface or on floating vegetation. The egg rafts of *Culex*, *Culiseta*, *Coquillettidia*, and *Uranotaenia* spp. are formed between the female's hindlegs as she deposits each egg on end in the water, one against the next. Some *Armigeres* spp. suspend the egg raft above the water with their hindlegs while forming it and then carry it with them before placing it on the water. *Trichoprosopon digitatum* females stand guard over the raft until the eggs hatch. *Mansonia* spp. prepare their egg clusters underwater, attached to a

plant, while standing on the floating leaves of aquatic plants. Exceptional species in various genera deposit egg rafts on top of floating vegetation, lay their eggs singly underwater on the sides of rock pools, or enter beetle holes in bamboo and extrude the eggs in ribbons.

3. **Eggs dropped onto water**. Species in this group oviposit aerially while hovering. *Anopheles* drop all of them at one site or distribute them among several smaller sites. *Toxorhynchites* and most culicine species in the tribe Sabethini (e.g., *Wyeomyia* and *Sabethes*) propel a few eggs into each of many container habitats with a flick of the abdomen, often through small openings in tree limbs or bamboo. If a mosquito cannot find suitable oviposition sites when the eggs are mature, it may lay them in suboptimal situations or retain them until a suitable site is found. Retained eggs gradually lose their viability over several weeks or months. An extensive review of oviposition behavior is given by Bentley and Day (1989).

Dormancy occurs in all species of mosquitoes except those living in tropical and subtropical habitats that provide conditions for year-round larval development. The life stage that becomes dormant depends on the severity of a region's winter or dry season and on the species. Species of *Aedes*, *Psorophora*, and *Haemagogus*, which all have quiescent eggs requiring inundation regardless of season, typically overwinter (hibernate) or oversummer (aestivate) as embryonated eggs. Larvae serve as the dormant stage in mosquitoes whose adult activity is precluded seasonally but whose breeding sites are protected from severe cold or complete drying. When adults overwinter as the dormant stage, typically in *Anopheles* and *Culex*, they seclude themselves in well-protected harborages, or **hibernacula**.

Prior to dormancy, mosquitoes often enter **diapause**, a physiological state of arrested development that is induced or broken only by specific environmental cues. Mosquito diapause is reviewed by (Denlinger and Armbruster, 2014). **Facultative egg diapause**, a feature of multivoltine species, is induced by exposure of the pupae or adult females to lowered temperatures and short photoperiods. They lay diapausing eggs, which will not hatch until the day length is appropriate, even during unseasonably warm periods in autumn, winter, or early spring, when the resulting larvae and adults might not survive. **Obligate egg diapause** occurs in univoltine species, regardless of preceding conditions, and is maintained despite warm, long-day conditions. This is typical of snow pool and spring *Aedes* spp. in cold and temperate climates. Diapause is broken after the eggs have been subjected to winter conditions (in the case of obligate diapause) and when favorable temperatures and long days resume. **Larval diapause** is similar to facultative egg diapause in its induction and termination. Diapausing larvae feed and grow little or not at all, and they do not molt.

Temperate species destined to overwinter as adult females emerge in a state of **reproductive diapause** induced by larval and pupal exposure to shortening photoperiod and cool temperatures. Although these females mate, their egg follicles do not reach the resting stage, despite frequent sugar-feeding and accumulation of extensive fat reserves. Fattened female *Culex* spp. that hibernate through hard winters forego all further feeding until the onset of spring, whereas in milder climates they periodically leave their overwintering sites to take sugar meals.

Although diapausing *Culex* adults rarely feed on blood, some *Anopheles* species may take bloodmeals fairly regularly from hosts near these sites. They develop no eggs, however, exhibiting **gonotrophic dissociation**. Other overwintering *Anopheles* continue to feed and develop eggs, but these are not laid. Similarly, some tropical *Anopheles* take blood repeatedly during the dry season and remain continually gravid because there is nowhere to oviposit. These phenomena are sometimes referred to as **gonotrophic discordance**, a term that also applies to the taking of nonvitellogenic or otherwise supplementary bloodmeals, mentioned previously.

GENETICS

The formal and molecular genetics of moquitoes have been investigated in detail and remain the subject of intensive research, owing to two objectives: (1) controlling vector populations (Rai, 1996; Alphey, 2014; Burt, 2014) and (2) understanding genotype/phenotype relationships, such as the genetic basis for vector competence (Beerntsen et al., 2000) and insecticide resistance (Liu, 2015).

Mosquitoes typically have three pairs of **chromosomes**, with a single species (*Chagasia baffana*, of the Anophelinae) having four. Two pairs are autosomes and the third pair are sex chromosomes, which may be heteromorphic (anophelines) or homomorphic (culicines). Sex is determined by chromosomal heteromorphy in the former and by a sex-determining locus in the latter. In anophelines, males are the heterogametic sex and females are homogametic. When the sex-determining gene *Nix*, operating at the sex locus M, was knocked out in male *Aedes aegypti*, they were feminized; and when it was expressed in females, they developed male genitalia (Hall et al., 2015). This study thus isolated the genetic factor determining male sex in *Aedes aegypti* and illuminated the role of the *doublesex* gene in determining female sex.

Phenotypic expression of sex traits is also modulated in some groups of mosquitoes by temperature, such as when larval mosquitoes normally developing in cold water are forced to develop in warm water.

Formal genetics of mosquitoes has included isolation of morphological (e.g., eye color) and biochemical (e.g., enzymatic resistance to DDT) mutants. Crosses

between traits have revealed their Mendelian relationships, such as their segregation or linkage tendencies with other genes. Chromosomal morphology, karyotyping, description and quantification of translocations and crossovers, and genetic mapping are all well-developed endeavors in the field of mosquito research, using the banding patterns of polytene chromosomes. This research has yielded a wealth of information on genome structure; on population genetic structure; on species complexes in *Culex*, *Aedes*, and *Anopheles*; and on the potential to use genetic constructs such as translocation homozygotes or sterile-hybrid mating as tools for vector control. In their review of mosquito genomics, Severson and Behura (2012) note that genome sequence data for *Anopheles gambiae*, *Aedes aegypti*, and *Culex quinquefasciatus* "have revealed detailed information on genome architecture as well as phenotype-specific transcriptomics and proteomics. These resources allow for detailed comparative analyses within and across populations as well as species."

The **genomes** of 16 *Anopheles* spp. have been sequenced (Neafsey et al., 2015). Although incomplete annotation and mapping of these genomes present challenges, there has been substantial progress in genome manipulation and genome engineering, using such tools as zinc-finger nucleases, TAL-effector nucleases, homing endonucleases, and the bacterial type II CRISPR-cas9 system (Kistler et al., 2015). Applications of these tools include dominant lethal constructs (Alphey, 2014) and mechanisms to drive beneficial genes into populations (Burt, 2014).

PUBLIC HEALTH IMPORTANCE

Mosquitoes are of public health significance because they feed on human blood. Blood-feeding compromises skin, presenting the possibility of secondary infection with bacteria. It introduces foreign proteins with saliva that stimulate histamine reactions, causing localized irritation, and that may be antigenic, leading to hypersensitivity; and it allows for acquisition and transmission of microorganisms that cause infection and disease in humans, domestic animals, and wild animals. Mosquito-borne diseases are caused by three groups of pathogens: viruses, malaria protozoans, and filarial nematodes. Mosquitoes are not known to transmit pathogenic bacteria to humans, with the exception of mechanical transmission of the causative agents of tularemia (*Francisella tularensis*) and anthrax (*Bacillus anthracis*).

Mosquito Bites

In addition to the tremendous impact of mosquitoes on human health as vectors of disease pathogens, the bites themselves are important. Aside from the mosquito's annoying flight and buzzing sound, a single bite can be irritating and a distracting nuisance. In Rangoon, Myanmar, *Culex quinquefasciatus* has been estimated to have densities of 15 million per square kilometer, and residents in poor districts receive 80,000 bites by this species per year. In Burkina Faso, in West Africa, residents of cities are estimated to experience 25,000 bites by this species per year. In northern Canada, the spring melt of snow brings with it hordes of snow pool *Aedes*; counts on an exposed human forearm can be as high as 280 to 300 bites per minute. It has been estimated that this rate of biting, without protection, can reduce the total blood volume in a human body by half in 90 min.

As with the other blood-feeding arthropods, the wound created at the bite site may allow secondary infection by bacteria, which can be exacerbated by scratching. In the absence of prior exposure to mosquitoes, a bite rarely produces more than a temporary tingling or burning sensation and sometimes a tiny spot of blood on or just beneath the surface of the skin. After one or more bites within a brief time, the proteins in mosquito saliva, which are injected both before and during feeding, normally stimulate the immune system so that subsequent bites give rise to one or both of two general kinds of cutaneous allergic response: **immediate reactions** and **delayed reactions**. The immediate reaction, called type I hypersensitivity, is an antibody-based inflammation of the skin known as **wheal-and-flare** caused by the release of histamine and arachidonic acid from antibody-sensitized mast cells. It usually starts within minutes of the bite and lasts a few hours at most. The typical delayed reaction, designated type IV hypersensitivity, involves a cellular immune response caused by lymphokines that are secreted by antigen-sensitized T cells. Both delayed and immediate reactions result in itching, redness, and swelling. The typical delayed reaction takes about 1 day to develop, may last for up to a week, and tends to result in a larger wheal with a deeper discoloration. The appearance of these two reactions, starting with the initial bites, changes after repeated bites by the same genus or species of mosquito over a period of days, weeks, and months. This timing, known as the Jones-Mote hypersensitivity series, typically proceeds as follows: (1) no reaction, (2) delayed only, (3) immediate and delayed, (4) delayed only, and, in some cases, (5) absence of any skin reaction (i.e., desensitization).

Mosquito-Borne Viruses

Among the more than 620 viruses associated with arthropods and registered in the US Centers for Disease Control and Prevention's Arbovirus Catalog. Somewhat less than half have biologic relationships with mosquitoes, and about 100 infect humans. The term **arbovirus** is a contraction of "arthropod-borne virus" and has no formal taxonomic meaning. The most significant mosquito-borne viruses causing human illness belong to four genera in three families (Table 15.2): the **Togaviridae**, genus

TABLE 15.2 Geographic Distribution of Selected Mosquito-Borne Viruses of Importance to Humans or Domestic Animals

Family (Genus)	Virus Species and Serotypes	Distribution
Togaviridae (*Alphavirus*)	Eastern equine encephalomyelitis	Americas
	Venezuelan equine encephalomyelitis	South and Central America, Mexico, United States (Florida)
	Western equine encephalomyelitis	North America, Mexico, South America (eastern)
	Chikungunya	Africa, Asia, incl. Philippines, South, Central, and North America
	O'nyong nyong	Africa
	Ross River	Australia, New Guinea, Fiji, American Samoa
	Semliki Forest	Africa, Asia, incl. Philippines
	Mayaro	South America (northern), Trinidad
Flaviviridae (*Flavivirus*)	Dengue (4 serotypes)	Tropics, especially southern Asia and Caribbean
	Yellow fever	Africa, Central and South America
	St. Louis encephalitis	Americas
	Murray Valley encephalitis	Australia, New Guinea
	Japanese encephalitis	Asia (eastern), incl. Philippines
	West Nile	Africa, Europe, Israel, Asia; South, Central, and North America
	Ilheus	Central and South America
	Rocio	Brazil
	Wesselsbron	Africa, Asia (southern)
	Zika	Africa, Asia, South Pacific, South and Central America, Caribbean, southern North America
Bunyaviridae (*Orthobunyavirus*)	Bunyamwera	Africa
	Germiston	Africa
	Ilesha	Africa
	Wyeomyia	Central America
	Itaqui	South America
	Marituba	South America
	Murutucu	South America
	Oriboca	South America
	Madrid	Central America
	Nepuyo	Central and South America
	California encephalitis	United States (western)
	Jamestown Canyon	North America
	La Crosse encephalitis	United States (eastern)
	Inkoo	Finland
	Tahyna	Europe
	Guaroa	South America
Bunyaviridae (*Phlebovirus*)	Rift Valley fever	Africa (northern, eastern)

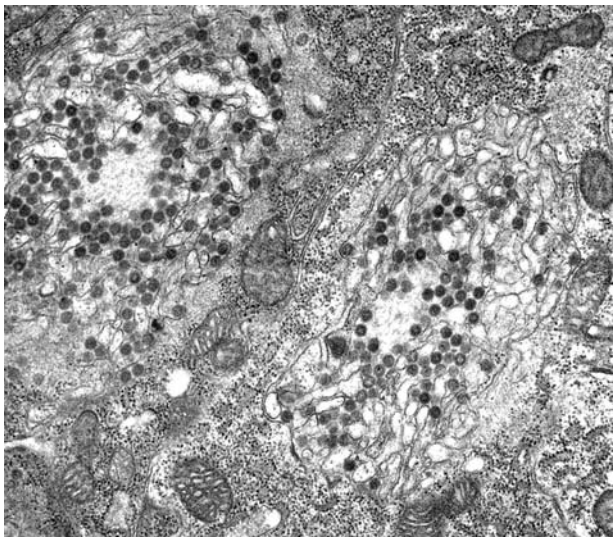


FIGURE 15.20 Rift Valley Fever virus virions in salivary gland tissues of *Culex pipiens* female, 42,000 X. *Courtesy of K. Lerthudsnee and W. S. Romoser.*

Alphavirus; the **Flaviviridae**, genus *Flavivirus*; and the **Bunyaviridae**, genera *Orthobunyavirus* and *Phlebovirus*. Some of the arboviruses infect both humans and animals and may cause disease in both. A representative of these anthropozoonotic arboviruses, Rift Valley fever virus, is shown in Fig. 15.20.

Aside from their genomic organization and morphology, mosquito-borne viruses may be viewed in terms of disease symptoms they cause. In humans, generally speaking, the mosquito-borne viruses cause infection with either no apparent symptoms, or acute disease of either systemic febrile illness (fever), encephalomyelitis (inflammation of the brain and spinal cord), hemorrhagic fever (bleeding and fever), or febrile myalgia and arthralgia (fever with muscle and joint pain, or arthritis). The case-fatality rate tends to be low for fevers, although morbidity (illness) may be high. For the encephalitis and hemorrhagic fevers, morbidity and mortality may range from low to high, depending on the virus and factors such as age. After the acute phase, humans either recover fully or show various sequelae, such as neurological problems after acute encephalitis. Long-term, chronic infection with the mosquito-borne viruses does not occur in humans, although the consequences of infection may be long lasting.

Table 15.2 is a list of the more important mosquito-borne arboviruses and their geographic distribution. The viruses may be classified hierarchically as follows: family, genus, serogroup, complex, species, serotype, and strain. The word **strain** is used to refer to different viruses of the same serotype that were isolated from different locations or different biologic sources or that show minor differences in antigenicity or genotype but not enough to justify elevating them to the varietal level. For example, Altamont virus is an eastern New York State strain of La Crosse virus, which along with Snowshoe hare virus is a serotype of the species California encephalitis virus in the California encephalitis virus complex of the California serogroup in the genus *Orthobunyavirus*, family Bunyaviridae. The relationships among the viruses are determined on the basis of similarities and differences in antigenic reactions to antibodies in immunological tests, and on genetic relationships determined by molecular analyses (i.e., nucleotide sequences or oligonucleotide fingerprint patterns). Arbovirus classification is presented by Fauquet et al. (2005).

Mosquito-borne viruses multiply in both invertebrate and vertebrate cells. Many arboviruses cause cytopathic effects and cell destruction in vertebrate cells; in invertebrate cells the same viruses typically cause a chronic cellular infection without cytopathology. Competent mosquitoes become infected when they feed on blood of a viremic vertebrate host in which there is sufficient circulating virus in its blood to provide an infectious dose to the mosquito. After blood has entered the midgut, the virions bind to and then pass through the microvillar membrane and into midgut epithelial cells. Within these cells, viruses replicate and virions bud off from the cells, pass through the basal lamina, and enter the hemolymph. Virions disseminate throughout the body of the mosquito and may infect and replicate in a variety of tissues, including salivary glands, fat body, ovaries, and nerves. A mosquito with a salivary-gland infection (Fig. 15.20) can transmit infectious virions during salivation as it probes the tissues of another vertebrate host. In some mosquitoes and for some viruses, **transovarial transmission** of virions occurs from the female mosquito to her progeny, and females of the next generation can transmit the virus orally without having become infected by a prior bloodmeal. Also, **venereal transmission** of some arboviruses from male to female mosquito has been documented experimentally. The rate of virus infection and dissemination in mosquitoes is temperature-dependent; higher temperature results in shorter extrinsic incubation. Virus infection may harm mosquitoes in some cases, and they become infected for life.

Because the flaviviruses, alphaviruses, and bunyaviruses have RNA genomes and can replicate in both invertebrate and vertebrate hosts, these viruses have a high capacity for rapid evolution into antigenically variable strains of varying virulence. This capacity for change is important, because it may result in emergence of virulent strains; indeed there is evidence for this process (see sections on Chikungunya, O'nyong nyong, and Rocio encephalitis viruses, later).

The literature on the mosquito-borne viruses is voluminous. Extensive reviews are presented in the multivolume series edited by Monath (1988), in Strickland

TABLE 15.3 Relationships of Selected Alphaviruses to Mosquito Vectors and Vertebrate Reservoir Hosts

Virus Species	Vector(s) Reservoirs	Vertebrate
Eastern equine encephalomyelitis	*Culiseta melanura, Aedes sollicitans, Coquillettidia perturbans, Culex nigripalpus, Culex (Melaniconion)* spp.	Birds
Western equine encephalomyelitis	*Culex tarsalis, Culex (Melaniconion)* spp., *Aedes albifasciatus, Aedes melanimon, Aedes dorsalis*	Birds, lagomorphs
Venezuelan equine encephalomyelitis[a]	*Culex (Melanoconion)* spp., *Aedes, Psorophora, Anopheles, Mansonia* spp.	Rodents
Chikungunya	*Aedes aegypti, Aedes africanus*	Primates, incl. humans
O'nyong nyong	*Anopheles* spp.	Humans
Ross River	*Culex annulirostris, Aedes vigilax, Aedes polynesiensis*	Humans, rodents

[a]*See Table 15.4 for elaboration of this virus complex.*

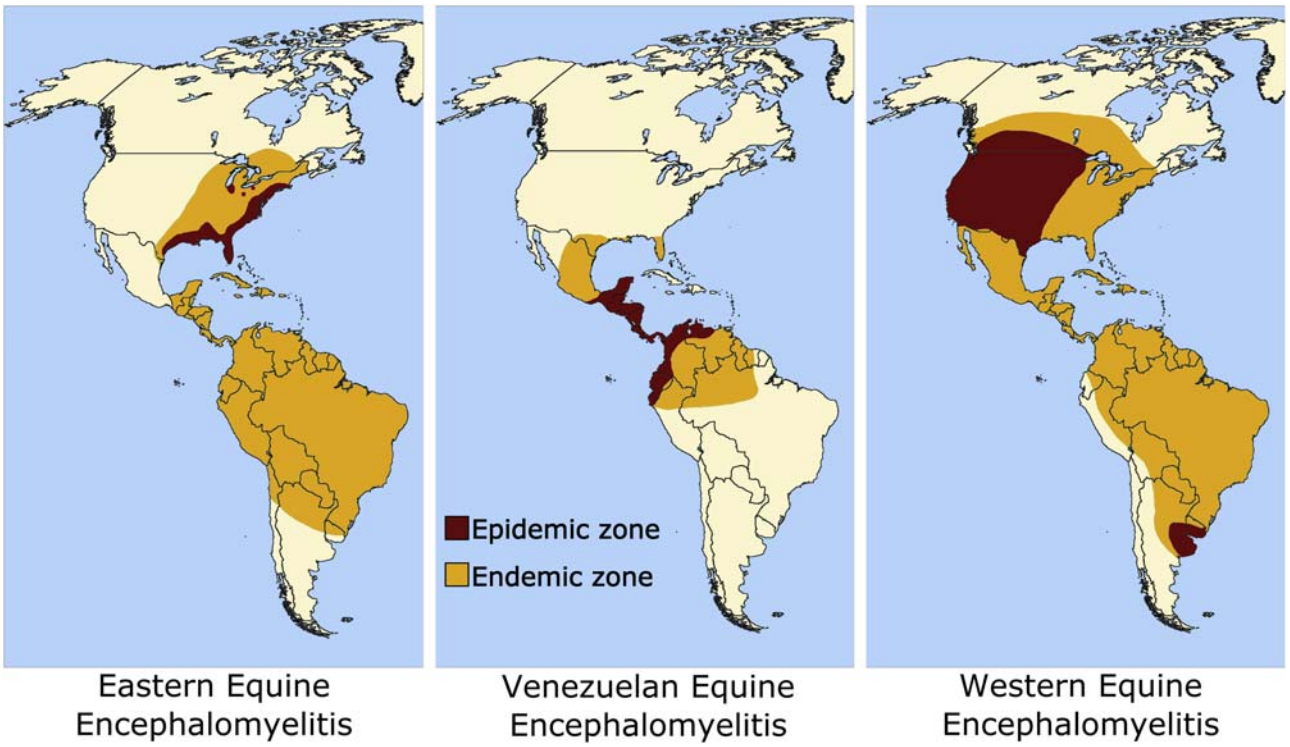


FIGURE 15.21 Endemic distribution and epidemic zones of mosquito-borne alphaviruses that cause encephalitis in the New World. *Reconstructed from Mitchell, 1977, and other sources.*

(1991), in the sections on Togaviridae, Flaviviridae, and Bunyaviridae by Fields et al. (1996), in Reeves (1990), and in Clements (2011), as well as in references mentioned under specific groups later and at the end of this chapter.

Togaviridae (*Alphavirus*)

The Togaviridae, genus *Alphavirus*, contains seven antigenic complexes of viruses involving 37 types, subtypes, and varieties distributed worldwide, 35 of which have been isolated from mosquitoes. Many of these are medically important. Table 15.3 summarizes relationships between primary mosquito vectors and important alphaviruses, whereas Fig. 15.21 shows their distribution in the Western Hemisphere.

Eastern equine encephalomyelitis (EEE) virus. This virus is distributed in South America, Central America, the

Caribbean basin, and eastern North America. Analyses of geographic strains of EEE virus have revealed two varieties: South American and North American/Caribbean. The virus has been isolated from many different states in the United States, but most cases of human or equine disease are in coastal states from Massachusetts to Louisiana, an area of upstate New York near Syracuse, a swamp focus in east-central Ohio, and southern Michigan and part of northern Indiana.

Eastern equine encephalomyelitis virus is one of the most pathogenic among all of the mosquito-borne encephalitis viruses. In humans, disease caused by EEE virus infection results in high morbidity and mortality. The type and severity of illness in humans depend on the age and health status of the individuals. Children, the elderly, immunocompromised individuals, and sometimes apparently healthy adults develop acute encephalitis with high fever, drowsiness, lethargy, vomiting, convulsions, and coma. Mortality rates among clinical cases exceed 50%. Individuals who survive infection often show neurologic sequelae, although some survivors recover completely, sometimes showing rapid and dramatic improvement from coma.

In the eastern United States, EEE virus occurs in a bird−mosquito enzootic cycle in swamps that support the biology of the enzootic vector, *Culiseta melanura*. The swamps comprising EEE foci are characterized in the northern distribution of its range by northern white cedar, black spruce, tamarack, or red maple trees, typical of swamp or bog ecosystems. Larvae of *Cs. melanura* occur in water-filled cavities underneath raised tree hummocks, water-filled depressions formed by uprooted trees, and in holes in bog mats. Adults feed on birds in the swamp and may leave swamps for open areas to locate hosts, returning later to oviposit. *Culiseta melanura* females are highly efficient vectors of EEE virus and transmit it primarily to swamp-dwelling passeriform birds. More than 48 species of wild, native birds have shown evidence of infection. The mechanism of overwintering of EEE virus is unknown. Studies in New Jersey indicate that the virus may recrudesce in resident birds in the spring, whereas other studies incriminate reptiles as overwintering hosts. Still other scenarios suggest that this virus is introduced by migrating birds from southern regions where viral transmission may occur year-round.

In some summers, for reasons possibly related to weather patterns and density of bird and mosquito hosts, EEE virus in its enzootic swamp setting becomes amplified to high levels so that epizootics and epidemics develop. Certain mosquito species function as bridge vectors, especially *Aedes sollicitans* in coastal areas and *Coquillettidia perturbans* in inland areas. They acquire virus infection by feeding on viremic birds, later blood-feeding on mammals and transmitting the virus to them. In Central and South America, EEE virus apparently circulates among rodents and birds through mosquitoes in the *Culex* (*Melanoconion*) group; however, the relationships among vectors and vertebrate hosts involved in enzootic, epizootic, and epidemic cycles of EEE virus in these regions are poorly known compared with in North America.

Epidemics and concurrent epizootics have been documented in the eastern United States since the 1930s, generally involving cases in horses, pheasants, and humans. The virus was first isolated from brains of horses that died during a 1933 epizootic along the eastern coast of the United States, in Virginia, Delaware, and Maryland. Disease in humans was first recognized in 1938 in Massachusetts. Generally, outbreaks involve many horse cases and very few human cases. Nearly all outbreaks involving human cases have occurred along the eastern coast of the United States. From 1964 to 1995, a total of 151 human cases of EEE infection were reported in the United States, with an average of about five cases (range, usually 0 to 14) per year. From 2007 to 2016, there were 70 diagnosed cases, including 28 deaths. The greatest number of human cases occurred in 1959, when 36 were documented, mainly in New Jersey. Morris (1988) reviewed the ecology, epidemiology, and vector relationships of EEE.

Western equine encephalomyelitis (WEE) virus. This virus is a complex of six types and six subtypes. All are mosquito-borne, with the exception of **Fort Morgan virus**, which is associated with cliff swallows and their parasitic cimicid bugs (*Oeciacus vicarius*) in western North America. The other viruses are widely distributed across the high plains of the United States and Canada, in California, and through Central and South America. The WEE virus type that occurs in North America has been responsible for acute encephalitis in horses and humans. An apparently less pathogenic virus, **Highlands J virus**, is closely related to Fort Morgan virus, yet it exists in the same basic North American enzootic cycle as EEE virus.

Disease caused by WEE virus in humans is less severe than that caused by EEE virus, and some infections are unapparent. Symptoms are generally similar, with acute onset of meningitis or encephalitis with headache, fever, drowsiness, and coma and death in severe cases. Fatality rates are in the range of 5% or less. Most morbidity and mortality occur in infants rather than teens, adults, or the elderly. Neurologic sequelae are often evident in survivors.

In North America, WEE virus apparently exists enzootically in at least two different cycles. The primary cycle involves passerine and columbiform birds (especially house sparrows, house finches, blackbirds, orioles, and mourning doves), with *Culex tarsalis* functioning as the enzootic, epizootic, and epidemic vector. Nestling birds, which are more exposed to mosquito bites, may be more

important as virus amplifier hosts than adult birds. The other cycle involves jackrabbits as vertebrate hosts and *Aedes melanimon* or *Ae. dorsalis* as vectors in California, Utah, and Colorado. Ground and tree squirrels also may function as vertebrate hosts in some areas, and in the central United States *Ae. trivittatus* may be a secondary vector. In Central and South America, WEE virus apparently circulates in nature among *Culex* (*Melanoconion*) mosquitoes, although a cycle involving *Ae. albifasciatus* and lagomorphs has been elucidated in Argentina.

Epizootics and epidemics of WEE in North America appear to be related to cumulative summertime precipitation and wintertime snowpack development, both of which can increase populations of *Culex tarsalis* and favor virus transmission. The larvae of this mosquito are often associated with agricultural irrigation and vernal flooding. Areas that normally are dry can produce large populations of *Cx. tarsalis* if irrigation activities result in the accumulation of pools of still water long enough to support larval development. For example, *Cx. tarsalis* populations burgeon after winter rain and vernal snow melt result in inundation of saltwater marshes along the north shore of Salton Sea in southern California (USA). Western equine encephalitis virus is detected in *Cx. tarsalis* populations about 2 months after these populations increase, during March through June. The virus spreads to upland sites to the northwest of the sea, where mosquitoes are produced primarily from poorly maintained irrigation systems. The movement of WEE virus along this corridor is probably due to dispersal of infected *Cx. tarsalis* rather than to movement of birds. The mechanism by which WEE virus overwinters at this site, or is introduced there, is not known.

WEE virus was first isolated in 1930 from the brain of a dead horse in the San Joaquin Valley of California (USA). It later was identified as the causative agent of human disease in a child who died of encephalitis in the San Joaquin Valley of California in 1938. Since that time, WEE virus has been implicated in epizootics in horses and concurrent epidemics in humans, with cases numbering from hundreds to thousands in some instances. Large outbreaks occurred in 1941 and 1975 in the Red River Valley in Minnesota and North Dakota (USA), and Manitoba (Canada), in 1952 in the Central Valley of California, and in 1965 in Hale County in western Texas (USA). There are horse cases almost every summer within the range of the virus, but epizootics do not occur every summer. From 1964 to 1995, a total of 639 human cases of WEE infection were reported in the United States, with an average of about 20 cases (range, 0–172) per year. Reisen and Monath (1988) provided a review of WEE.

Venezuelan equine encephalomyelitis (VEE) virus. This is complex of 12 viruses that cause disease in humans and equids (horses, burros, and mules) and occurs in northern South America, Central America and Mexico, occasionally extending into Texas (Walton and Grayson, 1988). These viruses exist as either enzootic or epizootic varieties and strains, with overlapping or disjunct geographic distributions and with variable vector and vertebrate–host relationships. Table 15.4 shows the classification and vector associations of these viruses.

Many VEE "enzootic" virus subtypes and varieties exist in cycles involving rodents and mosquitoes of the *Culex* (*Melanoconion*) group, such as *Culex ocossa*, *Cx. panocossa*, and *Cx. taeniopus* in Central and South America. Rodents in the genera *Sigmodon*, *Oryzomys*, *Zygodontomys*, *Heteromys*, *Peromyscus*, and *Proechimys* are important vertebrate hosts; birds, opossums, and bats also may be reservoir hosts. The ecology of the epizootic viruses is quite different. A large number of species of mosquitoes in several different genera (see Table 15.4) have been implicated as vectors of the epizootic/epidemic virus strains. Equids attain sufficient viremia to infect these mosquitoes. VEE epidemics can be maintained by mosquito–equid–mosquito transmission, unlike WEE and EEE epidemics, in which equids are for the most part dead-end hosts. Wading birds, particularly green herons, have been incriminated as vertebrate hosts of epizootic strain IA-B in Panama, with the crab hole mosquito *Deinocerites pseudes* functioning as vector. Persistence of epizootic strains of VEE in interepidemic periods is not well understood, thus their emergence in epidemics among equids and humans is difficult to predict.

The single representative of the VEE viruses in the United States, other than during epizootics in Texas, occurs in the Everglades region of southern Florida. Called **Everglades virus**, it is associated with *Culex* (*Melanoconion*) *cedecei* (formerly, *Cx. opisthopus*) as a vector and cotton rats (*Sigmodon hispidus*) and cotton mice (*Peromyscus gossypinus*) as vertebrate hosts. The zoonotic setting is the hardwood hammocks of the Everglades, where mosquito and rodent habitats overlap. Serosurveys of Seminole and Miccosukee Indians in these regions have shown that many Indians have antibodies to VEE virus, but there have been very few cases of human disease attributable to this virus.

Humans infected with an epizootic or certain enzootic strains of VEE virus may show no symptoms, only mild flulike symptoms, or severe encephalitis with acute onset of vomiting, headache, seizures, and fever. Symptoms tend to be most severe in children. During epidemics, the mortality rate is typically less than 1%, although in some epidemics the mortality rate has been considerably higher.

The VEE viruses in Central America and northern South America have been intensively studied because of the history of epidemics among equids and humans in these regions. Outbreaks of VEE have occurred periodically in South America, Central America, and Mexico since the 1930s. The first VEE virus was isolated in 1938 from a dead horse in Venezuela. In 1969, a large outbreak of VEE

TABLE 15.4 Venezuelan Equine Encephalomyelitis Virus

Subtype	Variety	Name	Vector Associations	Geographic Distribution
I	A-B[a]	–	*Aedes, Psorophora, Mansonia, Anopheles, Deinocerites pseudes*	Central America, South America (northern)
	C[a]	–	(same as IA-B)	Central America, South America (northern)
	D	–	*Culex* (*Mel.*) *ocossa* *Culex* (*Mel.*) *panocossa*	Central America, South America (northern)
	E	–	*Culex* (*Mel.*) *taeniopus*	Cental America
	F	–	Unknown	Brazil
II		Everglades	*Culex* (*Mel.*) *cedecei*	United States (southern Florida)
III	A	Mucambo	*Culex* (*Mel.*) *portesi*	South America (northern)
	B[b]	Tonate	Unknown	South America (northern)
	C	–	Unknown	Peru
IV		Pixuna	Unknown	Brazil
V		Cabassou	Unknown	French Guiana
VI		–	Unknown	Argentina

Classification, mosquito associations, and geographic distribution of subtypes and varieties of this virus.
[a]*Virulent to equids and humans and involved in epizootics. The other subtypes and varieties are enzootic.*
[b]*A related variety IIIB (Bijou Bridge virus) is not listed here. It is associated with cliff swallow bugs (*Oeciacus vicarius*; Hemiptera: Cimicidae) in western North America.*

involving both equids and humans in Central America spread northward through Mexico in the next 2 years, moving into Texas (USA) in 1971. Cases continued in Mexico through 1972. There were thousands of both horse and human cases throughout this region during that time, but epizootic virus activity did not occur again there until an outbreak in Venezuela in 1992–1993 and in Chiapas, Mexico, in 1993. More recently, an outbreak of VEE occurred in northern Colombia and Venezuela in 1995, with about 50,000 equines and 75,000 humans infected, including 3000 humans with neurologic symptoms and 300 fatalities. The rapidity of spread of these outbreaks over large geographic areas undoubtedly is due to the role of horses as competent reservoir hosts. Weaver et al. (2004) have presented a comprehensive review of VEE.

Chikungunya (CHIK) virus. This virus, which has caused outbreaks for many years in Africa and parts of India and southeastern Asia (Jupp and McIntosh, 1988), has recently surged in human populations. In 2004, almost half a million human cases were reported in Africa, primarily coastal Kenya. An outbreak starting in 2005 spread across the islands of the Indian Ocean, resulting by early 2006 in 244,000 cases on Réunion, with an attack rate of 35% and more than 200 deaths. From there it spread to India, causing over 1 million cases. Travelers returning to Italy and France from the islands as Réunion, Mauritius, the Comoros, and Seychelles brought the virus with them. This resulted in cases diagnosed in Europe and a focal epidemic involving local transmission. More recently it has spread to the Americas. Overviews of CHIK virus biology, clinical aspects, and global expansion have been presented by Powers and Logue (2007), Morens and Fauci (2014), and Wahid et al. (2017).

CHIK virus generally does not cause the encephalitis-type symptoms characteristic of EEE, WEE, and VEE viral infections but rather a denguelike arthralgic illness with fever, rash, and severe joint pain. Some infections lead to chronic arthritis. Neurologic and other severe complications occurred in an Indian Ocean epidemic, with deaths reported for the first time.

In Africa, CHIK virus infects nonhuman primates such as the vervet monkey (*Cercopithecus aethiops*) and baboon (*Papio ursinus*), and wild primates often carry antibodies to it, suggesting their zoonotic role in its epidemiology. The enzootic vectors include *Aedes africanus*, *Ae. luteocephalus*, *Ae. opok*, *Ae. furcifer*, *Ae. taylori*, and *Ae. cordellieri* in the savanna and forest cycles. Additional reservoirs appear to include rodents and cattle and the mosquito species that feed on them. The virus appears to lack a zoonotic reservoir in Asia and the Indian Ocean region. Humans infected with CHIK virus often develop viremia sufficient to infect *Ae. aegypti* and *Ae. albopictus*, important vectors in urban and suburban areas.

Aedes aegypti is the primary vector of CHIK virus in urban areas of India and Asia, where it causes epidemics of arthralgic disease during rainy seasons. A 1994 epidemic in Vellore, southern India, was characterized by human cases increasing during August and September, reaching a peak in October as the human-biting frequency and viral infection rate of *Ae. aegypti* increased. The epidemic lasted about 5 months and affected 44% of the city's population. This epidemic and others, along with experimental studies, indicate that interrupted feeding by mosquitoes, resulting in partial bloodmeals, may facilitate both mechanical and biological transmission of CHIK virus, thus rapidly amplifying the virus in human populations.

Increases in CHIK virus activity that started along the East African coast are thought to have been caused not by abundant rainfall but rather by several years of drought. Drought promotes the use of water-storage containers around human habitations, producing large numbers of *Ae. aegypti* as vectors.

The epidemics on the Indian Ocean islands and in Italy and France have involved *Ae. albopictus* as the primary vector, rather than *Ae. aegypti*. A small genetic change in the virus apparently has significantly increased the vector competence of this typically more rural and less anthropophilic mosquito (Schuffenecker et al., 2006). The establishment of *Ae. albopictus* in many temperate areas of the world in recent years increases the opportunities for CHIK virus to spread in other human populations. The virus was discovered in the Americas in 2013, first in the Caribbean Islands and subsequently in many countries in North, Central, and South America, from the United States to Argentina. As of 2016, locally transmitted cases in the continental United States have been reported only from Florida and Texas. The relative roles of *Ae. albopictus* and *Ae. aegypti* in the Americas are under investigation and probably vary with locality. There is the opportunity for CHIK to use nonhuman primates in enzootic cycles in the Americas, though these remain to be documented.

Other Alphaviruses

There are other important alphaviruses that occur endemically and epidemically and cause fever, arthralgia, and other symptoms in humans.

O'nyong-nyong (ONN) virus. This is an antigenic subtype of CHIK virus that is transmitted among humans by *Anopheles* species in widespread parts of Africa. The vectors are the same ones that transmit human malaria parasites (i.e., *An. gambiae* and *An. funestus*). A large epidemic occurred from 1959 through the 1960s, infecting about 2 million people in Uganda, Kenya, Tanzania, Malawi, Zambia, and Mozambique. The virus, whose name comes from an Acholi African word meaning "weakening of the joints," was first isolated from humans during this time and was later isolated from *An. funestus* in 1974. Only isolated cases had occurred since then, until an epidemic in Uganda in 1997. Neither a vertebrate animal host nor the mechanism of persistence of ONN virus between these epidemics is known. The reservoir host is probably humans. A closely related virus, called **Igbo Ora**, also is transmitted by *Anopheles* mosquitoes and occurs in parts of West Africa, where it was associated with an outbreak of CHIK-like disease in the Ivory Coast in 1984. ONN and Igbo Ora are the only arboviruses causing human disease that have *Anopheles* mosquitoes as the primary vectors.

Sindbis (SIN) virus. This virus is distributed widely in eastern Europe, Scandinavia, the former Soviet Union, Asia, Africa, the Middle East, and Australia. The virus is a member of the WEE virus complex. It was originally isolated from *Culex univittatus* in Egypt in 1952 and has been associated with a human disease of rash, fever, and muscle and joint pain in Uganda, South Africa, and Australia. Birds are vertebrate hosts. A subtype of SIN virus is **Ockelbo**, which is distributed in Sweden, Finland, and northern Russia. It is transmitted by *Culiseta ochroptera*, and possibly *Culex* and *Aedes* spp., and has been associated with human disease similar to that caused by SIN virus.

Ross River (RR) virus. This virus occurs in Australia, Fiji, and the Cook Islands, where it causes an illness known as **epidemic polyarthritis**, consisting of fever, rash, and arthralgia (Kay et al., 1988). In Australia, RR virus is transmitted by *Aedes camptorhynchus*, *Ae. Vigilax*, and *Culex annulirostris*, whereas in the islands the vectors are *Ae. aegypti* and *Ae. polynesiensis*. The virus was first isolated from *Ae. vigilax* in 1963. The vertebrate reservoir hosts are uncertain, but in Australia they appear to be marsupials. RR virus ecology and distribution were reviewed by Russell (2002). **Barmah Forest virus** is another mosquito-borne alphavirus in Australia that causes symptoms similar to RR virus.

Mayaro (MAY) virus. This virus occurs in the Caribbean and parts of South America. In humans the illness is similar to CHIK. The virus was first isolated from febrile patients in Trinidad in 1954. Marmosets are the reservoir hosts of the virus, and *Haemagogus* mosquitoes are vectors.

Flaviviridae (*Flavivirus*)

The Flaviviridae, genus *Flavivirus*, contains eight antigenic complexes plus many unassigned viruses involving 70 types, subtypes, and varieties distributed worldwide. Some of these have mosquitoes as vectors while others are associated with ticks or with rodents or bats. The flavivirus

TABLE 15.5 Relationships of Selected Flaviviruses to Mosquito Vectors and Vertebrate Reservoir Hosts

Virus Species	Vector(s)	Vertebrate Reservoirs
Yellow fever	*Aedes aegypti*	Humans in urban environments
	Aedes africanus	Monkeys
	Aedes bromeliae	Monkeys
	Aedes furcifer	Monkeys
	Aedes luteocephalus	Monkeys
	Aedes metallicus	Monkeys
	Aedes taylori	Monkeys
	Aedes vittatus	Monkeys
	Haemagogus spp.	Monkeys
	Sabethes spp.	Monkeys
Dengue	*Aedes aegypti*	Humans
	Aedes albopictus	Humans (monkeys?)
	Aedes niveus group	Monkeys
	Aedes africanus	Monkeys
	Aedes furcifer	Monkeys
	Aedes taylori	Monkeys
	Aedes luteocephalus	Monkeys
	Aedes opok	Monkeys
	Aedes scutellaris	Humans
	Aedes polynesiensis	Humans
	Aedes pseudoscutellaris	Humans
	Aedes rotumae	Humans
Japanese encephalitis	*Culex tritaeniorhynchus*	Birds, pigs
	Culex gelidus	Birds
	Culex vishnui complex	Birds
St. Louis encephalitis	*Culex pipiens*	Birds
	Culex quinquefasciatus	Birds
	Culex tarsalis	Birds
	Culex nigripalpus	Birds
Murray Valley encephalitis	*Culex annulirostris*	Birds
West Nile	*Culex* spp.	Birds
	Culex univittatus	Birds
	Culex modestus	Birds
Zika	*Aedes aegypti*	Humans
	Aedes albopictus	Humans
	Aedes africanus, Ae. luteocephalus	Monkeys

Continued

diseases include some of the most dangerous and historically significant infections of humans. Table 15.5 shows the mosquito—vector and vertebrate—host associations of some of the more important mosquito-borne flaviviruses. Among them are yellow fever, dengue, Japanese encephalitis, St. Louis encephalitis, Murray Valley encephalitis, West Nile, and Ilheus viruses.

Yellow Fever (YF) virus

This disease is caused by yellow fever (YF) virus and occurs over broad portions of lowland equatorial Africa and South and Central America (Fig. 15.22), as either isolated cases or epidemics (Monath, 1988). Yellow fever virus exists principally in two epidemiological forms: an enzootic form, maintained in monkey populations by forest mosquitoes in a sylvan cycle and responsible for most of

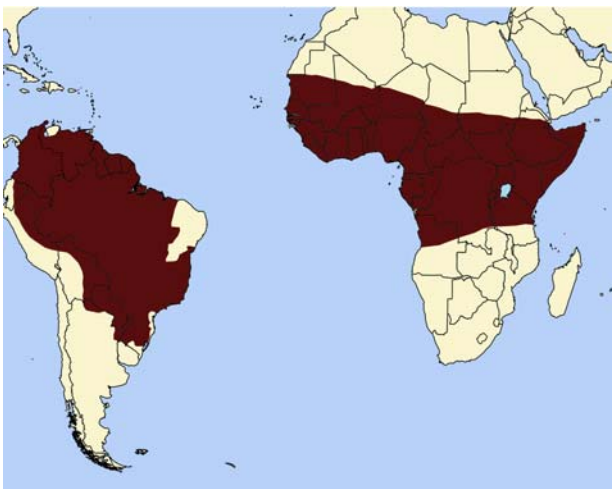


FIGURE 15.22 Geographic distribution of human yellow fever cases. *Based on data from the Centers for Disease Control and Prevention, USA, and the World Health Organization.*

FIGURE 15.23 *Aedes aegypti* female feeding on blood. This is the primary vector of dengue, urban yellow fever, Chikungunya, and Zika viruses. The "lyre" pattern on the thorax is distinctive. *Photograph by Robert G. Hancock.*

the isolated human cases (**jungle yellow fever**), and an epidemic form, spreading rapidly through human populations by the domestic form of *Aedes aegypti* (Fig. 15.23) in an urban cycle. The urban vector is particularly efficient because it readily enters and typically rests in houses, feeds almost exclusively on humans, and oviposits in artificial containers. It frequently takes two or three bloodmeals per gonotrophic cycle to supplement its energy reserves in lieu of sugar-feeding.

In previous centuries, **urban yellow fever** affected subtropical and temperate regions of North America with devastating effects. It remains a serious cause of mortality, particularly in village settings in tropical Africa, and is a constant threat in South America. There are sporadic, annual cases and occasional epidemics in Africa and 50−300 cases of jungle YF annually in South America. Jungle YF occurred in Brazil (1932), Panama, and northward into Central America (1948−1955) and several episodes (including some urban transmission) in Trinidad (1954, 1959, 1978−79). Epidemics in Africa have involved diverse areas, including Ethiopia (1960−1962), the Gambia (1978), Ghana (1979−1980, 1983, 1993, 1996), Benin (1996), Burkina Faso (1983), Nigeria (1986, 1994), and Kenya (1992−1993). Substantial outbreaks have occurred as recently as 2016 in Angola and the Democratic Republic of the Congo, and 2017 in Brazil. The global prospect for continuing YF epidemics was given by Barrett and Higgs (2007).

Yellow fever is a hemorrhagic disease. Mortality in infected humans ranges from 5%−75% or greater. After infection by mosquito bite, there is an incubation period of three to 6 days, followed by sudden high fever (>104°F [40°C]), headache, nausea, and pain. Humans are viremic during this initial acute phase for only about 3 days. The virus is viscerotropic and causes parenchymal cell necrosis in the liver, resulting in elevated blood bilirubin levels and jaundice—thus the name "yellow fever." Jaundice leads to systemic toxemia. Hemorrhage manifests as bleeding gums, easy bruising, and sloughing of the stomach lining, causing a characteristic **black vomit**. Delirium and coma often precede death.

The causative agent of YF is the prototype virus of the Flaviviridae and the first arbovirus to be associated with human disease. Although the disease was first recognized in the New World in the 17th century, the virus was isolated first in 1927 in Ghana, Africa, when the blood of a man with YF was inoculated into rhesus monkeys. The virus apparently originated in Central Africa among monkeys (e.g., *Cercopithecus* spp., family Cercopithecidae) and mosquitoes dwelling in the forest canopy. In this environment, the vector is the monkey-feeding *Aedes africanus*, a relative of *Ae. aegypti*. There also is evidence for infection with chimpanzees (*Pan troglodytes*, family Pongidae), baboons (*Papio*, family Cercopithecidae), and bushbabies (*Galago*, family Lorisidae). Generally, Old World monkeys do not suffer mortality from infection. In western Africa, where YF virus also is enzootic, the likely sylvatic vectors are *Aedes vittatus*, *Ae. metallicus*, *Ae. furcifer*, *Ae. taylori*, and *Ae. luteocephalus*, which develop in tree holes and sometimes other natural containers and rock pools.

Human disease occurs when humans enter the habitat where the mosquito−monkey cycle is ongoing and are bitten by infected mosquitoes. Alternatively, infected monkeys or baboons sometimes enter human habitat on raids in gardens or banana plantations, bringing YF virus to mosquitoes at the interface between forest and human habitation. The African mosquito *Aedes bromeliae* (formerly *Ae. simpsoni*), the larvae of which develop in water-filled leaf axils of plants such as banana, occupies this interface and frequently has become involved in forest-edge and rural transmission of YF virus to humans. *Aedes bromeliae* sometimes has served as the primary vector in massive rural epidemics. Thus, it functions both as a bridge vector between sylvatic and rural cycles and as an interhuman vector in that peridomestic environment. It was the principal vector during the 1960−1962 epidemic in Ethiopia, in which about 100,000 people became infected and 30,000 died.

The spread of both the domestic form of *Aedes aegypti* and YF virus from Africa to other parts of the world apparently occurred within the past 400 years. Trading and slaving ships, with their potentially virus-infected cargoes of slaves and with water barrels as a mosquito development site, greatly facilitated spread and establishment of both vector and virus. The need for slave labor in the newly established sugar cane-molasses-rum economy of the Caribbean region undoubtedly promoted movement of YF virus about the New World. Often, crews became ill with YF while their ships were in transit. Epidemics regularly

occurred in port cities both in West Africa and in coastal South America, North America, and the Caribbean. *Aedes aegypti* also moved into the Arabian peninsula and Indian subcontinent, and then to Asia and the Pacific region, probably via dhow traffic along sea trade routes. Yellow fever has not become established in Asia, even though vector-competent mosquitoes and humans occur there.

Yellow fever epidemics have occurred in the New World over a period of three centuries, beginning in the mid 1600s. Epidemics occurred in such places as Barbados and Trinidad (Caribbean), Havana (Cuba), Yucatan (Mexico), Guadeloupe (Caribbean), and Guayaquil (Ecuador) and in Charleston (South Carolina), Mobile (Alabama), Pensacola (Florida), and other areas of the United States as far north as Boston and New York. In the 1700s and 1800s, epidemics continued in ports of tropical and temperate America, including an outbreak in Philadelphia (Pennsylvania) in 1793, where some 4000 deaths occurred in a population of 55,000. A large outbreak in Haiti in 1802 decimated the French military force there, causing Napoleon to abandon his New World ambitions, contributing to the Louisiana Purchase by the US government. New Orleans (Louisiana) had regular epidemics of YF from 1796 through 1905. The shipping blockade enforced by the navy on the federalist side of the US civil war (1861−1865) prevented YF in New Orleans during that period. Probably, the blockade stopped importation of infected ship crews. An epidemic in the Mississippi River valley in 1878, extending north as far as Gallipolis, Ohio, caused over 13,000 deaths. The last epidemic in the United States was in New Orleans in 1905, involving 3402 cases and 452 deaths.

As YF virus invaded the New World, it became established in a mosquito−monkey cycle in forested parts of Central and South America. In the sylvatic cycle there, the vectors are forest canopy−dwelling mosquitoes, particularly *Haemagogus* spp. and *Sabethes chloropterus*, whose larvae develop in tree holes. New World Monkeys in the family Cebidae are highly susceptible to infection. During epizootics, howler monkeys (*Alouatta* spp.), squirrel monkeys (*Saimiri* spp.), spider monkeys (*Ateles* spp.), and owl monkeys (*Aotus* spp.) may show considerable mortality in their populations. However, capuchin monkeys (*Cebus* spp.) and woolly monkeys (*Lagothrix* spp.) circulate mosquito-infective viremias, but they do not die from infection. Therefore, in this cycle noticeable die-offs of monkeys may or may not precede sylvatic transmission of YF virus to humans. Sylvan YF in the Americas can lead to urban outbreaks when humans enter the tropical forests where transmission is ongoing, become infected by mosquito bite, then return to villages or cities. If *Aedes aegypti* is present in these settlements, it can initiate an epidemic of urban yellow fever.

The history of the discovery that YF virus is transmitted by mosquitoes is intriguing. The discovery is particularly important because YF was the first arbovirus to be recognized as a mosquito-borne agent, and mosquito control measures imposed quickly afterward caused a dramatic reduction in this devastating disease. Although suspicion that the agent causing YF might be transmitted by mosquitoes can be traced to several independent sources in the 1800s, it was the intuition of the Cuban physician **Carlos Finlay** (Fig. 1.2A), followed by the research activities of the United States Yellow Fever Commission in Havana, Cuba, in 1900, that resulted in experimental evidence that *Aedes aegypti* was the vector. This commission was comprised of the US Army officers **Walter Reed** (Fig. 1.2B), **James Carroll**, **Jesse Lazear**, **Aristides Agramonte**, and others. After consulting with Finlay, this team carried out a series of experiments that demonstrated the transmissibility of the agent from infected to uninfected humans by mosquito bite, after a suitable incubation period in mosquitoes. During this work, James Carroll allowed himself to be bitten by an infected mosquito and later developed YF, but he recovered. Jesse Lazear was accidentally bitten, and he died of the disease.

The team's findings stimulated **William Gorgas**, a physician in the United States Army, to impose a control program against *Ae. aegypti*, resulting in the elimination of urban YF in Havana and, soon after, in the Panama Canal Zone. Later, the Rockefeller Foundation sponsored teams of biomedical scientists and public health practitioners to begin intensive studies of YF by establishing a research center in Guayaquil, Ecuador, in 1918. This foundation eventually established research institutes in Brazil, Nigeria, Uganda, the United States, and elsewhere, and supported research and disease control programs in many sites. For a time, the causative agent of YF was thought to be a spirochete. Within 10 years of isolation of the virus in 1927, an attenuated, live vaccine was produced that was shown to provide excellent protection against infection. **Max Theiler** was awarded the Nobel Prize in Medicine for his efforts in this regard. In many areas of South America, antimosquito programs resulted in virtual elimination of urban outbreaks, although sylvan transmission continued. Despite these early advances, cases of jungle and rural YF, and epidemics in Africa, continue to occur. The reintroduction and resurgence of *Ae. aegypti* populations throughout Central and South America and the Caribbean islands in recent decades, and the immense growth of cities that provide a habitat for it, have created an increased potential for urban epidemics. Recent ongoing outbreaks have been reported in Democratic Republic of the Congo (2012), Angola (2015), Uganda (2016), Nigeria (2017), and Brazil (2017), among others.

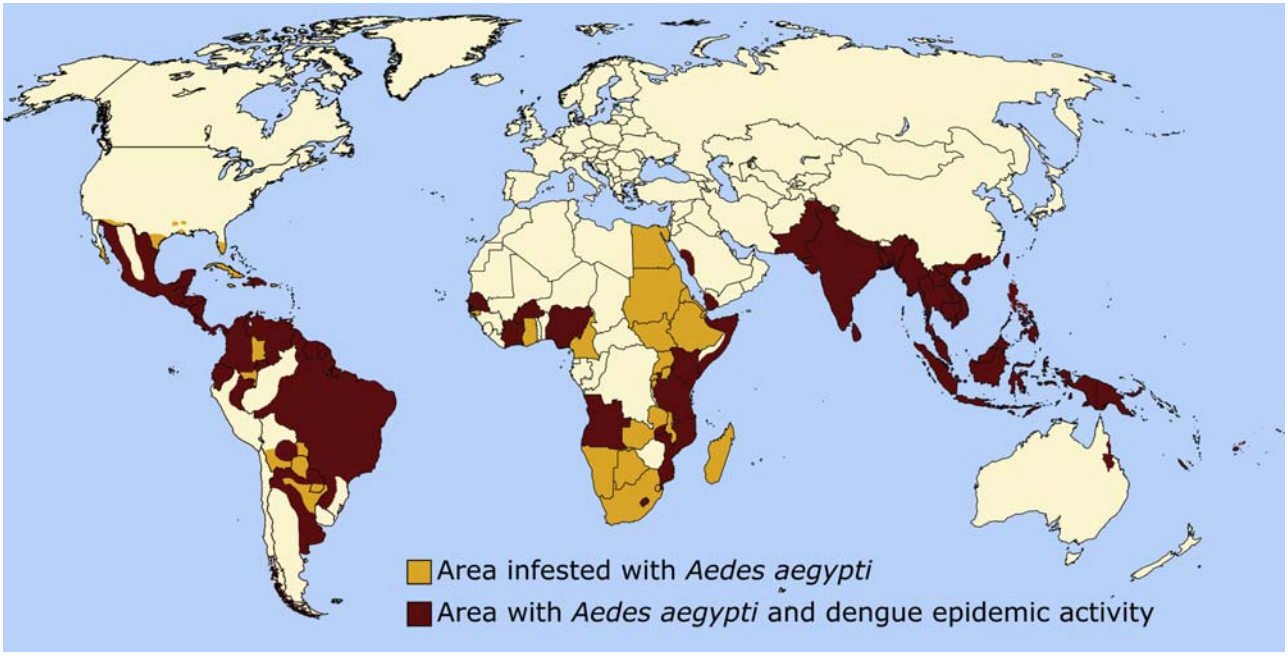


FIGURE 15.24 Geographic distribution of countries reporting human dengue fever cases and infestations with *Aedes aegypti* mosquitoes. *Based on data from the Centers for Disease Control and Prevention, USA, and Barcellos and Lowe, 2014.*

Dengue (DEN) virus

This disease is caused by **dengue (DEN) virus**, represented by four closely related serotypes called dengue 1, 2, 3, and 4 (Gubler, 1988). The disease in humans is either classic dengue fever or the more **severe dengue hemorrhagic fever (DHF)** or **dengue shock syndrome (DSS)**. It has been estimated recently that about 390 million dengue infections occur per year, worldwide. The DEN viruses are transmitted by mosquitoes, principally *Aedes aegypti*. The current distribution of DEN viruses includes Southeast Asia, the south Pacific, the Caribbean basin, Mexico, Central America, and South America. However, epidemics of DEN have occurred elsewhere in the past, including the United States, Japan, Australia, Greece, and both eastern and western Africa. Fig. 15.24 shows its current distribution and the regions with projected risk for occurrence, given the appropriate climate and availability of vectors. Dengue is commonly reported as an introduced disease in the continental United States, but indigenous transmission has occurred in Texas several times, and in both Texas and Florida as recently as 2013. All four serotypes now occur in the Western Hemisphere. In addition, to large-scale epidemics in the Americas, there have been recent, large outbreaks involving dengue hemorrhagic fever in Africa, China, Taiwan, India, Maldives, and Sri Lanka. In the hyperendemic areas of southeastern Asia, such as Thailand and the Philippines, the severe forms of disease have become more common and appear in epidemics at 3- to 5-year intervals.

Dengue is characterized by fever, rash, severe headache, and excruciating pain in muscles and joints, earning it the name **breakbone fever**. Clinical disease develops 5–8 days after bite of an infected mosquito. Often the disease runs a mild course of about a week, leading to complete recovery. However, DEN can become severe in cases of DHF and DSS, both of which generally occur in children and may be fatal. These manifestations were first observed as complications of dengue fever in 1954 and are characterized by blotchy rash, bleeding from the nose and gums (Fig. 15.25), and shock.

The increased frequency of DHF and DSS in Southeast Asia and parts of the Caribbean and Latin America have

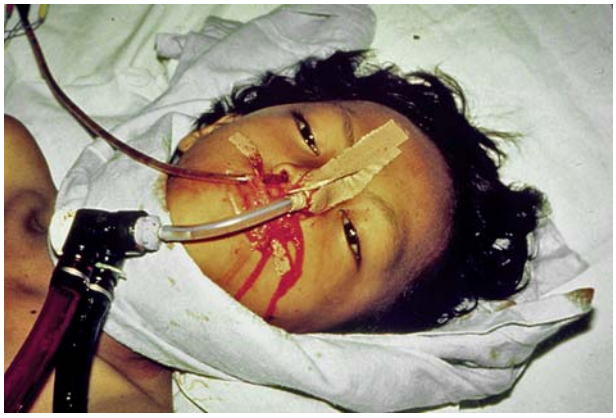


FIGURE 15.25 Dengue hemorrhagic fever (DHF), showing external bleeding from mucous membranes. *Photograph by Duane J. Gubler.*

stimulated a debate in the biomedical community regarding the mechanisms by which DHF emerges during epidemics of classic dengue fever. One hypothesis is that there are variable forms of the different serotypes of DEN viruses, some of which are more pathogenic than others. Another idea is that people of different races differ in propensity to develop severe symptoms and that the virus has been spreading into more susceptible populations. Still another hypothesis is that, as human populations in tropical cities in Asia and the Americas increase and as DEN epidemics in general increase in frequency, there are simply more cases with the noticeable manifestations of DHF and DSS. The fourth hypothesis, and the one currently viewed as most likely, is that prior exposure to one serotype, followed by exposure to another serotype within a critical period of 5 years, leads to the development of hemorrhagic fever and, in some cases, shock syndrome. The more recent epidemics taking place in various parts of the world, especially in the Americas, indicate that DEN has replaced YF as the major urban, epidemic flavivirus of importance, worldwide.

The main epidemic vector of DEN viruses, *Aedes aegypti* (Fig. 15.23), is ideal, because it commonly rests indoors, feeds preferentially on humans, has a tendency to take supplementary bloodmeals, and often moves from one residence to another as it oviposits. The larvae develop in such vessels as water barrels and jars, potted-plant containers, cemetery urns, and discarded tires. The close proximity of these larval habitats to human dwellings further facilitates *Ae. aegypti*—human contact and allows large mosquito populations to develop. To the extent that larval sites are hand-filled, DEN transmission is independent of rainfall patterns. Indeed, in some places where water stores are replenished independently of rainfall, epidemics may occur in the hot, dry season when temperatures are higher and the extrinsic incubation period of the viruses in the mosquitoes is shortened. However, in areas where breeding containers depend primarily on rainwater, DEN epidemics occur during rainy seasons.

Other vectors of DEN viruses are *Aedes albopictus* (Fig. 15.26) in rural areas of Southeast Asia and *Ae. polynesiensis*, *Ae. scutellaris*, *Ae. pseudoscutellaris*, and *Ae. rotumae* in the Pacific region. The role of the newly introduced *Ae. albopictus* in the New World as a vector of DEN viruses remains to be determined. In peninsular Malaysia, a series of studies showed that all four serotypes of DEN virus circulated between monkeys and mosquitoes of the *Ae. niveus* group, suggesting the possibility of an enzootic sylvatic cycle. Similarly, studies in West Africa provide equally strong evidence that monkey populations there maintain a sylvatic form of DEN type 2 virus. Where monkeys and humans are loosely associated in forested areas and human habitations are intermixed with patches of forest, this strain of the virus has been isolated from humans, monkeys, and arboreal mosquitoes such as *Ae. africanus*, *Ae. opok*, *Ae. luteocephalus*, *Ae. furcifer*, and *Ae. taylori,* which appear to transmit the virus indiscriminately among monkeys and humans. Thus, DEN may have a zoonotic reservoir and system of transmission in West Africa similar to that of YF and CHIK viruses in the same region. But in this case the enzootic and endemic systems probably remain distinct and separate from one another.

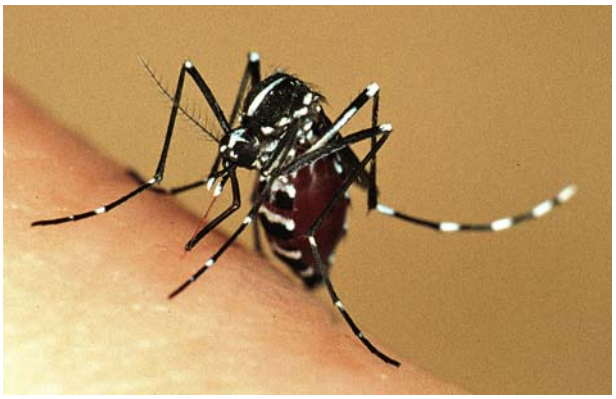


FIGURE 15.26 Asian tiger mosquito (*Aedes albopictus*), female feeding on human. It transmits Chikungunya and and dengue viruses in some locations. The median longitudinal silver stripe on the thorax is its most prominent identifying feature. *Photograph by Woodbridge A. Foster.*

Humans generally are considered to be the only vertebrate host of the endemic/epidemic form of the virus in situations where monkeys do not occur, such as congested urban slums in huge tropical cities including Bangkok, Manila, Jakarta, Caracas, and Guayaquil. Thus, a mosquito—human—mosquito cycle of DEN transmission by *Ae. aegypti* is the usual means of virus maintenance and epidemic spread. Transovarial transmission of some of the DEN viruses has been demonstrated in *Ae. aegypti* and *Ae. albopictus*, and therefore mosquitoes may be reservoirs, particularly in periods of low-level transmission among humans. A multiauthored book on dengue, Gubler et al. [eds] (2014), presents a comprehensive treatment of DEN virus and the disease.

Japanese Encephalitis Virus Complex

Another important group of mosquito-borne flaviviruses is the Japanese encephalitis virus antigenic complex, including West Nile, Japanese encephalitis, St. Louis encephalitis, and Murray Valley encephalitis viruses. Some authorities refer to this complex as the **West Nile antigenic complex**. These viruses occur in widely separated geographic regions but show similarities in the nature of their enzootic cycles. Each has *Culex* vectors (Table 15.5) and birds as vertebrate reservoirs. In the case of Japanese encephalitis virus, pigs often serve as amplifying hosts. Fig. 15.27 shows the worldwide distribution of this complex of viruses. The most important of these in terms of human morbidity and mortality is Japanese encephalitis

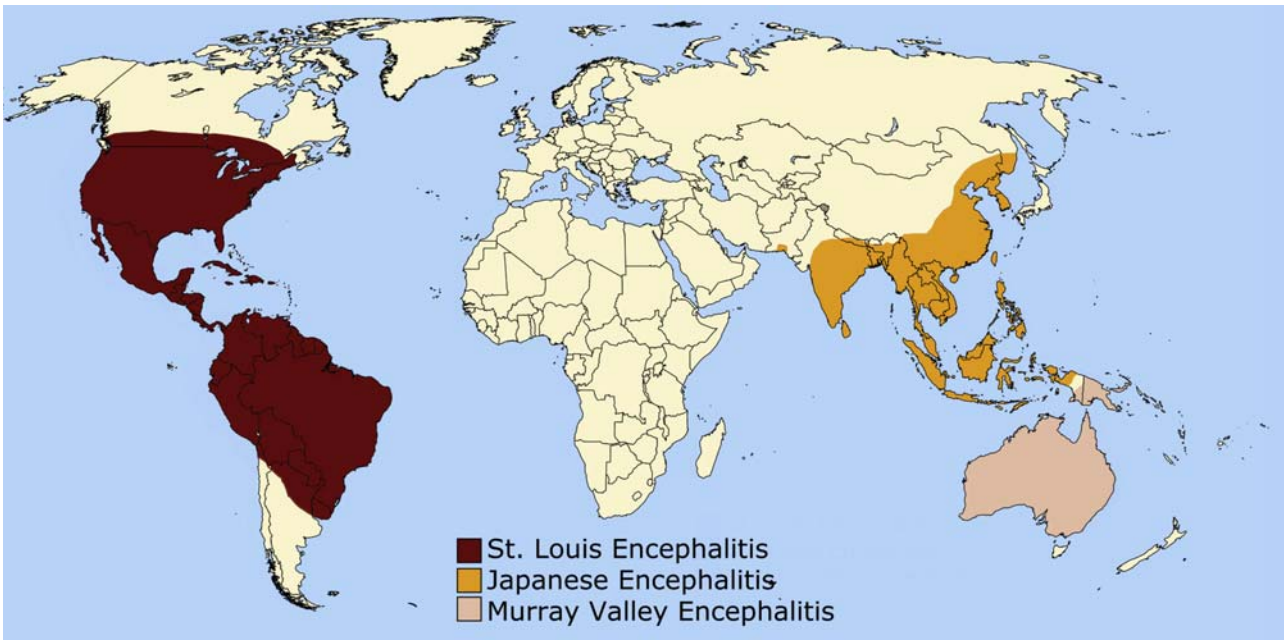


FIGURE 15.27 Geographic distribution of mosquito-borne flaviviruses causing encephalitis. *Reconstructed from Mitchell, 1977, the Centers for Disease Control and Prevention, USA, and other sources.*

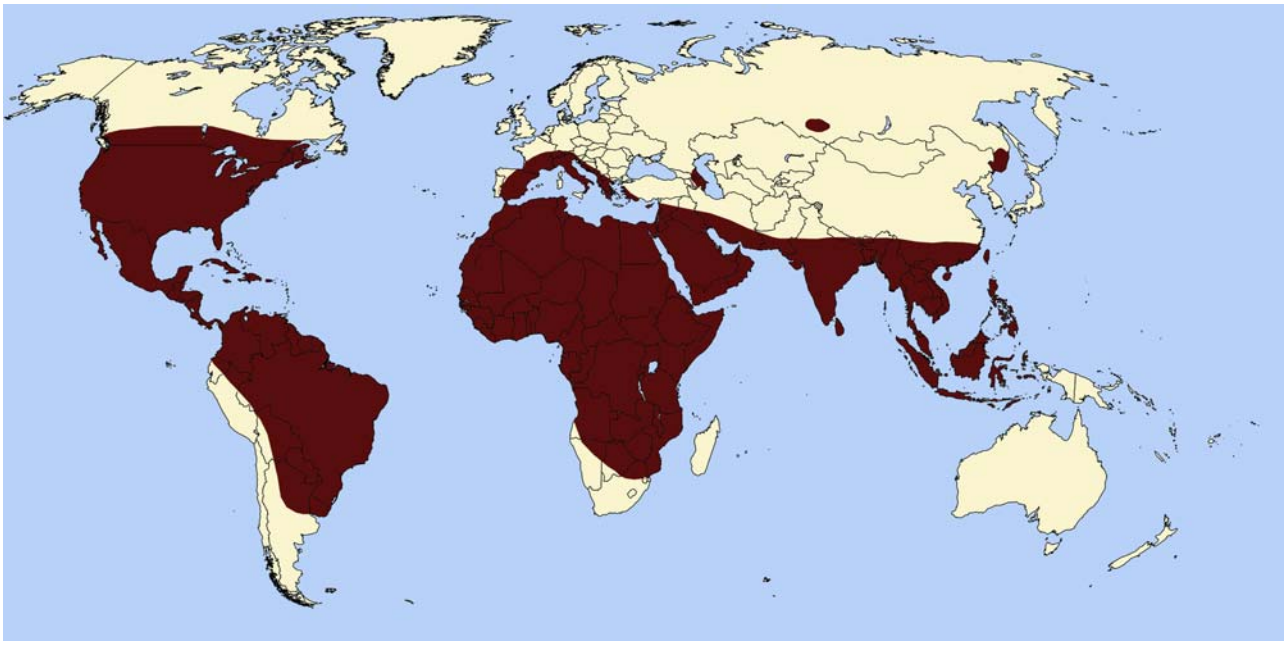


FIGURE 15.28 Geographic distribution of West Nile virus, a mosquito-borne flavivirus. *Based on data from the Centers for Disease Control and Prevention, USA.*

virus. However, all cause human illness of varying severity, depending on the virus and age and health of the person.

West Nile (WN) Virus. This virus was first isolated from the blood of a febrile man in Uganda in 1937. The primary mosquito vectors are *Culex* spp., particularly *Cx. pipiens* and *Cx. univittatus*. Birds are the vertebrate reservoirs and amplifying hosts during epidemics. WN virus is widely distributed in Africa, the Middle East, Europe, parts of the former Soviet Union, India, Indonesia, and North and South America (Fig. 15.28). It has been the cause of endemic and epidemic fever, myalgia, and rash, especially in children in the Middle East, and has caused encephalitis in some instances. Seroprevalence of antibody

to WN virus in Egypt can exceed 60% in adults. Epidemics have occurred in Israel (1950s), southern France (1962), South Africa (1974, and 1983−1984), Romania (1996) and other eastern European countries, and most recently in North America. A review of the epidemiology of WN virus is presented by Kramer et al. (2008).

In 1999, WN virus was introduced through unknown means into New York City, USA, and was linked to human encephalitis in 61 confirmed cases (mostly in the city borough of Queens); there were seven fatalities. Initially, the outbreak was thought to be due to St. Louis encephalitis. However, an unusual feature of the outbreak was the large numbers of birds that died, especially crows (*Corvus brachyrhynchos*). The vectors are believed to have been *Cx. pipiens* and other *Culex* species, although WN virus later was isolated from other mosquito genera as well. A serosurvey conducted by the CDC in the Queens area indicated that as many as 1,250 people had been infected during the course of the outbreak, and that about 240 of these people (19%) may have experienced clinical illness due to infection.

The virus successfully overwintered in the New York area, despite efforts to control *Culex* mosquitoes, and spread to adjoining northeastern states in 2000. By 2001 it had spread into the South and Midwest regions of the United States and Canada. In the Midwest human cases and high bird mortality peaked in 2002. By that time the first human case had already occurred on the West Coast and virus was isolated from birds and mosquitoes. The following year, infections peaked in the South, and highest numbers of human cases occurred in the prairie and Rocky Mountain States, followed by a high incidence of cases in California during 2004 and 2005. The highest virus activity in the United States was recorded in 2003, with more than 9862 human cases of WN, including 264 deaths. Large fluctuations in incidence have occurred since that year. During the period 2002 to 2007, the virus also spread steadily southward into Mexico, the Caribbean islands, Central America, and finally South America as far as Argentina. However, WN virus activity remains remarkably low in much of Latin America. In the United States, total clinical cases between 1999 and 2016 were 46,086, with 2017 deaths. Epidemiological studies indicate that about 80% of human infections are asymptomatic. The great majority of clinical cases are classified by CDC as "West Nile fever," with fewer than 5% presenting "West Nile neuroinvasive disease," including these categories: encephalitis, meningitis, and acute flaccid paralysis (sometimes called WN poliomyelitis).

In the North American outbreaks over 200 species of birds suffered mortality from WN virus infections, with corvids (crows and jays), raptors, and exotic species being particularly vulnerable. About 40% of infected equines (horses, donkeys) died. Bird species vary widely in their tolerance of infections and in their ability to generate high and prolonged viremias. Jays, grackles, crows, house finches, and house sparrows are all highly competent in the latter regard, making them good reservoir hosts.

FIGURE 15.29 *Culex quinquefasciatus* females laying eggs in form of floating rafts; this species is a vector of filariasis, West Nile virus infection, and St. Louis encephalitis, among other diseases. *Photograph by Woodbridge A. Foster.*

Many mosquito species are capable of supporting and transmitting WN virus. In the United States *Culex* species are the principal vectors that maintain it in bird populations: particularly *Cx. pipiens* in the Northeast and upper Midwest, *Cx. quinquefasciatus* (Fig. 15.29) in the South, and *Cx. tarsalis* in several western regions. Mosquitoes that feed on both birds and mammals, such as *Coquillettidia perturbans* and *Aedes vexans*, may act as bridge vectors, transmitting the virus from birds to equines and humans. However, many *Cx. pipiens* populations feed readily on both birds and mammals and can likewise, serve as bridge vectors.

Studies in northeastern North America have shown that a small proportion of infected *Cx. pipiens* adults can pass WN virus to their offspring by transovarial transmission. Thus diapausing adults emerging in the fall may carry the virus through the winter in their hibernacula and transmit it to birds the following spring.

Japanese encephalitis (JE) virus. Japanese encephalitis is a severe disease of acute encephalitis, with children and the elderly primarily affected and with mortality rates reaching over 25% of those with overt disease. Many infections are asymptomatic or mild. Survivors often show neurologic sequelae. The disease is distributed throughout the rice-growing areas of Asia, from Japan south to Papua New Guinea and west to India and Nepal (Fig. 15.27). In Japan, JE epidemics have occurred in August and September in many different years since its discovery there. The virus first was isolated from the brain of a human who died of encephalitis in 1935, then from brain tissue of a

horse in 1937, and from *Culex tritaeniorhynchus* in 1938. Numbers of cases have declined in Japan, Korea, and Taiwan, because of vector control, vaccination, and changes in agricultural practices (Burke and Leake, 1988).

The enzootic cycle of JE virus involves *Cx. tritaeniorhynchus* and several other *Culex* spp. including *Cx. vishnui*, *Cx. pseudovishnui*, *Cx. fuscocephala*, and *Cx. gelidus*, all of which are associated with rice culture. Wading birds such as herons are important enzootic reservoirs. Pigs are important amplifier hosts in rural, rice-growing areas where swine are kept. Japanese encephalitis is probably the most important of the mosquito-borne encephalitis diseases, owing to its epidemic nature, widespread distribution, and large number of humans who acquire infection, die, or recover yet suffer neurologic sequelae.

St. Louis encephalitis (SLE) virus. This virus was identified as the causative agent of disease during an outbreak of encephalitis-like illness in Paris, Illinois (USA), in 1932 and St. Louis, Missouri (USA), in 1933. It was isolated from a patient with encephalitis in the Yakima Valley of Washington (USA) in the early 1940s and found to be a frequent cause of human illness in the Central Valley of California in the 1930s and 1940s. This virus is distributed widely in North America and also occurs in parts of Mexico, Central America, the Caribbean, and South America to Argentina (Fig. 15.27). The encephalitic illness caused by SLE virus shows a bimodal age distribution, with children and elderly people most frequently affected. Attack rates during epidemics range from 5 to 800 per 100,000 population, depending on location, year, strain virulence, and population immunity due to earlier epidemics. In the eastern United States, mortality rates have ranged from about 3% to 20% of laboratory-diagnosed cases, but in the western United States mortality rates are lower.

In North America, three enzootic cycles of SLE virus have been described. In the eastern United States, north of Florida but including Texas, the primary vectors are *Culex pipiens* and *Cx. quinquefasciatus* (Fig. 15.29). The former mosquito occurs in a more northerly distribution, whereas the latter is more southerly, with a hybrid zone at about the latitude of Memphis, Tennessee (USA). Females of both species feed on birds. In addition, *Cx. quinquefasciatus* females frequently feed on mammals as the summer progresses. Whether these mosquitoes alone or other vectors function in transmission to humans depends on the abundance of these two species and other competent vectors. House sparrows are important vertebrate amplifier hosts in peridomestic settings. In the western United States, a mosquito–bird–mosquito cycle similar to that of western equine encephalomyelitis virus, involving *Cx. tarsalis*, has been elucidated. In addition, in California both *Cx. pipiens* and *Cx. quinquefasciatus*, and possibly *Cx. stigmatosoma*, function secondarily as either enzootic or epidemic vectors. In Florida, *Cx. nigripalpus* apparently is the enzootic, epizootic, and epidemic vector of SLE virus. In Latin America and in the Caribbean basin, SLE virus has been isolated from many different species of *Culex*, *Sabethes*, *Mansonia*, *Wyeomyia*, and other genera, and from a wide variety of birds and mammals. In these areas, human SLE generally is rare. The mechanism of virus overwintering in North America is not well known. There is some evidence of virus persistence in overwintering, diapausing female mosquitoes.

The history of SLE in North America has been that of epidemics, either local or widespread, with intervening years when there was apparently no virus activity or epidemics, and either no or a few isolated human cases. The first epidemic, in the early 1930s in St. Louis, Missouri, was accompanied by hot and dry weather, which favored the development of populations of mosquitoes of the *Culex pipiens* complex, the larvae of which develop in water rich in sewage or other organic matter. The epidemic involved about 1,100 human cases and 200 deaths. Since that time, there have been some 50 outbreaks of SLE in the United States. Cases during three recent decades are shown by state in Fig. 15.30. Human cases also have occurred in Manitoba and Ontario, Canada. These epidemics have been both rural and urban. A very large outbreak occurred in 1975, involving 30 states and the District of Columbia, with over 1800 cases reported. More recently, epidemics have occurred in such disparate locations of the United States as Pine Bluff (Arkansas), Florida, Los Angeles (California), Houston (Texas), New Orleans (Louisiana), and Grand Junction (Colorado). A total of 4,437 human cases of SLE infection were reported to the CDC from 1964 to 1995, with an average of 139 cases per year (range, 4–1967). From 2007 to 2016, there were 95 diagnosed cases and 5 deaths. Monath (1980) and Tsai and Mitchell

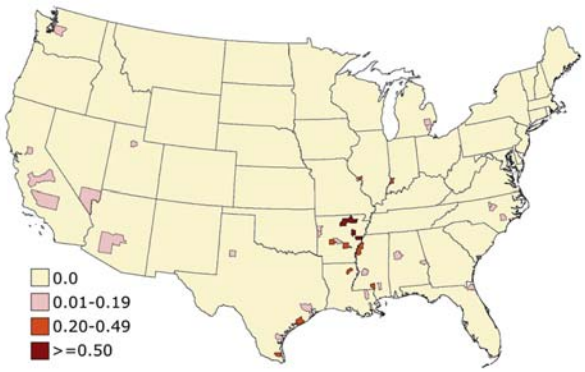


FIGURE 15.30 Historical distribution of human cases of St. Louis encephalitis virus (SLE) neuroinvasive disease (encephalitis and/or meningitis) in the United States; average annual incidence by county of residence per 100,000 population, 2007 to 2016. *Based on data from the Centers for Disease Control and Prevention, USA.*

(1988) have reviewed the ecology and public health significance of SLE.

Murray Valley encephalitis (MVE) virus. This virus has been associated with encephalitis-type illness in humans in eastern and western parts of Australia and in New Guinea (Marshall, 1988) (Fig. 15.27). Mortality rates vary greatly during outbreaks, ranging from 18% to 80%, with long-term neurological effects occurring in 30%−50% of survivors. Epidemics of encephalitis in Australia in 1917, 1918, 1922, and 1925 were probably caused by MVE virus. It was isolated from the brain of a human in 1951 and from a pool of *Culex annulirostris* in 1960. Later periods of transmission were detected in Australia in 1956, 1971, 1974, 1978, 1981, and 1984. A resurgence of virus activity and human infections (17) occurred in 2011, the first outbreak of significance since 1974. The enzootic cycle involves *Cx. annulirostris* as vector and birds as vertebrate reservoir and amplifier hosts. Epidemics appear to be associated with excessive rainfall, which allows an increase of mosquito populations to high densities and the immigration of wading birds. **Kunjin virus** is closely related to MVE virus. It is also mosquito-borne, but it is associated rarely with human disease in Australia.

Zika (ZIK) virus. This virus was first isolated from a sentinel rhesus monkey in Zika Forest, Uganda, in 1947, and soon after in the mosquito *Aedes africanus* in that forest. Human cases were first documented in Africa in 1952. However, the first major outbreak in humans occurred much later, in Micronesia in 2007. It has now spread through nearly all of equatorial Africa, to southern Asia, including Pakistan, India, Bangladesh, and nearly all of Southeast Asia and to many of the South Pacific Islands. More recently it also has been introduced into the Americas, first appearing in Brazil in 2015 and spreading through mainland South, Central, and North America, from central Argentina, through the Caribbean Islands, to the southern United States (Fig. 15.31). Worldwide, cases numbered in the hundreds of thousands by 2016. Within the United States local transmission had been documented by 2016 in limited localities of southern Florida and Texas. As large proportions of vulnerable human populations in the Americas become immune following infection, it is expected that ZIK's incidence will stabilize or decline, as already observed in Brazil between 2015 and 2017. Current figures are available from the CDC Website.

Typical ZIK infection in humans is manifested as a mild febrile illness, including headache, fever, rash, and sometimes joint pain. Death is uncommon. It lasts about 1 week, with many human infections being asymptomatic. However, it has the potential to cause more insidious consequences, apparently due to a single mutation in the virus (Yuan et al., 2017). It was first linked to Guillain-Barré syndrome, a neurological disease. Subsequently, an association was noticed between ZIK and fetal microcephaly and to postinfection neurological disorders in children and adults. These sequelae, particularly the resulting thousands of microcephalic babies infected in utero that have appeared in Brazil and other Latin American countries,

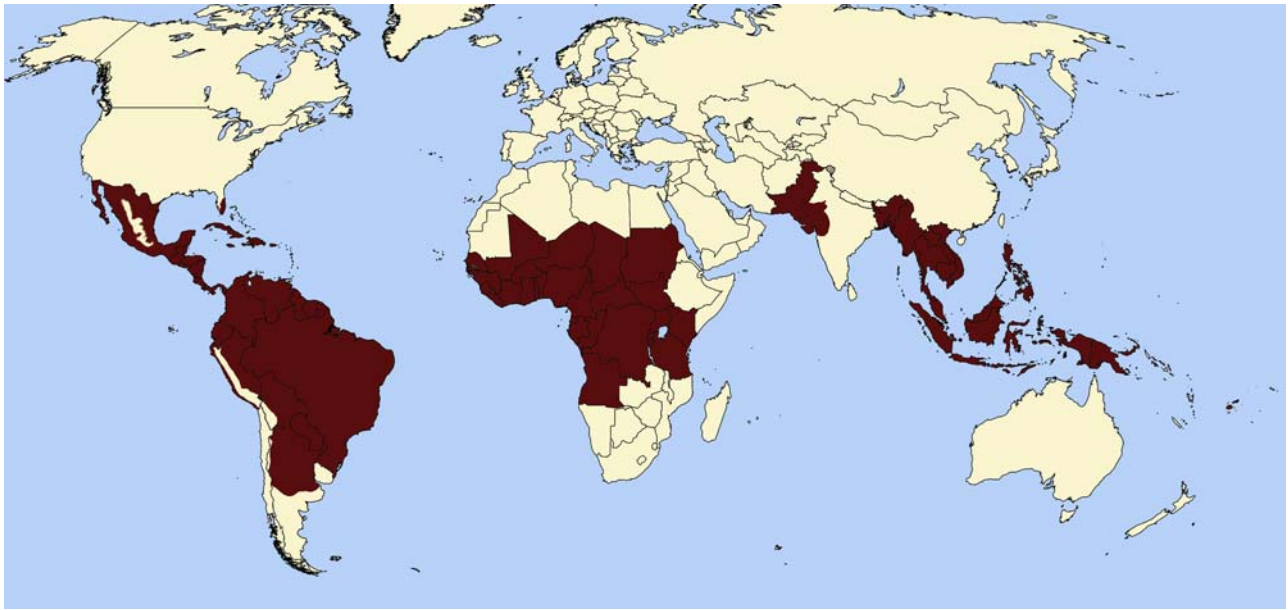


FIGURE 15.31 Approximate global distribution of human infection and likely transmission of Zika virus, as of early 2017; transmission is expected to be rare or absent in deserts and in mountains above 2,000 m; locally acquired cases occurred in southern Florida and Texas, USA. *Based on data from the Centers for Disease Control and Prevention, USA, and individual national reports from some countries.*

have made this a disease more alarming than dengue and CHIK virus infections that also threaten subtropical and temperate regions.

Transmission of ZIK appears to be achieved primarily by the same daytime biting, container-breeding peridomestic *Aedes aegypti* and *Ae. albopictus* that deliver DEN and CHIK viruses to people. As with those viruses, in most instances *Ae. aegypti* appears to be the main vector, because of its stronger preference for human hosts and utilization of water associated with houses. *Ae. albopictus* is less narrow in its blood-feeding tendencies, lowering its efficiency as a vector between humans. But it presents more of a threat in cooler climates because of its ability to undergo a winter egg diapause. Countering that threat is the higher animal-biting rate of *Ae. albopictus*, the shorter warm season, and the greater avoidance or protection from mosquito bites that humans use in temperate countries. Evidence suggests that most animals do not serve as alternate hosts, and therefore that zoonotic reservoirs for ZIK are unlikely in temperate and urban areas. However, the role of primates as reservoirs in subtropical and tropical countries is likely, considering its isolation from various primates in Africa, and more recent detection in monkeys and marmosets in Brazil (Favoretto et al., 2016). One very unusual characteristic of ZIK virus transmission is its ability to be sexually transmitted between humans, particularly from men to women. This means that infected, and even asymptomatic, males pose a risk to females and their offspring during pregnancy. For a review of ZIK virus and a perspective on recent ZIK outbreaks, see Weaver et al. (2016), Shuaib et al. (2016), Fauci and Morens (2016), and Aliota et al. (2017).

Other Flaviviruses

There are many other mosquito-borne flaviviruses of importance to human health, including Rocio virus and Ilheus virus.

Rocio (ROC) virus. This virus occurs in Brazil (Iversson, 1988). It appeared suddenly in São Paulo State in 1975 and caused an encephalitis-type illness in about 1,100 cases over the following 5 years, with some fatalities. Most cases occurred among young, male agricultural workers. Human infections have occurred sporadically since that time, most recently in 2010. The vector and vertebrate host relationships for this virus are poorly known, although *Psorophora ferox* and *Aedes scapularis* have been incriminated as vectors, based on virus isolation in the field and laboratory transmission experiments.

Ilheus (ILH) virus, which is closely related to Rocio virus, occurs in Trinidad, Panama, and parts of South America. It has been associated with about 10 documented human cases of illness and has been isolated from mosquitoes in eight different genera, mostly from *Psorophora* spp.

Bunyaviridae (*Orthobunyavirus* and *Phlebovirus*)

The Bunyaviridae include the genera *Orthobunyavirus* and *Phlebovirus*. The viruses in the genus *Orthobunyavirus* comprise a complex and diverse group of more than 50 virus species distributed worldwide. Mosquitoes or biting midges serve as vectors, and small mammals, ungulates, or birds are the vertebrate hosts. *Phlebovirus* contains 37 viruses, most of which have phlebotomine sand fly vectors. It also includes the important mosquito-borne Rift Valley Fever virus.

Among the 30 virus species associated with mosquitoes and with human or animal diseases in the genus *Orthobunyavirus* are 14 serotypes in the species California encephalitis virus (Grimstad, 1988; Eldridge, 1990; Fauquet et al., 2005). Table 15.6 lists these virus serotypes and indicates their geographic distribution, mosquito vectors, and vertebrate host relationships. Characteristically they are transmitted by *Aedes* spp., but other genera such as *Culiseta* and *Anopheles* may be involved. Nine occur only in North America. The others are distributed in various places worldwide. The viruses in this species were isolated and described during the period from 1943 through the 1970s, culminating in a monograph by Calisher and Thompson (1983). Some of them are described next.

California encephalitis (CE) virus. This is the prototype virus for the complex of the same name and was the first of the California serogroup viruses to be associated with human disease, involving three cases in California (USA) in 1943. It was isolated from *Aedes melanimon* and *Culex tarsalis* at that place and time. Extensive studies of the mosquito and vertebrate—host relationships of this virus in California have shown that *Ae. melanimon* and *Ae. dorsalis* are the principal vectors and that the virus is transmitted transovarially by these species. Serologic surveys have implicated jackrabbits, cottontail rabbits, California ground squirrels, and kangaroo rats as vertebrate hosts. The virus also has been isolated in New Mexico, Utah, and Texas (USA), and in Canada. However, since the time of original discovery, CE virus only rarely has been associated with human disease.

La Crosse encephalitis (LAC) virus. This is the most important human pathogen in the California serogroup, causing an acute, febrile illness in children. Most cases are subclinical or mild, but some progress to severe encephalitis and, rarely, death. In 1964, LAC virus was isolated from preserved brain tissue of a child who had died of

TABLE 15.6 California Encephalitis Virus (*Orthobunyavirus*, Bunyaviridae): Mosquito Associations, Vertebrate Hosts, and Geographic Distribution of the Serotypes of This Virus Species

Serotype	Vector Associations	Vertebrate Associations	Geographic Distribution
California encephalitis	*Aedes melanimon* *Ae. dorsalis*	Lagomorphs	United States (western, southwestern)
Inkoo	*Aedes* spp.	Lagomorphs?	Finland
La Crosse	*Ae. triseriatus*	Sciurid rodents, foxes	United States (eastern)
Snowshoe Hare	*Ae. stimulans* group *Ae. canadensis* *Culiseta inornata*	Lagomorphs	United States (northern), Canada
San Angelo	*Aedes, Anopheles, Psorophora*?	Unknown	United States (southwestern)
Tahyna	*Ae. vexans, Cs. annulata*	Lagomorphs	Europe, Tajikistan, Azerbaijan
Lumbo	*Ae. pembaensis*?	Unknown	East Africa
Melao	*Ae. scapularis*?	Unknown	Trinidad, Brazil, Panama
Jamestown Canyon[a]	*Cs. inornata* *Ae. communis group* *Ae. provocans* *Ae. abserratus* *Ae. intrudens* *Ae. stimulans group* *Anopheles* spp.	Deer	United States, Canada
South River		Deer?	United States (northeastern)
Keystone	*Ae. atlanticus, Ae. tormentor*	Lagomorphs, cotton rats	United States (coastal, eastern)
Serra do Navio	*Ae. fulvus*	Unknown	Brazil (Amapa state)
Trivittatus	*Ae. trivittatus*	Lagomorphs	United States
Guaroa	*Anopheles* spp.	Unknown	Panama, Colombia, Brazil

[a]*Jerry Slough virus is a strain of Jamestown Canyon virus in the western United States.*

encephalitis in 1960 in the vicinity of La Crosse, Wisconsin (USA). It currently is distributed in the eastern United States, including the midwestern states bordering the Great Lakes, east to New York and Pennsylvania, south to West Virginia and North Carolina, and west to Texas. However, most human cases occur in West Virginia, Wisconsin, Illinois, Indiana, and Ohio.

The disease tends to be highly focal within its known range, such that particular regions or towns are known to be endemic. Prevalence varies regionally. In the United States, there were 2,245 cases of LAC reported to the CDC from 1964 to 1995, with an average of 70 per year (range, 29−160). In Ohio, where cases are particularly well documented, there was an average of 26 cases per year between 1963 and 1995. La Crosse encephalitis probably is underreported to public health agencies.

The principal vector of LAC virus is the Eastern tree hole mosquito, *Aedes triseriatus*. The virus is transmitted both horizontally to sciurid rodents, particularly chipmunks and squirrels, and vertically from female mosquitoes to their progeny. The discovery of transovarial transmission of LAC virus was one of the first documentations of this phenomenon in mosquitoes and revealed an overwintering mechanism for LAC virus. It also demonstrated that vertebrate reservoirs were not always essential to the persistence of mosquito-borne viruses in nature and that the mosquito itself could be a reservoir host. Thus an infected female is able to transmit the virus at its first blood-feeding without previously having taken an infectious bloodmeal. Another important finding was that *Ae. triseriatus* males, infected transovarially, transferred LAC virus to females via mating (i.e., venereal transmission).

Epidemiologic investigations of cases of encephalitis or aseptic meningitis of unknown origin often reveal LAC encephalitis in areas where previously it was unknown. Such investigations almost always reveal populations of *Aedes triseriatus* in the immediate vicinity where infection was thought to occur, such as backyards or wooded areas where children play. Water-filled artificial containers, particularly discarded tires, have become important habitats for *Ae.*

triseriatus larvae and provide a link between the sylvan La Crosse cycle and humans. In Ohio and New York State, LAC virus also has been isolated repeatedly from *Aedes canadensis*; however, the role of this mosquito as a vector to humans and its role in an enzootic cycle are not well understood.

Snowshoe Hare (SSH) virus. This pathogen is closely related to La Crosse virus, but its ecology is very different. It originally was isolated from the blood of a snowshoe hare in Montana in 1958. Lagomorphs (hares and rabbits) are the enzootic vertebrate hosts. Snowshoe hare virus is distributed in the northern parts of the United States and in Canada, where it has been isolated from a variety of *Aedes* spp. and from *Culiseta inornata*. Even though SSH virus is very similar antigenically to La Crosse virus, human disease rarely has been documented except in Ontario, Quebec, and Nova Scotia (Canada), where 10 cases of an encephalitis-like illness have been attributed to SSH virus.

Keystone (KEY) virus. This was first isolated in 1964 from a collection of blood-fed *Aedes atlanticus* and *Ae. tormentor* in Florida (USA). It is not considered to be a human pathogen. It occurs along the eastern seaboard of the United States where it has been isolated from *Ae. atlanticus*, *Ae. tormentor*, *Ae. infirmatus*, and other mosquitoes. Transovarial transmission of the virus has been demonstrated for *Ae. atlanticus* in the field. Gray squirrels and cottontail rabbits in northern coastal areas, and cottontail rabbits and cotton rats in Florida and Texas (USA), have been identified as vertebrate hosts.

Trivittatus (TVT) virus. This was first isolated from *Aedes trivittatus* in North Dakota (USA) in 1948. It also has been isolated from other mosquitoes, including *Ae. infirmatus* in the southeastern USA, where *Ae. trivittatus* is absent. Transovarial transmission has been demonstrated in the latter species. Trivittatus virus shows a widespread distribution in the eastern half of the USA. Cottontail rabbits are vertebrate hosts.

Jamestown Canyon (JC) virus. This was originally isolated from *Culiseta inornata* in Colorado (USA) in 1961. Since that time it has been isolated in both Canada and the United States from *Aedes*, *Culiseta,* and *Anopheles* species in regions from Alaska east to Ontario and New England, south to Maryland, and in western and southwestern states, including California. The principal vectors are *Aedes* spp. with univoltine life cycles (i.e., snow pool and spring species). An antigenic strain known only from California (USA) is **Jerry Slough virus**, which is transmitted by *Culiseta inornata*. In the eastern USA, a JC-related serotype is **South River virus**. Transovarial transmission of JC virus has been demonstrated in some mosquito species. Its vertebrate hosts are large wild ungulates, especially deer. JC virus has been associated with encephalitis-type illness in humans in Ontario, New York, and Michigan.

Tahyna (TAH) virus. This is distributed widely in Europe and parts of western Asia. It has been associated with human febrile and central nervous system illnesses in France, the former Czechoslovakia, and Tajikistan. Foci are now known from Finland south to Tajikistan. Although the prevalence of infection in humans is poorly known, serosurveys in the Rhine River valley of Germany documented antibody to TAH virus in up to 23% of humans living in the area. In the former Czechoslovakia, TAH virus was implicated in 1% febrile illnesses of children in an endemic area, and 20% of central nervous system illnesses. This virus was first isolated from *Aedes vexans* and *Aedes caspius* in Slovakia in 1958. The mosquito vectors are *Ae. vexans*, *Ae. caspius*, and *Culiseta annulata*. Hares and pigs are vertebrate reservoir hosts. **Lumbo virus** is a variety of TAH and occurs in parts of Africa.

Rift Valley fever (RVF) virus. This pathogen (Fig. 15.20) is classified with viruses in the genus *Phlebovirus* in the Bunyaviridae (Meegan and Bailey, 1988). It is distributed in eastern Africa north to Egypt, and in parts of West Africa, where it has been associated with large outbreaks of acute illness in livestock (see "Veterinary Importance" later). Humans may become infected by mosquito bite, or more commonly by contact with virus-contaminated blood or through inhalation of virus in aerosols during slaughter of livestock. Humans rarely die of infection but develop an illness including fever, headache, myalgia, retinitis, and in rare cases liver involvement. Large epizootics and epidemics have occurred in South Africa (1950–1951, 1953), Zimbabwe (1968–1969), Egypt (1977–1978, 1993), Mauritania (1987), and Kenya and Somalia (1997–1998). These outbreaks generally involved thousands to hundreds of thousands of cases in livestock. The epidemic in Egypt in 1977–1978 probably involved some 200,000 human cases and 600 deaths. The epidemic in Mauritania involved about 1,000 human cases and 50 deaths. In late 2006, an epidemic of RVF virus began in Kenya and spread to Somalia and Tanzania, causing 1062 confirmed human illnesses and 315 deaths, a higher case-fatality rate than in previous outbreaks.

A variety of mosquito vectors have been associated with RVF virus, including *Culex pipiens* in the Egyptian outbreak. In parts of the eastern African savanna, an enzootic cycle of RVF virus has been identified in and around *dambos*, low-lying temporary wetlands. Prolonged rainfall floods these areas and allows development of large populations of *Aedes mcintoshi* and other *Aedes* spp. These mosquitoes maintain RVF virus through transovarial transmission and transmit it to domestic and wild ungulates

that come to the dambos for water, functioning as enzootic vectors. As amplification ensues, epizootic vectors such as *Culex theileri* become important in transmission to domestic livestock. RVF has been reviewed by Linthicum et al. (2016).

Malaria

Malaria is one of the most widespread and prevalent of infectious human diseases. It is caused by sporozoan protists that infect blood tissues and other organs of the body, primarily the liver. The organisms are transmitted by *Anopheles* mosquitoes. The word "malaria" derives from the Italian *mala aria*, or "bad air." Another term for malaria is **paludism** from the French *paludisme* and the Spanish *paludismo*. Both of these words are derived from the Latin *palus*, meaning "swamp." The connotation is that malaria was contracted through association with swamps and inhaling the bad air emanating from them. In English-speaking countries, malaria was called **ague** from the Old French *agu* ("sharp"), from the context of the Latin *febris acuta*, or acute fever. Ague referred to the cyclic fevers and chills, or **paroxysms**, which are characteristic of malaria.

The organisms that cause human malaria are protozoans of the genus *Plasmodium*, family Plasmodiidae (Order Haemosporidida, Class Haemosporidea, Phylum Sporozoa). They are obligate intracellular parasites. There are four species of exclusively human malaria: *P. falciparum*, causing **malignant tertian malaria**; *P. vivax*, causing **benign tertian malaria**; *P. malariae*, causing **quartan malaria**; and *P. ovale*. The first two species are widespread in the tropics, whereas *P. vivax* also occurs in some temperate areas. *Plasmodium malariae* also is distributed widely but less commonly, and *P. ovale* is rare, occurring mainly in Africa. Recently, a fifth species of *Plasmodium* that causes **quotidian malaria**, *P. knowlesi*, has been discovered to be relatively common in the human population of some countries of Southeast Asia (28%–84% of malaria cases in Malaysian Borneo). Heretofore this species had been known to occur almost exclusively in monkeys, with only occasional human infections (see Collins, 2012). Evidence indicates that similar *Plasmodium* anthropozoonoses occur in Venezuela and Brazil, involving the primate malarias *P. brasilianum* (Lalremruata et al., 2015) and *P. simium* (Brasil et al., 2017), respectively. Primate malarias are discussed later in this chapter.

Currently, 1.6 billion people are at direct risk of malaria infection via mosquito bite. Globally, in 2016 an estimated 216 million cases occurred, including 445,000 deaths. Of that total, 194 million cases and 407,000 deaths occurred in Africa (WHO World Malaria Report, 2017). Travelers may become infected during visits to endemic areas. Malaria currently occurs in and parts of northern Africa and most of southern Africa; the Middle East to Iran, Afghanistan, and Pakistan; India and Sri Lanka; parts of China, and Southeast Asia, including Indonesia, the Philippines, Irian Jaya, New Guinea; and Latin America from Mexico through Central America to most of the northern half of South America (Fig. 15.32).

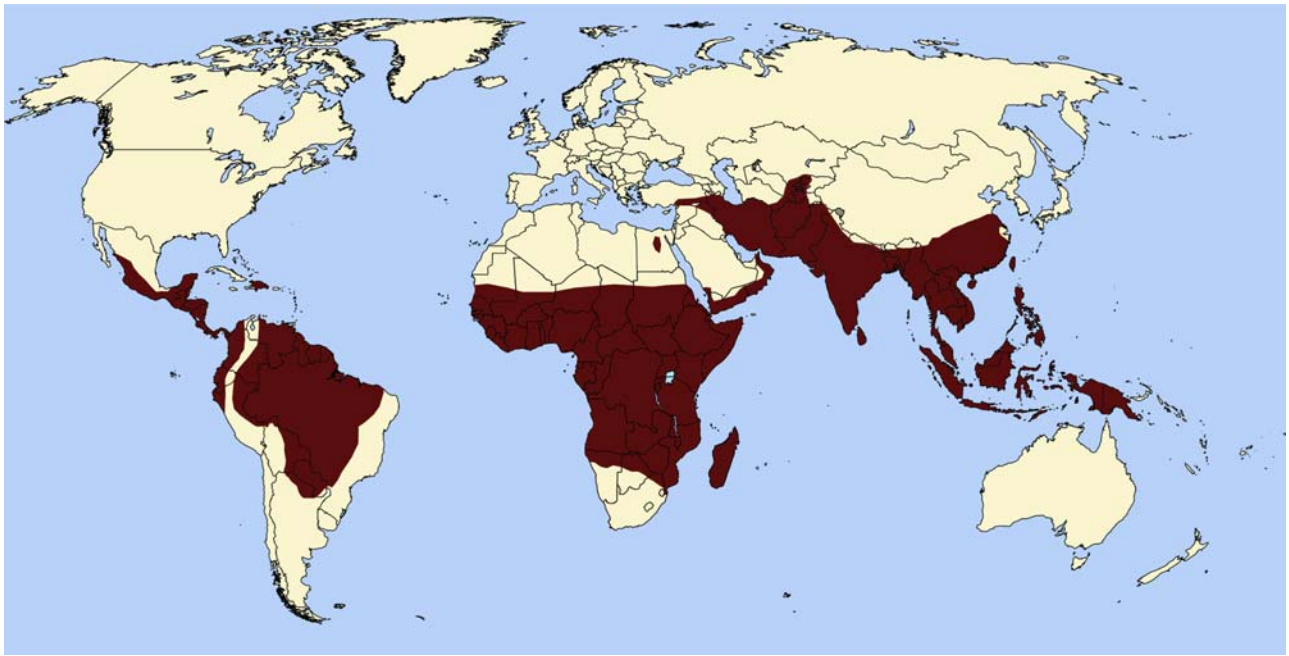


FIGURE 15.32 Geographic distribution of human malaria. *Based on data from the Centers for Disease Control and Prevention, USA.*

The degree of endemicity, or disease prevalence, depends on a variety of factors, including the species of malaria present, environmental and social factors, and species of vectors present. Human malaria is thought not to have occurred in the Western Hemisphere prior to the period of European exploration, colonization, importation of African slaves, and establishment of intercontinental trade.

The literature on human malaria and its vectors is voluminous. A comprehensive source is Gilles and Warrell (1994). Other sources include Boyd (1949), Macdonald (1957), Molineaux and Gramiccia (1980), Wernsdorfer and McGregor (1988), and Strickland (1991).

Plasmodium Life Cycle

The complex life cycle of *Plasmodium* spp. involves both sexual and asexual reproduction. The sexual phase (**gametogony**) begins in the blood of the human host and is completed within the lumen of the midgut of the mosquito. The first phase of asexual reproduction (**sporogony**) occurs on the outer wall of the midgut. The second phase of asexual reproduction occurs first in the liver and later in the blood of the human host. The process in both sites is termed **schizogony** or **merogony**. Sporogony is often referred to as the **exogenous phase** of malaria parasite development, because it occurs outside of the human host. Conversely, merogony is often referred to as the **endogenous phase** of development within the human host. These two developmental phases are depicted in Fig. 15.33 and discussed in more detail later.

An *Anopheles* female becomes infected with malarial parasites when she ingests blood containing red blood cells that are infected with **gametocytes**, specifically the sexual microgametocyte and macrogametocyte stages of the parasite. A microgametocyte bursts from its host red blood cell within the bloodmeal in the midgut lumen of the mosquito, where it extends four to eight flagella-like forms called **microgametes** in a process termed **exflagellation**. A macrogametocyte sheds the erythrocytic membrane and transforms into a single mature macrogamete. One microgamete locates and fertilizes a macrogamete, forming a diploid zygote. The zygote transforms into a motile **ookinete**. The ookinete passes through the peritrophic membrane, then through the midgut epithelial cell membrane, and forms an **oocyst** between the midgut epithelial cells and the basement membrane of the epithelium. A single, malaria-infected *Anopheles* may have few to hundreds of oocysts, depending on the original number of gametocyte-infected red blood cells in the bloodmeal and on the number of macrogametocytes that become fertilized. During sporogony, the encysted parasite becomes haploid again and undergoes multiple mitoses until the oocyst contains thousands of motile **sporozoites**. The oocyst bursts, releasing sporozoites, which make their way to the salivary glands and penetrate the secretory cells. The sporozoites

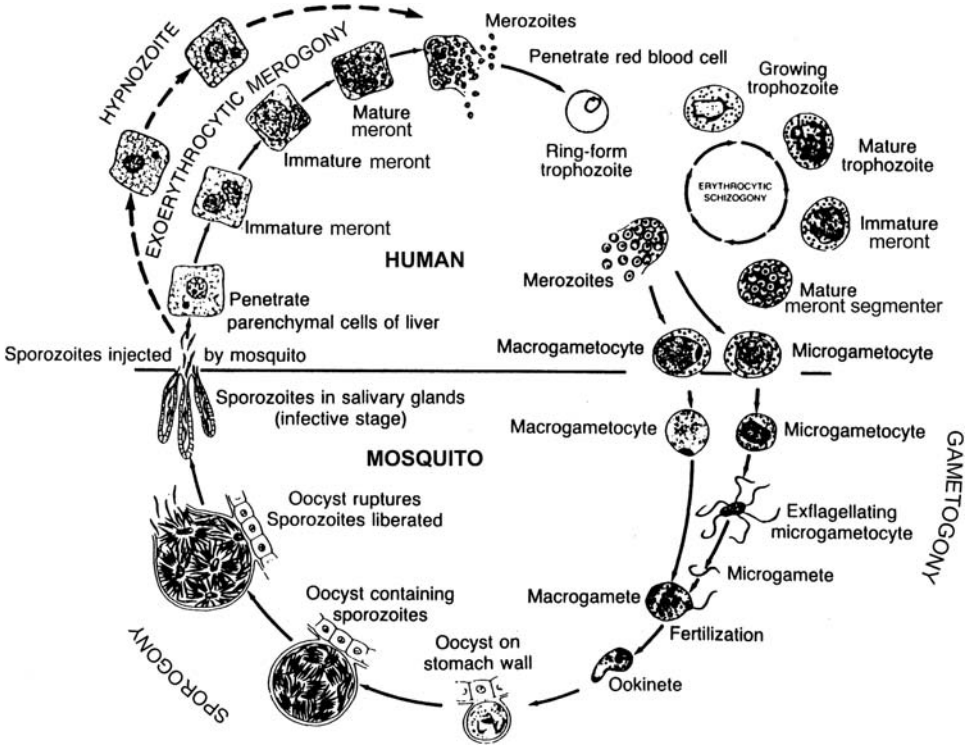


FIGURE 15.33 Life cycle of *Plasmodium vivax* in the human and *Anopheles* hosts. *Modified from Strickland, 1991.*

accumulate within these cells, with some also passing into the salivary ducts. The mosquito then is infective. The sporozoites enter a human host when the mosquito probes and the salivary gland cells release saliva into the skin prior to ingesting blood. The period of time between ingestion of gametocytes and infection of the salivary glands with sporozoites is the extrinsic incubation period.

In the human host, sporozoites migrate in the blood to the liver within minutes of entering subdermal capillaries. They invade liver parenchymal cells where they typically form a **primary tissue meront**. In the case of *P. vivax* and *P. ovale* they may form a **hypnozoite**, a quiescent or resting stage of the parasite. Inside each meront, **merozoites** develop through the process of exoerythrocytic merogony. The meront then bursts, releasing merozoites into the blood stream, where they circulate and invade red blood cells. The merozoites form meronts in the red blood cells, where they produce more merozoites through a process called erythrocytic merogony. An invasive merozoite, once inside a red blood cell, transforms into a **trophozoite**, which uses hemoglobin for nutrients, then into a segmenter form distinguished by dark dots of heme in the red blood cell, and finally into a mature meront, which produces still more merozoites. Merozoites released from infected red blood cells invade new red blood cells where the process of erythrocytic merogony begins anew. Other invasive merozoites form microgametocytes or macrogametocytes within the blood cells, which will mature into microgametes and macrogametes if they are ingested by *Anopheles* mosquitoes during blood-feeding.

Clinical Disease

Malaria is characterized by sudden paroxysms of fever and chills that recur at highly predictable intervals, often in the afternoon. Other acute symptoms include headache, lethargy, fatigue, and profuse sweating after each bout of fever. After infection of erythrocytes by merozoites, erythrocytic merogony leads to the synchronous rupture of the erythrocytes and release of new merozoites, toxins, and heme digestion products. This event in the circulatory system prompts each episode of chills and fever. The next episode occurs in 24, 48, or 72 h, depending on the species of *Plasmodium*.

Malarial infections in humans can result in severe illness and sometimes death. However, the particular symptoms, including timing and severity, vary with the species of *Plasmodium*. The most severe form of malaria is caused by *P. falciparum*, in which merozoites invade both young and old red blood cells. Over time, repeated reinvasions and mass destruction of red blood cells may lead to high parasitemia, severe anemia, and anoxia of tissues. In some cases, hemolysis results in a condition of hemoglobinuria, called **blackwater fever** when the urine contains hemoglobin and turns reddish-brown. Toxins from dead red blood cells stimulate macrophages to produce chemicals such as tumor necrosis factor and other cytokines, which cause characteristic malaria symptoms such as fever. In falciparum malaria, infected red blood cells stick to the vascular epithelium of capillaries in organs including the brain, impeding blood flow and causing a serious and sometimes lethal condition called **cerebral malaria**. Because of this affinity for internal organs, only very young trophozoites and gametocytes are common in peripheral blood. Malaria caused by *P. falciparum* is called **malignant tertian malaria** because of the severity of symptoms and because of the typical 48-h interval between paroxysms. The term "tertian" for a 2-day cycle originated from counting the day when the paroxysm occurs as the first day, so that the next paroxysm occurs on the third. Left untreated, nonfatal infections with *P. falciparum* last 5 months or more, depending on the immune status of the individual.

Plasmodium vivax malaria is called **benign tertian malaria** because symptoms are less severe than *P. falciparum* malaria, and death rarely occurs. Paroxysms occur on a 48-h cycle. In this type of malaria, the merozoites invade only immature red blood cells, called reticulocytes, which typically comprise less than 6% of the total red blood cell count in circulation. Thus vivax malaria, compared to falciparum malaria, has less severe symptoms of anemia and toxemia, making death unlikely. The infected red blood cells do not stick to the epithelial lining of capillaries as they do in falciparum malaria. Vivax malaria can evolve into chronic infection with development of an enlarged spleen, or **splenomegaly**. However, persons infected with other malarias also may have enlarged spleens, as these organs work to replace red blood cells lost to infection. The hypnozoite stage of *P. vivax* provides a mechanism for the parasite to overwinter in humans in temperate areas with short transmission seasons. The period between infection and onset of symptoms can last up to 9 months. Untreated infection persists in the body for many months to many years, with relapses recurring at irregular intervals after initial infection and acute onset of disease.

Plasmodium ovale is a less common tertian malaria with milder symptoms. Its course of infection is similar to that of *P. vivax*. Two subspecies of *P. ovale* have been detected by genetic means, *P. ovale curtisi* and *P. ovale wallikeri*. These appear to be distinct nonrecombinant lineages and may be valid species. Though they are sympatric in many regions and morphologically identical, certain biological and clinical differences are emerging.

Plasmodium knowlesi, a species previously known only as one of the monkey malarias, morphologically similar to *P. malariae*, has a 24-h or quotidian cycle of paroxysms. The high frequency of asexual reproduction in red blood cells results in hyperparasitemia, making it especially

virulent if left untreated. Still another monkey malaria species that recently has been detected in a Brazilian human population is *P. simium*, which is closely allied to *P. vivax*.

Plasmodium malariae, which causes **quartan malaria**, differs from *P. vivax* and *P. falciparum* in that the parasites invade only mature erythrocytes. Therefore, symptoms can be more severe than in vivax malaria in the acute phase. However, infections tend to develop more slowly and become chronic. Malaria caused by *P. malariae* has a 72-h erythrocytic cycle. The term *quartan* refers to the 4 days included within one cycle, with a 3-day interval from the beginning of the first paroxysm to the beginning of the next. Recrudescences of *P. malariae* may occur in individuals up to 50 years after initial infection, owing to low levels of parasitemia that increase under periods of immunosuppression. Its characteristics (including morphological, biological, and molecular-genetic attributes) are identical to *P. brasilianum*, indicating that it is probably the same species and occurs naturally in monkey populations in South America (Lalremruata et al., 2015; Rayner, 2015).

After a person is bitten and inoculated with sporozoites, and exoerythrocytic merogony commences in the liver, symptoms do not appear until days to weeks later (up to a month in *P. malariae*), when erythrocytic merogony begins in the blood. In *P. vivax* and *P. ovale*, if the sporozoites develop into hypnozoites in the liver cells, relapses are possible long after inoculation and initial onset of symptoms, with an intervening period of no apparent symptoms of infection. For *P. falciparum* and *P. malariae*, there are no persistent exoerythrocytic stages of the parasites and relapses do not occur. However, infection with *P. malariae* may recrudesce years after initial infection owing to persistent erythrocytic infections. Therefore, in human malaria there is a clear distinction between relapse and recrudescence of infection. The course of infection of malaria in humans varies with many factors, including history of past exposure and presence of antibodies; age, health, and nutritional status; and genetic resistance factors such as the sickle-cell anemia trait, Duffy-negative blood type, certain hemoglobin types such as hemoglobin S and fetal hemoglobin; and deficiency of the erythrocytic enzyme glucose-6-phospate dehydrogenase.

Mosquito Vectors and Epidemiology

Many different species of *Anopheles* mosquitoes are competent vectors of malaria organisms (Table 15.7). However, most *Anopheles* spp. are not, because of variation in host-selection patterns, longevity, abundance, and vector competence. In North America, *Anopheles quadrimaculatus*, which forms a complex with four more localized but nearly identical species (Reinert et al., 1997), is the principal vector of malaria in the eastern two-thirds of the continent. It develops along the edges of permanent pools, lakes, and swamps that provide relatively clean, still, sunlit water, with lush emergent vegetation, marginal brush, or floating debris that provides partial shade and protection from wave action. In western North America, *An. freeborni* is the main vector, an inhabitant of clear water in open, shallow, sunlit pools, ponds, ditches, and seepage areas that are partially shaded by vegetation. *Anopheles hermsi* also is a vector in California.

Other important vectors include *An. albimanus* in Central America, *An. darlingi* in South America, *An. gambiae* (Fig. 15.34) and *An. funestus* in Africa, *An. culicifacies* in Asia, and *An. dirus* in Southeast Asia. *Anopheles gambiae* is considered the most important of all, because of its involvement in such large numbers of malaria cases and deaths, mainly in Africa. This species lives in close association with humans, on which it primarily feeds, and can complete a gonotrophic cycle in only 2 days. Larvae develop in a wide variety of sunlit surface pools during the rainy seasons, many of which are associated with human activity. These include borrow pits, roadside ditches, wheel ruts, and the hoof prints of domestic animals. Larval development normally takes only about 1 week.

Malaria has been viewed in the context of stable or unstable transmission, reflecting in part the attributes of the *Anopheles* population that affect its vectorial capacity. These include density, longevity, tendency to feed on humans, and duration of the extrinsic incubation period of the parasite in the vector. **Stable malaria** is most often associated with *P. falciparum* infection in highly endemic settings. It is characterized by low fluctuations in parasite incidence in human and vector populations, high prevalence, and high seroprevalence for antibodies. Epidemics are unlikely under these conditions, even though transmission continues at high rates. In such settings, vectors tend to be highly anthropophagic, exhibit greater longevity, and have relatively low, stable densities but still exhibit considerable seasonal variation. **Unstable malaria** tends to be associated with *P. vivax* infections in endemic settings of high fluctuation in disease incidence. Vectors tend to be zoophagic, have seasonally profound variation in population densities, have low or nondetectable field infection rates, and may have shorter longevity than do those in stable malaria settings. Epidemics can occur in conditions of unstable malaria if environmental changes favor increased vector–human contact—such as during civil strife, following water projects such as dams or irrigation schemes, or when a new vector species is introduced into an area.

The epidemiological implications of infection of humans in Southeast Asia with *P. knowlesi* have not yet been fully investigated. Because the same species is maintained in monkeys, this form of malaria may be considered an anthropozoonosis. If so, even complete interruption of transmission between humans will fail to

TABLE 15.7 ***Anopheles*** **Vectors of Human Malaria Parasites in 12 Epidemiologic Zones**

Malaria Epidemiologic Zone	*Anopheles* Vectors
North American	Subgenus *Anopheles*: *freeborni, punctipennis, quadrimaculatus* Subgenus *Nyssorhynchus*: *albimanus*
Central American	Subgenus *Anopheles: aztecus, pseudopunctipennis, punctimacula* Subgenus *Nyssorhynchus: albimanus, albitarsis, allopha, aquasalis, argyritarsis, darlingi*
South American	Subgenus *Anopheles*: *pseudopunctipennis, punctimacula* Subgenus *Nyssorhynchus: albimanus, albitarsis, aquasalis, argyritarsis, braziliensis, darlingi, nuneztovari* Subgenus *Kerteszia*: *bellator, cruzii*
North Eurasian	Subgenus *Anopheles*: *atroparvus, messeae, sacharovi, sinensis* Subgenus *Cellia*: *pattoni*
Mediterranean	Subgenus *Anopheles*: *atroparvus, claviger, labranchiae, messeae, sacharovi* Subgenus *Cellia*: *hispaniola, pattoni*
Africo-Arabian	Subgenus *Cellia*: *hispaniola, multicolor, pharoensis, sergentii*
Africo-Tropical	Subgenus *Cellia: arabiensis, christyi coluzzii, funestus, gambiae, melas, merus, moucheti, nili, pharoensis*
Indo-Iranian	Subgenus *Anopheles: sacharovi* Subgenus *Cellia: annularis, culicifacies, fluviatilis, pulcherrimus, stephensi, superpictus, tesselatus*
Indo-Chinese Hills	Subgenus *Anopheles*: *nigerrimus* Subgenus *Cellia: annularis, culicifacies, dirus, fluviatilis, maculatus, minimus*
Malaysian	Subgenus *Anopheles*: *campestris, donaldi, letifer, nigerrimus, whartoni* Subgenus *Cellia: aconitus, balabacensis, dirus, flavirostris, leucosphyrus, ludlowae, maculatus, minimus, philippinensis, subpictus, sundaicus*
Chinese	Subgenus *Anopheles*: *anthropophagus, sinensis* Subgenus *Cellia: pattoni*
Australasian	Subgenus *Anopheles*: *bancrofti* Subgenus *Cellia*: *annulipes, farauti, karwari, koliensis, punctulatus, subpictus*

Subgenera, species, and geographic distributions are given.
From Macdonald, 1957.

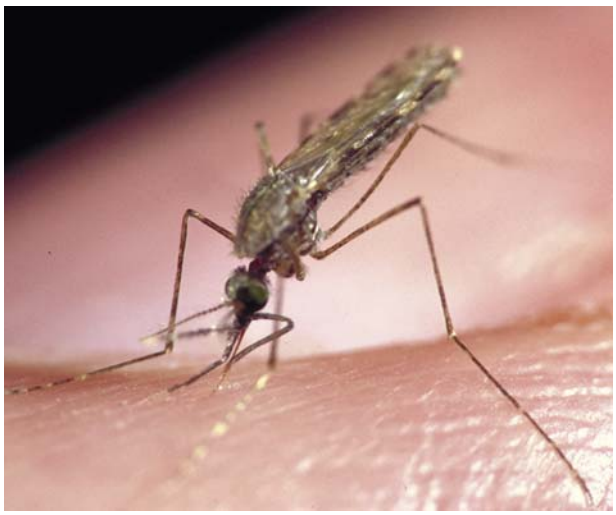


FIGURE 15.34 *Anopheles gambiae* female feeding on blood. This is a major vector of human malaria in Africa, where most cases occur. *Photograph by Woodbridge A. Foster.*

eradicate the parasite, as in the case of YF. The same implications apply to *P. malariae* and *P. simium*, both of which also appear to be anthropozoonoses (i.e., human malarias with monkey reservoirs).

Historical Perspective

After the development of the germ theory of disease by **Louis Pasteur**, the French Algerian physician **Charles Louis Alphonse Laveran** examined and described malarial organisms in the red blood cells of his patients in 1870. This finding, along with the work of **Patrick Manson** (Fig. 1.1A) on filarial nematodes and mosquitoes in China, inspired **Ronald Ross** (Fig. 1.1C), then a physician in British colonial India, to examine the hypothesis of mosquito transmission of malaria parasites in the 1890s. His persistent and careful experimentation and observation with both human and bird malarias, using *Anopheles* and *Culex*

mosquitoes, respectively, provided conclusive proof that mosquitoes transmit *Plasmodium* spp. via bite. In concurrent research, **Giovanni Batista Grassi** and colleagues demonstrated transmission of *Plasmodium falciparum* by *Anopheles maculipennis*—complex mosquitoes in the environs of Rome, Italy. Ross was awarded the Nobel Prize for Medicine in 1902.

Malaria was formerly endemic in many temperate areas of the United States, particularly in the south and southeast. Malaria became epidemic after the Civil War, as malaria-infected soldiers returned to their homes and brought the infection with them to their local communities. Malaria was an important rural disease in the eastern and southern states, California, and other areas of the United States through the 1930s, but gradually disappeared by the 1940s. This was due to a combination of antimosquito measures, improved medical care, a higher standard of living, and transformation of marshes and swamps to agricultural land largely through organized ditching efforts. Changes in life style because of technological advances such as window screens and the invention of the radio, television, and air conditioning also contributed to the decline in malaria. Boyd (1941) reviewed the history of malaria in the United States, and to a brief degree elsewhere in the New World.

Roughly 1000 cases of malaria are introduced into the United States each year. In addition, cases involving local or indigenous transmission occur sporadically, including outbreaks in California (1988, 1989, 1990), Florida (1990, 1996), Michigan (1995), New Jersey (1993), New York (1993), and Texas (1995). These incidents were due to introductions of infected humans into areas with competent *Anopheles* vectors. However, **airport malaria** has occurred near major international airports (e.g., London-Heathrow and Paris-DeGaulle) where infected mosquitoes have been imported on aircraft from endemic regions.

Filariasis

Filariasis is the infection of vertebrate tissues by filarial nematodes or roundworms (Phylum Nematoda, Order Spirurida, Superfamily Filarioidea, Family Onchocercidae). Mosquito-borne filarial nematodes are associated with acute and chronic human disease, termed **lymphatic filariasis**, which is widespread in tropical and subtropical regions (Grove, 1990). The three causative agents of lymphatic filariasis are *Wuchereria bancrofti*, *Brugia malayi*, and *Brugia timori*.

The areas of the world endemic for lymphatic filariasis (Figs. 15.35 and 15.36) include parts of western, central, and southern Africa; parts of northeastern South America (principally Brazil, Surinam, and French Guyana), the Dominican Republic, and Haiti; southern and eastern India, southeastern Asia, eastern China, and southern Japan; the Malay archipelago, Indonesia, the Philippines, Irian Jaya and Papua New Guinea; and many island groups of the south Pacific Ocean, including Melanesia, Micronesia, and Polynesia. Within the United States, filariasis was locally endemic in Charleston, South Carolina, but the disease disappeared there in the late 1930s. It disappeared at about

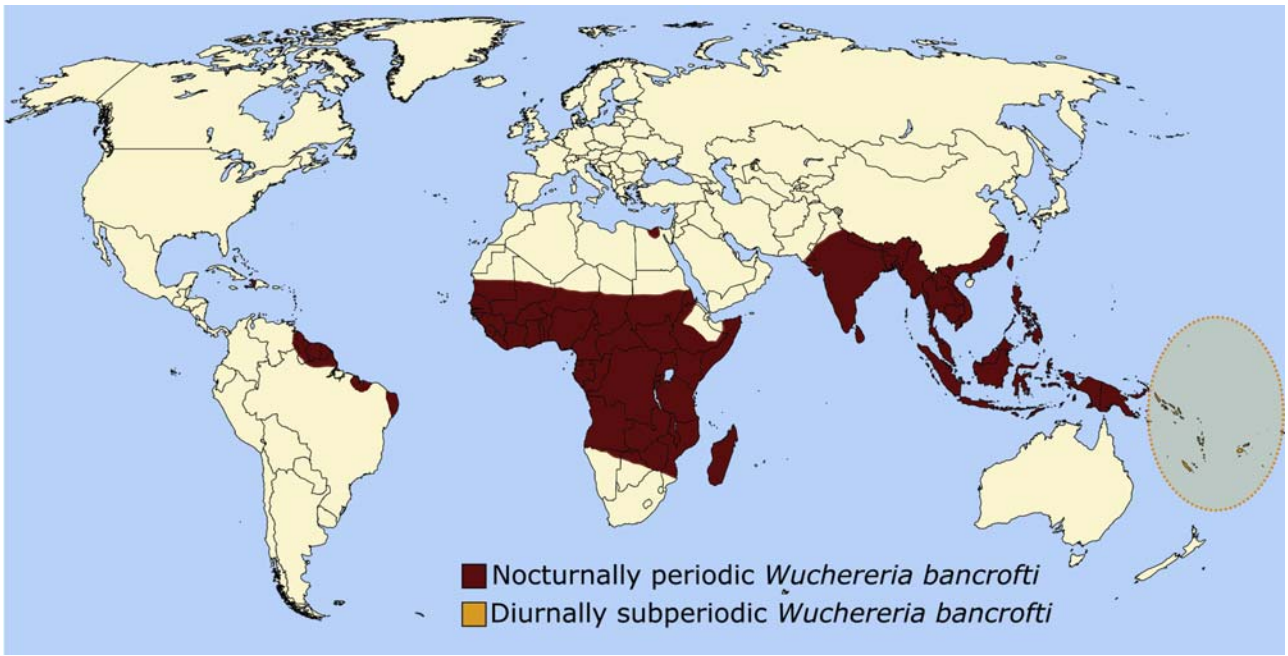


FIGURE 15.35 Geographic distribution of human lymphatic filariasis caused by *Wuchereria bancrofti*, showing both nocturnal and diurnal periodicity. *Based on data from the Centers for Disease Control and Prevention, USA, and the World Health Organization.*

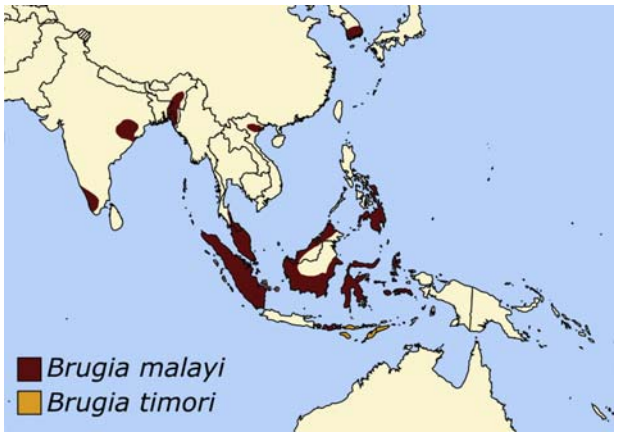


FIGURE 15.36 Geographic distribution of human lymphatic filariasis caused by *Brugia malayi* and *B. timori*. *Reconstructed from Strickland, 1991, and other sources.*

the same time from northern Australia. Lymphatic filariasis no longer occurs in most regions of the Mediterranean basin and on the Arabian Peninsula. Transmission continues, however, along the Nile River and in the Nile Delta of Egypt. Cano et al. (2014) presents a global perspective on its distribution and geographic limits.

Within the area of current distribution, there are an estimated 905 million people at risk of contracting lymphatic filariasis, and some 128 million active infections. Of these, about 115 million are caused by *W. bancrofti*, the causative agent of **Bancroftian filariasis**, which is widespread in both the Old World and New World tropics (Fig. 15.35). Another 13 million cases are caused by *B. malayi*, the causative agent of **Brugian filariasis** or **Malayan filariasis**, which is restricted to southeastern Asia (Fig. 15.36). About 43 million people have chronic symptoms of elephantiasis, hydrocele, or lymphedema (see later). Another Brugian filariasis, **Timorian filariasis**, is caused by infection with *B. timori* and occurs in localized foci among southern islands of Indonesia.

Filarial Life Cycle

Infection with filarial nematodes in humans begins when infective third-stage larvae enter the skin at the site of the mosquito bite, molt twice, and migrate to the lymphatic vessels and lymph nodes, particularly of the lower abdomen. There, the nematodes develop to the adult stage. Female worms (80−100 mm long at maturity for *W. bancrofti* and about half of that length for *B. malayi*) release active, immature worms called **microfilariae** into the peripheral circulatory system. A female can release 50,000 or more microfilariae each day. Microfilariae of *W. bancrofti* are 250−300 µm long and about 7−9 µm wide; those of *B. malayi* are somewhat shorter and thinner. Presence of microfilariae in the blood is called **microfilaremia** and first appears about 6 months to 1 year after adult worms become established in the lymphatic system. An infected human may be microfilaremic for longer than 10 years. The density of microfilariae in peripheral blood is highly variable, but it can range from one to more than 500 microfilariae per 20 mm^3.

The appearance of microfilariae in the peripheral blood has a 24-h cycle (i.e., they exhibit **diel periodicity**). If microfilariae completely disappear from the peripheral circulation at some time during the day, they are said to be **periodic**. If the microfilariae fluctuate in density during a 24-h period but are detectable at all times, they are said to be **subperiodic**. In most areas, the microfilariae appear only at night and are transmitted by mosquitoes that have night-biting habits. These are the **nocturnally periodic** forms of *W. bancrofti* and *B. malayi*. Both *W. bancrofti* and *B. malayi* also have nocturnally subperiodic and diurnally subperiodic forms, though nocturnally subperiodic Bancroftian and diurnally subperiodic Brugian forms have very restricted distributions. In all the subperiodic forms, the microfilariae appear in the peripheral blood of the human host mainly in the evening and night (nocturnally subperiodic) or mainly during the daytime (diurnally subperiodic). These nematodes are associated with two or more species of mosquito vectors, which differ in their typical biting times and whose combined diel patterns of human-biting density are matched by the periodicity of microfilariae. Both nocturnally periodic *W. bancrofti* and nocturnally periodic *B. malayi* are now considered to be strictly human pathogens. However, *B. malayi* in its subperiodic form is a zoonosis, with both leaf monkeys and humans as reservoirs, and with domestic cats and other carnivores also implicated as hosts. *Brugia timori* is nocturnally periodic and has no animal reservoir.

The development of *Wuchereria bancrofti* and *Brugia malayi* in mosquitoes is similar. Microfilariae ingested with the mosquito bloodmeal usually shed their outer, sheathlike membrane as they penetrate through the midgut epithelium. Some microfilariae retain the sheath during penetration. The microfilariae move to the indirect flight muscles of the thorax, penetrate individual cells, and transform to a short **sausage stage**, the L1 or first-stage larva. They molt to more slender L2 or second-stage larvae and then again to the elongate, filariform L3 or third-stage infective larvae (about 1.5 mm long). These larvae leave the thoracic flight muscles, traverse the hemocoel of the mosquito, enter the lumen of the mouthparts, and eventually arrive at the apex of the proboscis. When the mosquito blood-feeds, the L3 larvae exit through the cuticle of the labium, crawl onto the skin, and enter through the hole made by the mosquito during feeding. Therefore, technically the transmission method may be said to be contaminative, rather than inoculative. Heavy infections of larval nematodes can be fatal to the mosquito.

Many species of mosquitoes are refractory to filarial development, owing to genetic factors and to their ability to mount adequate immune responses, whereas other species are susceptible to parasite infection and support the development described earlier. The relationships between *W. bancrofti* and certain mosquito species exhibit evidence of local adaptation. For example, in West Africa, *Culex quinquefasciatus* is not a competent vector of *W. bancrofti*, whereas *Anopheles gambiae* is competent there. By contrast, in India, *Cx. quinquefasciatus* is a competent vector and most *Anopheles* species are not. Thus, there is geographic variation in susceptibility of mosquitoes to filarial nematodes.

Clinical Disease

Generally, a case of human infection does not occur after the bite of a single, infective mosquito. Rather it results after accumulation of hundreds to thousands of such infective bites, under conditions in which there is a high probability of parasite maturation and mating. Humans may show disease symptoms without having microfilaremia, or may have microfilaremia without showing signs of disease. Lymphatic filariasis has both acute and chronic manifestations in the human host. The disease may cause chronic debilitation in untreated cases. **Acute lymphatic filariasis** is characterized by episodes of fever, swelling, pain, and inflammation in the affected lymph nodes and lymph vessels, a condition called adenolymphangitis. The episodes may last for several days and incapacitate the affected individual because of the local and systemic effects. Over time, deep abscesses may develop at the sites of inflammation. Dermal ulcers may form through the skin over these sites, and secondary bacterial infection may ensue.

In the **chronic phase**, which often occurs years after onset of acute symptoms, the pathology may involve accumulation of lymphatic fluid (lymphedema) in the limbs, breasts, vulva, and scrotum, resulting in swelling and enlargement. In the scrotum, this condition is termed hydrocele. The grotesque distentions and thickening, folding, and nodulation of the skin, notably, the lower limbs, is a condition called **elephantiasis** (Figs. 15.37 and 15.38). Bacterial and fungal infections at affected sites exacerbate these conditions. Appearance of lymph fluid in urine may occur as a consequence of the disruption of the abdominal lymphatic vessels and leakage of lymph fluid into the urinary tract, causing urine to appear whitish, a condition called **chyluria**. In contrast with *W. bancrofti*, infection

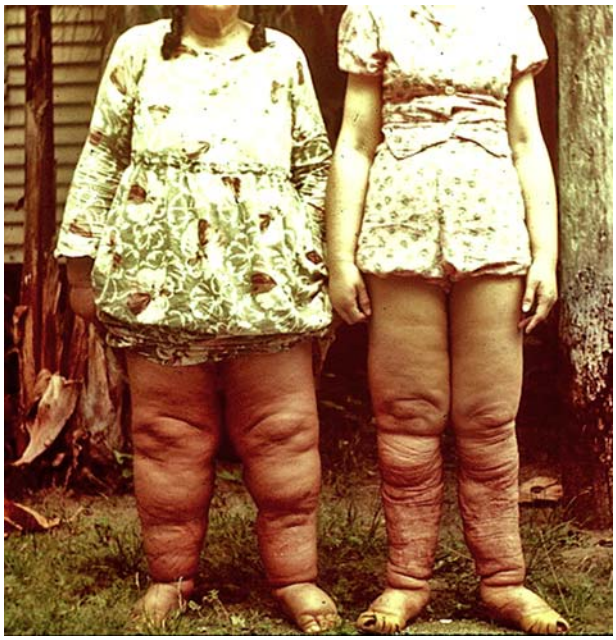


FIGURE 15.37 Human lymphatic filariasis in two Tahitian women, 60-year-old (left) and 30-year-old (right), in the early 1940s; both individuals after enlargement of legs reduced 30%–50% of their former size by tight bandaging for 6 months, enabling them to walk again. *Photograph by S. Allen Edgar.*

FIGURE 15.38 Human lymphatic filariasis in Tahitian man, with extreme case of elephantiasis involving notably, the left arm, left leg, and scrotum; photograph taken during World War II. *Photograph by S. Allen Edgar.*

with *B. malayi* is not associated with scrotal distension, but rather involves only the limbs. Hypersensitivity to parasite-associated antigens also may be part of the syndrome of lymphatic filariasis. It is mediated through elevated IgE and IgG4 antibodies and is characterized by increased production of eosinophils, coughing, and shortness of breath. This type of filarial disease is called **tropical eosinophilia**.

Lymphatic filariasis has important social implications in communities where it is endemic. Acutely affected individuals are often feverish and in pain, and they may have difficulty working and thus suffer economic loss. Hard work may bring on attacks of filarial fever, which require rest for recovery. In a study conducted in Ghana, West Africa, the episodes of acute adenolymphangitis lasted about 5 days, with 3 days of incapacitation; they occurred during those months of the year when peak agricultural work was required and when mosquito transmission of infective-stage larvae was highest (Gyapong et al., 1996). Likely, chronically infected individuals are immunologically sensitized to their worm infections, and exposure to new L3 larvae results in hypersensitive reactions such as adenitis. Chronically affected individuals with symptoms of scrotal hydrocele and elephantiasis may have difficulty in their social and personal lives and may suffer incontinence and impotence. Although hydrocele can be treated through fluid aspiration from the scrotum, the gross distention associated with elephantiasis is difficult to remedy even with surgery.

Mosquito Vectors and Epidemiology

The filarial nematodes that cause lymphatic filariasis have evolved associations with mosquitoes in the genera *Culex*, *Mansonia*, *Aedes*, and *Anopheles* (Table 15.8). This probably has occurred through a process of adaptive radiation from the original *Anopheles* vectors in Southeast Asia. A very likely scenario for *W. bancrofti* is that it arose as a human pathogen in forested regions of Indonesia and perhaps other parts of Southeast Asia, where the *Anopheles umbrosus* group serves as vectors of *Wuchereria kalimantani*. This filarioid nematode is a parasite of the silvered leaf monkey (brow-ridged langur), *Presbytis cristata*. Similarly, *B. malayi* infection in humans probably evolved from subperiodic *B. malayi* infections among leaf monkey species (*Presbytis* spp.), with *An. hyrcanus* as the vector. Human infection and new disease foci probably arose in forests and along forest ecotones with these same *Anopheles* vectors. Through time, as people developed agricultural systems and moved to other regions, both *W. bancrofti* and *B. malayi* adapted to these new settings and the competent mosquito vectors there. In some parts of Southeast Asia, members of the *Aedes niveus* group are important vectors of the subperiodic form of *W. bancrofti*, including *Ae. niveus* in Thailand; this system may have been the origin of the subperiodic strains that radiated into the Pacific regions.

Transmission of *W. bancrofti* occurs in both urban and rural areas. The primary mosquito vector in urban areas is *Culex quinquefasciatus*. It is the most important vector of nocturnally periodic *W. bancrofti* in the Americas and parts of Africa and Asia, particularly India. It feeds opportunistically at night on both mammals and birds. This mosquito occurs abundantly in areas with poor sanitation, open sewers, untreated waste water, and pit latrines, which provide the high organic content and low oxygen characteristic of the larval habitat. For this reason, *Cx. quinquefasciatus* is often more abundant during parts of the year when water stagnates from lack of rain. In the Nile River Delta of Egypt, *Culex molestus*, a name applied to the autogenous variant of *Cx. pipiens,* is the primary vector. In rural settings, nocturnally periodic Bancroftian and Brugian filariases are transmitted by *Anopheles* mosquitoes, which are also nocturnally active. Often, the same *Anopheles* species that transmit *W. bancrofti* or *B. malayi* in an area also are responsible for local malaria transmission (e.g., *An. darlingi* in South America and *An. gambiae* and *An. funestus* in parts of West and East Africa, respectively, for Bancroftian filariasis; and *An. sinensis* in rice-growing areas of China, for Brugian filariasis).

Nocturnally periodic Brugian filariasis occurs in rural parts of southern India, Malaysia, the Philippines, and Indonesia; there the nocturnally active *Mansonia annulifera* and *Ma. uniformis*, and also *Anopheles* species, are vectors near rice fields and open swamps. The nocturnally subperiodic form occurs in swamp forest areas of Southeast Asia and Indonesia, involving *Ma. bonneae* and *Ma. dives,* which are nocturnally active but also feed during the day within the swamp forests. All of these *Mansonia* species are associated with particular kinds of plants, where the larvae and pupae attach to their submerged roots and stems. In the Pacific region, where many island groups are endemic for *W. bancrofti*, the primary vectors are day-biting *Aedes* spp., but nocturnal biters also are involved, and the form of the parasite is diurnally subperiodic. *Anopheles barbirostris* is the vector of *Brugia timori*.

The endemicity of mosquito-borne filariasis depends on a high and steady rate of transmission of infective-stage larvae in the human population. In endemic areas, the inoculation rate (parasite transfer rate) can range as high as hundreds of infective bites and thousands of larval inoculations per person per year. The estimated number of L3 larvae transmitted per person per year is called the annual transmission potential. With each infective bite, only a few L3 larvae actually enter the skin. These larvae must then develop further and move to a person's lymphatic system, where mature male and female nematodes mate and initiate microfilarial production. Accumulation of thousands of infective bites over months or years eventually results in an

TABLE 15.8 Mosquito Vectors of Filarioid Nematodes of Humans: Geographic Distribution, and Associations With Periodicity of Microfilaremia

Geographic Region	Filarioid Species	Periodicity	Mosquito Vectors
Neotropical	*Wuchereria bancrofti*	Nocturnally periodic	*Anopheles aquasalis, An. bellator, An. darlingi, Aedes scapularis, Culex quinquefasciatus, Mansonia titillans.*
Afrotropical	*Wuchereria bancrofti*	Nocturnally periodic	*Anopheles funestus, An. gambiae, An. arabiensis, An. bwambae, An. melas, An. merus, An. nili, An. pauliani, Culex quinquefasciatus.*
Middle Eastern	*Wuchereria bancrofti*	Nocturnally periodic	*Culex molestus.*
Oriental	*Wuchereria bancrofti*	Nocturnally periodic	*Anopheles anthropophagus, An. kweiyangensis, An. nigerrimus, An. letifer, An. whartoni, An. aconitus, An. flavirostris, An. minimus, An. candidiensis, An. balabacensis, An. leucosphyrus, An. maculatus, An. philippinensis, An. subpictus, An. vagus, Aedes niveus, Ae. togoi, Ae. poicilius, Culex bitaeniorhynchus, Cx. sitiens complex, Cx. pallens, Cx. quinquefasciatus, Mansonia uniformis.*
	Wuchereria bancrofti	Nocturnally subperiodic	*Anopheles sinensis complex, Aedes harinasutai.*
	Brugia malayi	Nocturnally periodic	*Anopheles barbirostris, An. campestris, An. donaldi, An. anthropophagus, An. kweiyangensis, An. nigerrimus, Aedes togoi, Mansonia uniformis, Ma. bonneae, Ma. dives.*
	Brugia malayi	Nocturnally Subperiodic	*Anopheles sinensis complex, Mansonia uniformis, Ma. bonneae, Ma. annulata, Ma. indiana, Ma. dives.*
	Brugia timori	Nocturnally periodic	*Anopheles barbirostris.*
West Pacific	*Wuchereria bancrofti*	Nocturnally periodic	*Culex pallens, Aedes poicilius, Ae. togoi.*
	Brugia malayi	Nocturnally subperiodic	*Aedes togoi.*
Papuan	*Wuchereria bancrofti*	Nocturnally periodic	*Anopheles bancrofti, An. punctulatus, An. farauti, An. koliensis, Culex annulirostris Cx. bitaeniorhynchus, Mansonia uniformis*
South Pacific	*Wuchereria bancrofti*	Nocturnally subperiodic	*Aedes samoanus.*
		Diurnally subperiodic	*Aedes fijiensis, Ae. oceanicus, Ae. vigilax* group, *Ae. futunae, Ae. polynesiensis, Ae. pseudoscutellaris, Ae. tabu, Ae. tongae, Ae. upolensis.*
	Brugia malayi	Nocturnally periodic	*Aedes oceanicus.*
		Nocturnally subperiodic	*Aedes oceanicus.*

infection of mature worms in a human, who normally then will have a microfilaremia and possibly, chronic disease. Most microfilariae entering the circulatory system are never ingested by a mosquito, while those that are ingested become infective-stage larvae only in a competent vector, and only if the individual mosquitoes survive the extrinsic incubation period. Furthermore, many infective-stage larvae fail to reach a new human host or fail to mature if they do. The inefficiency of transmission and parasite perpetuation is compensated by the prodigious production of microfilariae and the long life of adult worms.

Historical Perspective

Association of infection with filarial nematodes and lymphatic filariasis was first established in the late 1800s. Our understanding of the natural history of lymphatic filariasis is related intimately to the initial discovery of a

link between human pathogens and insect vectors. During 1877 to 1878, **Patrick Manson** (Fig. 1.1A), working in China as a medical officer for the Chinese Imperial Customs Service, conducted experiments on the development of filarial nematodes. Manson had already discovered that the microfilariae occurred in the peripheral blood only at night. He speculated that this was timed to coincide with the night-time biting activity of mosquitoes. After feeding mosquitoes (*Culex quinquefasciatus*) on his gardener, who had a microfilaremia, and then dissecting the mosquitoes on successive days, he found that the worms developed within the mosquitoes into longer, different forms. He speculated that the mosquito functioned as a kind of "nurse" for the filarial worms, so that when a mosquito died on the water after laying an egg raft, the worms entered the water and later infected a person drinking it. At that time, it was not known that mosquitoes could bite more than once during their lives, so the principle of transmission by bite was not established. Yet, the idea that mosquitoes can function as intermediate hosts for a human pathogen is founded on Manson's experiments.

VETERINARY IMPORTANCE

Aside from their importance as vectors of disease agents of animals, mosquitoes are a cause of irritation, blood loss, and allergic reactions. They not are only annoying but also disrupt normal behavior of livestock and companion animals. Large swarms may cause livestock to discontinue feeding and to seek relief. Increased scratching behavior may result in skin abrasions, hair loss, and secondary infection with bacteria at the bite and scratch sites. For cattle, mosquito bites can result in decreased weight gains and milk production and prompt producers to alter pasturing practices. Deaths of cattle due to anemia and stress have been reported.

Mosquito-Borne Viruses of Animals

Mosquito-borne viruses affecting domesticated animals include the groups of alphaviruses that are associated with the equine encephalitides (EEE, WEE, and VEE), all of which cause an acute encephalitis with high fever in equids (i.e., horses, donkeys, mules). The history, distribution, vector relationships, and vertebrate reservoir hosts of these viruses were discussed earlier in the section "Public Health Importance." Other mosquito-borne viruses of veterinary significance include JE virus, Rift Valley fever virus, Wesselsbron virus, fowlpox virus, and myxomatosis virus. Equine infectious anemia (EIA) virus, a lentivirus in the family Retroviridae, may be mechanically transmitted by mosquitoes, but its more important mechanical vectors are larger biting flies (deer flies, horse flies, and stable flies).

FIGURE 15.39 Horse dying from infection with Eastern equine encephalomyelitis virus in Michigan outbreak in 1980. *Photograph by Harold D. Newson.*

Eastern Equine Encephalomyelitis (EEE) virus. This virus is an important cause of mortality of horses and other equids, caged pheasants, whooping cranes, and emus. It occurs in endemic areas of the United States in Texas, along the Gulf Coast and Atlantic Seaboard to Massachusetts, and at inland sites in upstate New York, Ohio, Michigan, Indiana, Georgia, and Alabama. Horses rapidly succumb to infection after a short incubation period of 2–5 days. They exhibit abnormal behavior and high fever, then drop to the ground and lapse into coma before death (Fig. 15.39). Few horses survive infection involving these acute symptoms. Viral infection in the brain shows characteristic lesions in nervous tissue, accompanied by perivascular cuffing with macrophages; viral antigen is detectable in neurons (Fig. 15.40). In pheasant flocks, often a single infected, sick bird will be pecked by other birds, thus transmitting the virus directly to healthy birds without mosquito bite. During such occurrences, called **epiornitics**, thousands of pheasants in a single outdoor pen may die, yet

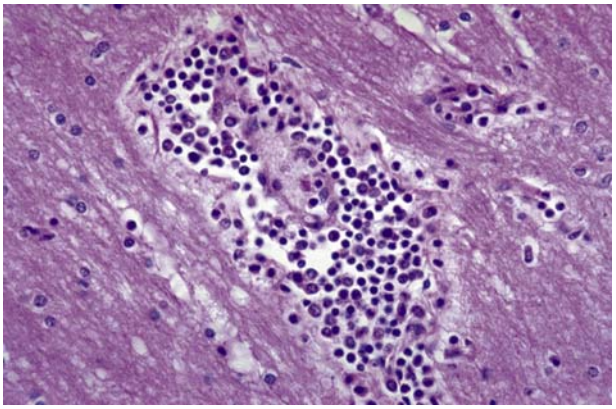


FIGURE 15.40 Section of horse brain (cerebrum) infected with Eastern equine encephalomyelitis virus, showing neutrophil invasion around capillary. *Photograph by Jonathan D. Patterson.*

none of the pheasants in adjacent pens become infected. Aside from equids and exotic birds, EEE viral infection has been reported in young dogs and pigs.

Cases of EEE in horses in cool temperate climates tend to occur in mid to late summer and early fall, whereas in milder climates horse cases begin to occur earlier in the spring and summer. In the tropics and subtropics, cases may occur year-round. Horse deaths due to EEE viral infection are an important indicator of virus activity promoted by bridge vectors in endemic areas. Rapid, differential diagnosis of horse cases is crucial if these animals are to be used as sentinels for potential transmission of EEE virus to humans.

Western Equine Encephalomyelitis (EEE) Virus. As with EEE virus, the primary epidemic host of significance for WEE virus is the horse. Human cases are rare. Since the first isolation of WEE virus from the brain of a dead horse in 1930 in the San Joaquin Valley of California (USA), this virus has been implicated in epizootics in horses, with cases numbering from hundreds to thousands in some instances. Large outbreaks occurred in 1941 and 1975 in the Red River Valley in Minnesota, North Dakota, and Manitoba (USA and Canada), in 1952 in the Central Valley of California, and in 1965 in Hale County in western Texas (USA). There are horse cases almost every summer within the range of the virus, but epizootics do not always occur. For both EEE and WEE viruses, immunization has reduced the frequency of horse cases.

Venezuelan Equine Encephalomyelitis (VEE) Complex. Encephalomyelitis in equids caused by viruses of the VEE complex occurs in northern South America, Central America, and Mexico. The epizootic viruses are transmitted by many species of mosquitoes (see Table 15.4) among horses, burros, and mules. These animals develop a viremia sufficient to infect the mosquitoes. Consequently, VEE epidemics can be maintained by transmission between mosquito and horses, differing in this regard from WEE and EEE, for which horses are largely dead-end hosts. An epidemic strain of VEE virus was first isolated in 1938 from a horse in Venezuela. In 1969, a large outbreak of VEE involving both equids and humans in Central America spread northward in the succeeding 2 years through Mexico, and in 1971 across the border into Texas (USA) (PAHO, 1972). Cases continued in Mexico through 1972. There were thousands of horse cases throughout this region during that time. Epizootic virus activity did not occur again in the region until an outbreak in Venezuela in 1992 and 1993 and in Chiapas, Mexico, in 1993. Another outbreak of VEE occurred in northern Colombia and Venezuela in 1995. The rapidity of spread of these outbreaks over large geographic areas is undoubtedly due to the role of both horses and birds as competent reservoir hosts. It is expedited by the evacuation of horses, already infected but not yet ill, away from an epizootic area.

Japanese Encephalitis (JE) Virus. Encephalitis caused by JE virus occurs in widespread parts of Asia, including Malaysia and Indonesia. In Japan, epizootics and epidemics have occurred in August and September in many years since the discovery of this disease in 1935 in that country. The virus was isolated from brain tissue of a horse in 1937. Japanese encephalitis virus causes acute infection in horses and swine. It is particularly an economic problem because of the importance of swine as a food source and market commodity in rural Asia. Pigs develop viremia sufficient for mosquito transmission, therefore serving as important amplifying hosts, and may develop encephalitic symptoms. Transplacental infection causes stillbirth and abortion. Infected boars may become sterile.

Rift Valley Fever (RVF) virus. This pathogen has caused epizootics of acute illness, elevated rates of abortion, and death in cattle, goats, and sheep in Egypt and parts of Africa. Outbreaks in Egypt and Mauritania were particularly noteworthy. The virus is both viscerotropic and neurotropic in these animals. Their viremias are of sufficient titer to infect mosquitoes. Disease outbreaks generally have involved thousands to hundreds of thousands of livestock cases, causing substantial economic losses, but these losses are rarely counted accurately.

Wesselsbron (WSL) Virus. This is a flavivirus with distribution in parts of Africa, Madagascar, and Thailand. It causes a disease similar to that of RVF in sheep and goats and also causes a mild illness in cattle. Infected ewes may abort their fetuses, and lambs suffer high mortality. Humans infected with Wesselsbron virus may develop a febrile illness with rash, fever, and myalgia. The virus is transmitted by *Aedes* spp., including *Ae. mcintoshi* and *Ae. circumluteolus* in South Africa.

Fowlpox (FP) virus. This virus belongs to a group of poxviruses that infect vertebrates and invertebrates and are classified within the family Poxviridae. Among the poxviruses are those in the bird-infecting genus *Avipoxvirus*, such as fowlpox, canarypox, and pigeonpox viruses. Mosquitoes may mechanically transmit the avipox viruses by contamination of mouthparts and subsequent transfer of infectious virions to noninfected birds. Fowlpox is an important disease of domestic fowl, particularly chickens. It causes development of papules along the comb and beak. While probing these papules, mosquitoes may contaminate their mouthparts with virions. If disturbed during feeding, they may move to another animal to feed, thus transferring the virus to a new host. Another form of fowlpox virus is transmitted directly by droplets of pus-containing the virus.

Myxoma (MYX) virus. This is a leporivirus and the causative agent of **myxomatosis**, an enzootic disease of lagomorphs in parts of South America and the western United States. It is transmitted mechanically by bite of arthropods, principally by mosquitoes and fleas, depending on the region. These viruses produce dermal, vascularized tumors. When vectors probe these tumors, the mouthparts become contaminated with virus particles. Later, if the mosquitoes probe another uninfected lagomorph, that animal may become infected. Natural infections of myxoma virus occur without acute disease in rabbits of the genus *Sylvilagus* in South America and California (USA). However, Old World rabbits (*Oryctolagus cuniculus*) are highly susceptible to infection and generally die. Outbreaks of acute disease among domesticated Old World rabbits have been documented in South America and California.

Myxoma virus was introduced into Australia in the 1950s as a means of controlling introduced European rabbits, a pest in that country. The virus spread rapidly through the rabbit populations via mechanical transmission by mosquitoes, fleas, and other means, and greatly reduced rabbit populations there. The mosquito vectors in Australia are *Culex annulirostris*, *Anopheles annulipes*, and *Aedes* spp.

Nonhuman Malarias

Many *Plasmodium* spp. infect animals other than humans, including reptiles, birds, rodents, and nonhuman primates.

Reptilian Malarias

The malarias of reptiles, formerly called saurian malarias, are caused by a group of 29 *Plasmodium* species. They infect a wide range of lizards and some snakes in 15 families (Telford, 1994). Vectors are biting midges, phlebotomine sand flies, and *Culex* mosquitoes. Haemoproteid and leucocytozooid malarias also occur in reptiles, but their vectors have not been established.

Avian Malarias

Malarial infection of birds is widespread geographically (Van Riper et al., 1994). Parasites in three common genera of hemosporine blood parasites of birds (*Hepatocystis*, *Haemoproteus*, and *Leucocytozoon*) are transmitted by biting midge, louse fly, and black fly vectors, respectively. The avian malarias in the genus *Plasmodium* are all mosquito-borne. *Plasmodium* spp. that infect birds have been important research models for studying malaria. Indeed, the original observations by Ronald Ross on the role of mosquitoes as malaria vectors were made with bird malaria.

Currently, about 30 species of avian *Plasmodium* are recognized. However, the taxonomic status of some species is uncertain and others remain to be described. Among the important species that cause disease in domestic fowl or wild birds are *P. gallinaceum* (sometimes called **chicken malaria**), *P. hermansi* (a parasite of wild and domestic turkeys in the United States), *P. relictum*, *P. lophurae*, *P. cathemerium*, *P. circumflexum*, and *P. elongatum*. As with human malarias, there is variation in life cycles and pathogenesis of the avian malarias. This variation is related to intrinsic qualities of the species and to variation in susceptibility among host species, age, and general health status.

A bird becomes infected after inoculation of sporozoites from an infective mosquito. Merogony occurs in bone marrow, endothelial cells, and in the erythrocytes. In acute infections, these parasites may cause severe anemia, damage to bone marrow tissues, and other pathology that may result in death. Younger birds tend to be more susceptible to overt illness than older birds.

Although *Anopheles* mosquitoes can be competent laboratory vectors for some bird malarias, field and laboratory data show that culicines in the genera *Culex*, *Culiseta*, and *Aedes* are the natural vectors. In Africa, *Aedes aegypti* is an important local vector of *P. gallinaceum* to chickens. The impact of bird malaria on natural bird populations is poorly known. It was introduced into Hawaii (USA) along with exotic birds and *Culex* mosquitoes and is thought to be responsible for the reduction and extinction of native bird populations there. Bird malaria occasionally has been documented as the cause of morbidity and mortality among penguins in zoos.

Rodent Malarias

The 12 *Plasmodium* spp./ infecting rodents, called rodent or **murine malarias**, all occur in Africa and Asia. The vectors are assumed to be *Anopheles* mosquitoes, but in most cases the vector species is unknown. *Plasmodium berghei*, *P. vinckei*, *P. yoelli*, *P. chabaudi,* and *P. aegyptensis* parasitize African murine rodents. The first two are transmitted by *Anopheles dureni* in Zaire, and *P. vinckei* is transmitted by *An. cinctus* in Nigeria. *Plasmodium atheruri* infects the African brush-tailed porcupine (*Atherurus africanus*) and is transmitted by *An. smithii. Plasmodium anomaluri*, *P. landauae*, and *P. pulmophilum* occur in African flying squirrels (*Anomalurus* spp.); *An. marchadyi* is the probable vector of the *P. atheruri*. The three species of *Plasmodium* found in Asian flying squirrels are *P. booliati*, *P. watteni*, and *P. incertae*. The significance of rodent malarias to the health and population dynamics of their natural hosts is largely unknown, although the prevalences of infection can be high. They have become important laboratory models for human malaria, particularly in host immunological responses, drug screening studies, and vaccine development. Cox (1993) provides a succinct review of the rodent malarias.

Primate Malarias

The nonhuman primate malarias are caused by a group of 25 *Plasmodium* spp., many of which are closely related to the human malarias (Collins and Aikawa, 1993). Seven of them infect lemurs in Madagascar and are poorly known. All 18 others have life cycles similar to those of the human malarias. Most have a tertian periodicity, but two species (*P. brasilianum* and *P. inui*) are quartan and one (*P. knowlesi*) is quotidian (i.e., has a periodicity of 1 day). *Plasmodium knowlesi* appears to have become a frequent parasite of humans, as well. Probably all are transmitted by *Anopheles* mosquitoes, but for 10 of them the vector species are unknown.

Of the 18 well-known primate malaria species, 13 occur in southern or southeastern Asia, where macaques, langurs (leaf monkeys), gibbons, and orangutans are the vertebrate hosts. These plasmodia include *P. pitheci* in the orangutan, the first nonhuman primate malaria to be described; *P. knowlesi*, a macaque parasite that has become an important laboratory model for development of human vaccines; and *P. cynomolgi*, a parasite of macaques and langurs that serves as an important model for human *P. vivax* malaria. The vectors of these Asian primate malarias include *Anopheles hackeri*, *An. dirus*, *An. balabacensis*, *An. elegans*, and *An. introlatus*.

Three primate malarias occur in Africa, where *Plasmodium gonderi* infects mangabeys and mandrills, and *P. reichenowi* and *P. schwetzi* infect chimpanzees and gorillas. Their natural vectors are unknown. Two *Plasmodium* species infect nonhuman primates in South America. *Plasmodium simium* infects howler monkeys and woolly spider monkeys in Brazil. It is similar to the human parasite *P. vivax* and infects humans under natural conditions. *Plasmodium brasilianum* infects a wide range of New World monkeys in the family Cebidae, including howler monkeys, spider monkeys, woolly spider monkeys, titis, capuchins, woolly monkeys, bearded sakis, and squirrel monkeys. It appears to be identical to the human parasite *P. malariae*, suggesting that the latter species also should be considered an anthropozoonosis and the name *P. brasilianum* be retired (Lalremruata et al., 2015; Rayner, 2015). Both South American species are transmitted by *Anopheles cruzii*, which also is an important vector of human malaria in parts of South America. The larvae inhabit water-filled leaf axils of bromeliad plants at heights of 5 m or more, where the adults are likely to encounter arboreal primates.

Many species of *Anopheles* are competent laboratory vectors of primate malarias, including *An. stephensi*, *An. maculatus*, *An. gambiae*, and *An. dirus*, which serve as vectors of human malaria. *Plasmodium knowlesi* infects humans experimentally and naturally, and can be transmitted by the bite of *An. dirus* to other humans. *Plasmodium cynomolgi* has infected laboratory workers, and experimental studies showed that mosquito transmission from monkeys to humans, and from humans to humans, can occur. *Plasmodium brasilianum* also infects humans and may be synonymous with *P. malariae*. Human malaria due to infection with *P. brasilianum* and *P. simium* possibly occurred as a zoonosis in the New World prior to the arrival of Europeans, with *An. cruzii* acting as the vector. Alternatively, these two simian parasites might be derived from human *Plasmodium* species to which they are closely related. Coatney et al. (1971) reviewed the infectivity of nonhuman primate malarias to humans, and Collins and Aikawa (1993) reviewed the primate malarias.

Dog Heartworm

Dog heartworm is caused by the mosquito-borne filarial nematode *Dirofilaria immitis*, a member of the family Onchocercidae (Boreham and Atwell, 1988). Adult *D. immitis* occupy the right ventricle of the canine heart and the pulmonary arteries (Fig. 15.41). The worms are 12−31 cm long and form aggregations of up to 50 or more individuals. In large aggregations, infection may extend to the right atrium. Contrary to popular belief, heartworm disease in dogs is not simply a consequence of a heavy worm burden in the ventricle resulting in impedance of blood flow. Rather, it is the result of deleterious changes in the endothelium and integrity of the walls of the pulmonary arteries, leading to pulmonary hypertension and right ventricular hypertrophy. These pathologic changes cause decreased cardiac output to the lungs, weakness, lethargy, chronic coughing, and ultimately congestive heart failure. Dogs may die if left untreated.

The **life cycle** of *Dirofilaria immitis* involves canids and mosquitoes. Dogs become infected by the bite of a mosquito whose labium carries third-stage larvae. These larvae break out of the labium while it is bent during feeding and are

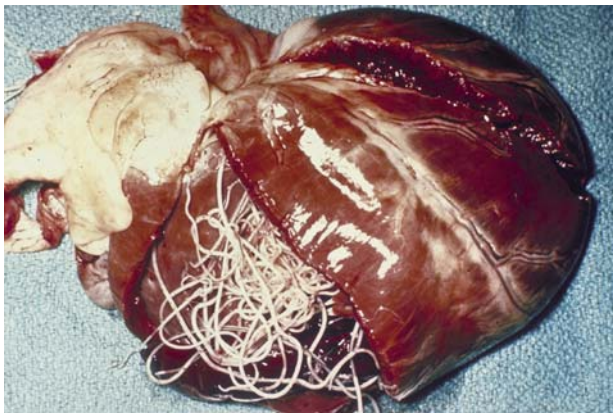


FIGURE 15.41 *Dirofilaria immitis* adults in right ventricle of dog heart. *Photograph from Harold D. Newson.*

deposited onto the dog's skin, along with a small droplet of mosquito hemolymph from the ruptured labium. Only about 10% of the larvae successfully enter the skin, generally through the hole made by the mosquito's fascicle. They remain in situ subcutaneously, where they molt to fourth-stage larvae. The larvae then migrate to other subcutaneous, adipose, or muscle tissues and molt again to a fifth-stage larva. These worms, now approximately 18 mm long, enter the venous circulation and become established in the heart and pulmonary arteries. Generally, the fifth-stage larvae reach the heart at about 70–90 days after infection.

In the heart and pulmonary arteries, the fifth-stage larvae develop into sexually mature adults. After mating, at 6–7 months, the females begin to release into circulation the microfilariae, active embyronic life stages about 300 μm long and 7 μm wide. The microfilaremia varies considerably, from 1,000 to 100,000 microfilariae per milliliter. It is nocturnally subperiodic, with peak concentrations occurring in the peripheral blood in the evening. Some dogs never develop microfilaremia, even though they support *D. immitis* adults and may have patent disease. These dogs are said to have occult infections.

Mosquitoes become infected with *Dirofilaria immitis* when they imbibe blood from a microfilaremic dog. In an average bloodmeal of 5 μL, a mosquito may ingest between five and 500 microfilariae. Within 48 h of ingestion, microfilariae migrate posteriorly in the midgut lumen to the Malpighian tubules and then into the distal cells of these tubules, where they develop intracellularly to "sausage forms" or first-stage larvae, taking about 4 days at 26°C. Some remain trapped in the midgut. If more than a few begin to develop in the tubules, the mosquito is likely to be killed. The first-stage larvae molt to the second-stage at about 8–10 days after ingestion. As they continue to grow they cause swelling and distention of the Malpighian tubules. At 12–14 days after ingestion, they molt to the third stage (Fig. 15.42). These filariform larvae break out of the Malpighian tubules and migrate through the hemolymph to the head and base of the mouthparts, then into the interior of the labium. The mosquito is then infective. The rate of these developmental processes is temperature dependent and varies with factors affecting competence for parasite development.

Vectors of *Dirofilaria immitis* differ with geographic region; many mosquito species in several genera are competent to transmit it. Grieve et al. (1983) listed 20 species field-caught in the United States, in the genera *Aedes*, *Psorophora*, *Anopheles*, and *Culex*, in which infective-stage larvae of *D. immitis* have been detected.

Other Filarial Nematodes of Animals

Other species of *Dirofilaria* infect mammals. These include *Dirofilaria ursi*, a bear parasite transmitted by the black fly *Simulium venustum*, and *D. roemeri*, a wallaroo (a type of small kangaroo) parasite transmitted by the horse fly *Dasybasis hebes*; and the following mosquito-transmitted *Dirofilaria* species: *D. repens* in canids; *D. carynodes* and *D. magnilarvatum* in monkeys; *D. scapiceps* in rabbits; *D. tenuis* in raccoons; and *D. subdermata* in porcupines.

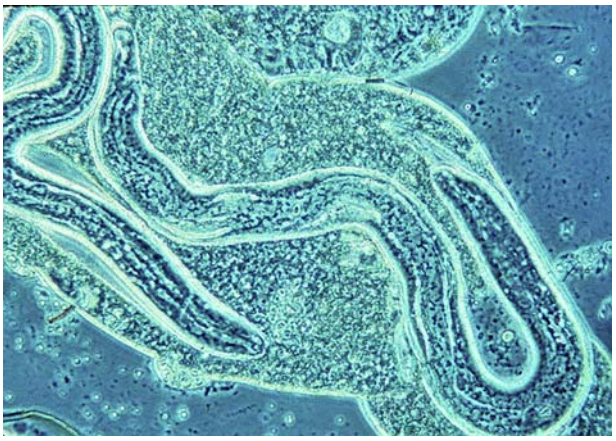


FIGURE 15.42 Filariform larva of *Dirofilaria immitis* within a Malpighian tubule of *Ae. aegypti*, prior to rupturing the tubule and migrating to the labium, where transfer to a dog occurs during blood-feeding. *Photograph by Bonnie Buxton.*

In addition to *Dirofilaria* spp., a large number of filarial nematodes in other genera of the Onchocercidae infect wild and domestic animals. Vectors include mosquitoes and a wide range of other blood-feeding Diptera, lice, fleas, mites, and ticks. The mosquito-borne onchocercid nematodes include species in the following genera; *Aproctella*, *Breinlia*, *Brugia*, *Cardiofilaria*, *Conispiculum*, *Dirofilaria*, *Deraiophoronema*, *Folyella*, *Loiana*, *Molinema*, *Pelecitus*, *Oswaldofilaria*, *Saurositus*, *Skrjabinofilaria*, *Waltonella*, and *Wuchereria* (Lavoipierre, 1958; Hawking and Worms, 1961; Bain and Chabaud, 1986; Anderson, 1992). *Brugia pahangi* of jirds (*Meriones*) is an important laboratory organism for studies on filariasis. *Brugia malayi* develops in the peritoneal cavity of gerbils, providing a laboratory infection model.

PREVENTION AND CONTROL

The four overlapping aims of mosquito control are to prevent mosquito bites, keep mosquito populations at acceptable densities, minimize mosquito–vertebrate contact, and reduce the longevity of female mosquitoes. All of these actions minimize the annoying and harmful effects of bites and blood loss, and they interrupt pathogen transmission. The eradication of either mosquito species or their associated diseases is no longer viewed as a viable objective, except in small, isolated regions or in the case of recent invasions. Two exemplary failures of the eradication approach, on a grand scale, were the World Health

Organization's global malaria eradication program and the Pan American Health Organization's attempt to eradicate *Aedes aegypti* from the Western Hemisphere. A notable exception was the successful elimination of the African immigrant *Anopheles gambiae* from Brazil. The more realistic objective of modern mosquito control programs is integrated pest management to reduce mosquito abundance and disease prevalence, using prudent combinations of methods.

Personal protection is the most direct and simple approach to prevention. Outdoor exposure can be avoided during peak mosquito activity, and window screens can prevent mosquito entry into houses and animal shelters. Head nets reduce annoyance and prevent bites about the face and neck. Bed nets, impregnated with synthetic pyrethroid and strung over beds at night, repel mosquitoes and kill those that land on the nets. Impregnated mesh suits with hoods work similarly, and can be worn over clothing. Other insecticidal devices create a repellent smoke or vapor that reduces mosquito attack in the immediate vicinity. Chemical repellents applied to skin or clothing prevent mosquitoes from landing or cause them to leave before probing. The most common one is *N,N*-diethyl-*meta*-toluamide, or DEET.

Organized control programs provides efficient, area-wide mosquito management at local, regional, or national levels. In the United States, mosquito programs typically are county-level abatement districts. These focus on the control of nuisance and vector species, but often also participate in surveillance for mosquito-borne disease pathogens. National organizations are usually parts of ministries of health and coordinate their disease and vector control efforts at that level. Especially in developing countries, there is now increasing emphasis on community cooperation, low technology, sustainability, and the integrated use of a variety of control tools that are adapted to local customs, conditions, and resources. Bed nets treated with synthetic pyrethroid insecticides have emerged as important community protection devices for malaria control and are now available in long-lasting, wash-durable formulations. They have been distributed in mass campaigns in countries of sub-Saharan Africa, resulting in reductions in malaria morbidity and mortality in large sections of some countries.

Habitat modification is a traditional and reliable tool in mosquito management. Adult resting places can be rendered unsuitable by harborage alteration. Changes in larval habitat that prevent oviposition, hatching, or larval development are called source reduction. Water is altered or eliminated in a variety of ways. This includes plastic foam beads that provide a floating barrier over latrine water, underground sewage lines, land drainage through ditches or underground tile pipes, waste tire shredding, trash-container disposal and natural container elimination, lids for water-storage barrels, vegetational changes in ponds, altered flow of tidal water through salt marshes, and water-level manipulation in reservoirs and rice fields. Each method is designed to interfere with specific features of a mosquito's natural history. Through appropriate application of ecological principles and an intimate knowledge of mosquito behavior and life cycles, desirable natural wetlands and newly created ones can be modified to minimize mosquito production while benefiting other wildlife.

Biological control of mosquitoes by predators or parasites has been studied extensively and has been reviewed by Chapman (1985), Beaty and Marquardt (1996), Hemingway (2005), and Floore (2007). Aerial predators, such as dragonflies, birds, and bats, receive much attention but do not specialize in adult mosquitoes and have little if any effect on their densities. Most efforts have been directed at the larval stage. Aquatic predators, both naturally occurring and introduced, include the mosquito fish (*Gambusia affinis*) and killifish (*Fundulus* spp.). Other fish, such as grass carp, e.g., *Tilapia* and *Cyprinus*, remove aquatic vegetation that provides harborage for larvae. Invertebrate predators include the predatory mosquito *Toxorhynchites*, several families of aquatic bugs and beetles, predatory copepods, hydras, and turbellarian flatworms; however, only copepods have been implemented with substantial success.

There have been attempts to develop the use of parasites and pathogens of mosquito larvae as control agents, including the nematode *Romanomermis culicivorax*; protozoans such as the ciliates *Lambornella* and *Tetrahymena*; the gregarine sporozoan *Ascogregarina*; and the microsporidian *Nosema*. Fungal pathogens include *Coelomomyces*, *Lagenidium*, *Culicinomyces*, and *Metarrhizium*. Viruses pathogenic to larvae include the iridescent viruses, densonucleosis viruses, polyhedrosis viruses such as the baculoviruses, and entomopox viruses. Generally, the above-mentioned parasites or pathogens of mosquito larvae are still in experimental stages of development, or they have limited effectiveness and have not been used routinely in operational programs.

An exception is the bacterium *Bacillus thuringiensis israelensis*, or *Bti*, which has been developed into commercial formulations since its original discovery in 1975. It is used extensively in mosquito control programs. Larvae die when they ingest crystalline, proteinaceous toxins produced by the bacterial cells during sporulation. The bacterium *Bacillus sphaericus* has a similar mode of action but is more specific. It is particularly effective against *Culex* larvae, and is more persistent in water, and more tolerant of water with a high organic content, than is *Bti*.

Genetic control, a category of biological control using a wide variety of genetic methods, has been successful against several insect pests; its practical application to mosquitoes, however, to halt pathogen transmission, is only recently gaining widespread traction. There are two

hypothetical approaches: (1) self-limiting systems; release of sterilized males or incompatible (female-killing) strains, resulting in attrition of the natural population, and (2) self-sustaining systems; replacement of natural vector populations with strains that are poor vectors or are refractory to the pathogens they formerly transmitted. The appeal of both approaches is that their effects are minimally disruptive to the environment and are species specific, because they depend on mate-seeking for their implementation. The special attraction of the replacement approach is that it is potentially self-sustaining, requiring no further input. However, it requires a drive mechanism to spread the refractory property through the mosquito population. Achieving driver-gene stability and selective propagation through males (to avoid enhancing the female population), among other developments and obstacles, have been reviewed by Rai (1996), Wood (2005), and Alphey (2014).

The intracellular bacterium *Wolbachia* has played a significant role in several of these potential genetic approaches to **vector manipulation**, including both self-limited and self-sustaining types. In addition, to its sex ratio—distorting and gene-drive features, *Wolbachia* interferes with the vector's competence for transmitting pathogens such as dengue virus.

The most prominent innovation among the **sterile-male techniques** is to release males carrying a dominant lethal gene or a genetic system that allows the altered males to sire only male offspring, which then continue spreading the gene in the dwindling natural population of females. For vector replacement, progress has been most rapid in developing transgenic *Anopheles* spp. that are refractory to infection by malaria parasites, transgenic *Aedes aegypti* that are incompetent to transmit dengue virus, and experimentation with gene-drive systems that allow their altered genomes to invade populations.

Chemical control is achieved with insecticides against either larvae or adults. **Larvicides** are placed in water where larvae develop, or where water will accumulate and provide habitat for larvae. Formerly used larvicides included inorganic compounds such copper arsenate, fuel oil, and organochlorine chemicals such as DDT and dieldrin. Currently, categories of registered larvicides are light mineral oils, organophosphates, and insect-growth regulators. Rapidly degradable oils spread over the water surface, penetrating the tracheal systems of larvae and pupae and suffocating them. Organophosphates, such as temephos, malathion, and chlorpyrifos, function as nerve poisons. The insect-growth regulator methoprene is a mimic of juvenile hormone and interferes with metamorphosis and emergence. The specific kind and formulation (dust, powder, water-soluble liquid, emulsion, oil-soluble liquid, granule, pellet, briquet) of the larvicide recommended depends on the biology of the target mosquito, the kind and size of habitat, the method of application, the chemical composition of the water, and the presence of nontarget organisms that might be adversely affected. Some can be formulated for slow release from a carrier. These may be applied to dry ground, releasing the active ingredient when inundated.

Adulticides are applied to surfaces where adults will rest or in the air where they fly. Residual insecticides applied to resting surfaces may retain their toxicity for days to months. They were central to the global malaria eradication program, in which DDT spraying of the inner walls of human dwellings at 6-month intervals killed all mosquitoes landing on these walls before or after taking blood. In areas where the vectors bit humans primarily indoors, this effectively interrupted most malaria transmission until mosquito populations developed resistance to the insecticide or when programs were abandoned. This approach is still used widely in some areas. Residual adulticides also can be used outdoors on vegetation or structures that serve as harborages. They tend to have short-term effects, because sunlight, wind, and rain cause the insecticide to degrade. A novel use for ivermectin, a systemic antiparasitic compound used widely against nematodes such as those causing river blindness (onchocerciasis) and nematode infections of livestock, appears to be its deleterious effects on mosquitoes such as *An. gambiae* in Africa when it feeds on the blood of the treated host. As a means of reducing mosquito populations or their vectorial capacity, this approach is being considered.

Adulticides intended for direct contact between airborne droplets and the mosquito are of two types: **thermal fogs** and low or **ultra-low volume (ULV) sprays**. Both can be applied from hand-carried equipment, motor vehicles, or aircraft. Thermal fogging involves mixing an insecticide with a combustible liquid such as kerosene. The mixture is heated, creating a fog of insecticide that drifts through the area to be treated. The ULV approach involves special nozzles and pumps that dispense fine droplets of insecticide, forming a mist that passes through the target area. Currently, insecticides registered for use in fogs and low-volume sprays are organophosphates, carbamates, pyrethrins, and synthetic pyrethroids. **Resistence to insecticides** is an important consequence of their use and has developed in many mosquito populations. The mechanisms of physiological resistance have been well characterized biochemically and genetically. **Behavioral resistance** also can develop. This is typically a change in adult feeding or resting behavior, so that mosquitoes no longer contact insecticide residues. Liu (2015) has reviewed the subject of mosquito resistance to insecticides.

Surveillance, which is at the core of effective mosquito control programs, determines mosquito distribution and abundance and degree of pathogen activity. The goal is to provide data so that control agencies can take action to prevent mosquito-related problems from occurring.

Unfortunately, there have been few control programs establishing action thresholds for mosquito density or infection rate, the levels of threat at which controls should be initiated. More often, action is based on human perception of a pest problem, conditions similar to past experience with disease outbreaks, or first detection of pathogen activity. Surveillance strategies and techniques for mosquito-borne encephalitis viruses have been presented by Moore et al. (1993) and Moore and Gage (1996). Bruce-Chwatt (1980) and Sasa (1976) reviewed traditional techniques for detecting malaria and filarial parasites, respectively, and several new ones are in use. Methods that make use of geographic information systems (GIS) and decision support systems (DSS) to predict, prevent, and control vector-borne diseases (e.g., dengue, malaria, and West Nile virus) have been evaluated by Eisen and Eisen (2011).

Control of Pathogen Transmission

Although all methods of reducing vector populations can lower the incidence of mosquito-borne disease, quantitative models of the dynamics of disease transmission have become important tools for setting realistic control objectives. They allow programs to focus efforts on parts of the pathogen-transmission system most vulnerable to attack. Useful references on this subject are Ross (1911), Macdonald (1957), Molineaux and Gramiccia (1980), Fine (1981), Koella (1991), and Dye (1992).

The Ross-Macdonald equation describes the case reproduction rate, the total number of new cases of a disease arising from a single infective case in a totally susceptible population. The vectorial capacity equation expresses that function on a daily basis using entomological parameters. Although the vectorial capacity measure is not epidemiologically comprehensive, it allows a comparison of the relative importance of different vectors and provides estimates of critical vector density, the adult mosquito density below which the case reproduction is less than one and the disease should die out. It also illustrates, mathematically, that even small changes in the interval between bites on susceptible hosts (which is squared) or in the longevity of vectors (which changes exponentially) cause large changes in transmission rates. The latter relationship has been critical in mounting effective disease-control operations, which target older females, rather than just female density in general.

More complex models sometimes show good agreement between predicted and observed results in extensive field studies of malaria (Molineaux and Gramiccia, 1980; Koella, 1991) and may become useful in establishing action thresholds. A simple and direct measure of transmission is the entomological inoculation rate (Onori and Grab, 1980), which is the product of the vector's human-biting rate and proportion of vectors that are infective.

Vaccines and drugs are important tools in protecting or treating humans and other animals susceptible to mosquito-borne disease. They serve not only to protect the individual but also to reduce transmission to others. Vaccines are available for several arboviral diseases, including yellow fever and Japanese encephalitis for humans and eastern, western, and Venezuelan encephalitis for equids. These vary in the duration of protection they provide. An experimental human vaccine against eastern equine encephalitis has been produced. None currently exists for the dengue viruses. Human malaria vaccines are under development, and some field trials have achieved limited success, but their wide-scale efficacy remains uncertain. The three kinds of malaria vaccines being considered use antigens from sporozoites, blood stages, or gametes; the last kind is called a transmission-blocking vaccine because the human antibodies take effect against stages that form within the midgut of the mosquito.

Among **drugs**, there exists a wide spectrum of antimalarials used for prophylaxis, therapy, or both. The most commonly used chemoprophylactic is chloroquine, against which there is now widespread resistance in *Plasmodium falciparum* and some *P. vivax* populations. Mefloquine is prescribed as a prophylactic for areas with resistant populations. Artemesinin, a drug derived from a plant long used by Chinese herbalists, is now widely used in derivative form in combination with other antimalarial compounds to treat uncomplicated malaria cases where resistance is widespread. Strickland (1991) presented a detailed review of malaria chemotherapy and chemoprophylaxis.

For lymphatic filariasis, diethylcarbamazine (DEC) is the standard chemotherapy, which reduces microfilaremia but does not kill adult worms. Advanced disease manifestations (e.g., elephantiasis) cannot be reversed, except by surgery, but sustained mass treatment of human populations can drive transmission to zero. This was achieved in parts of China in 1 year by the use of DEC-fortified cooking salt. Owing to the longevity of adult worms, mass treatment for 5–10 years is necessary to completely break the infection cycle in a community. Ivermectin and albendazole are two other drugs showing efficacy in lymphatic filariasis cases. Both DEC and ivermectin are used as chemoprophylaxis against dog heartworm infections.

REFERENCES AND FURTHER READING

Aliota, M. T., Bassit, L., Bradrick, S. S., Cox, B., Garcia-Blanco, M. A., et al. (2017). Zika in the Americas, year 2: What have we learned? What gaps remain? A report from the Global Virus Network. *Anti-viral, 144*, 223–246.

Allan, S. A., Day, J. F., & Edman, J. D. (1987). Visual ecology of biting flies. *Annual Review of Entomology, 32*, 297–316.

Alphey, L. (2014). Genetic control of mosquitoes. *Annual Review of Entomology, 59*, 205–224.

American Mosquito Control Association. (1979). *Mosquitoes and their control in the United States*. Fresno, California: American Mosquito Control Association.

Anderson, R. C. (1992). *Nematode parasites of Vertebrates: Their development and transmission*. Wallingford, Oxon, UK: C.A.B. International, 578 p.

Anonymous. (1994). Attractants for mosquito surveillance and control: A symposium. *Journal of the American Mosquito Control Association, 10*, 253–338.

Asman, S. M., McDonald, P. T., & Prout, T. (1981). Field studies of genetic control systems for mosquitoes. *Annual Review of Entomology, 26*, 289–318.

Bain, O., & Chabaud, A. G. (1986). Atlas des larves infestantes de filaires. *Annals of Tropical Medicine and Parasitology, 37*, 301–340.

Barr, A. R. (1958). The mosquitoes of Minnesota (Diptera: Culicidae: Culicinae). In *Agric. Expt. Sta., Tech. Bull. 228*. Univ. Minnesota, 154 p.

Barrett, A. D. T., & Higgs, S. (2007). Yellow fever: A disease that has yet to be conquered. *Annual Review of Entomology, 52*, 209–229.

Barsellos, C., & Lowe, R. (2014). Expansion of the dengue transmission area in Brazil: The role of climate and cities. *Tropical Medicine and International Health, 19*, 159–168.

Bates, M. (1949). *The natural history of mosquitoes*. New York: Macmillan (1965 edition: Harper & Row, New York). 378 pp.

Beaty, B. J., & Marquardt, W. C. (Eds.). (1996). *The biology of disease vectors*. Niwot, Colorado: University Press of Colorado, 632 p.

Beaty, B., Miller, B. R., Shope, R. E., Rozhon, E. J., & Bishop, D. H. (1982). Molecular basis of bunyavirus per os infection of mosquitoes: Role of the middle-sized RNA segment. *Proceedings of the National Academy of Sciences, 79*, 1295–1297.

Beerntsen, B. T., James, A. A., & Christensen, B. M. (2000). Genetics of mosquito vector competence. *Microbiology and Molecular Biology Reviews, 64*, 115–137.

Belkin, J. N. (1962). *The mosquitoes of the south Pacific (Diptera: Culicidae)*. Los Angeles: Univ. California Press. Vol. 1, 608 p., Vol. 2, 412 figs.

Belkin, J. N., Schick, R. X., Galindo, P., & Aitken, T. H. (1965). Mosquito studies (Diptera: Culicidae) I. A project for a systematic study of the mosquitoes of Middle America. *Contributions of the American Entomological Institute, 2*, 1–17.

Bentley, M. D., & Day, J. F. (1989). Chemical ecology and behavioral aspects of mosquito oviposition. *Annual Review of Entomology, 34*, 401–421.

Besansky, N. J., Finnerty, V., & Collins, F. H. (1992). A molecular genetic perspective on mosquitoes. *Advances in Genetics, 30*, 123–184.

Bock, G. R., & Cardew, G. (Eds.). (1996). *Olfaction in mosquito-host interactions*. Chichester, UK: Wiley, 331 p.

Boddy, D. W. (1948). An annotated list of the Culicidae of Washington. *The Pan-Pacific Entomologist, 24*, 85–94.

Bohart, R. M., & Washino, R. K. (1978). *Mosquitoes of California*. Berkeley: Div. Agric. Sci., Univ. Calif., 153 p.

Boreham, P. F. L., & Atwell, R. B. (Eds.). (1988). *Dirofilariasis*. Boca Raton, FL: CRC Press, 249 p.

Bosik, J. J. (1997). Common names of insects and related organisms. *Entomological Society of America*, 200 p.

Bowen, M. F. (1991). The sensory physiology of host-seeking behavior in mosquitoes. *Annual Review of Entomology, 36*, 139–158.

Bowen, G. S., & Francy, D. B. (1980). Surveillance. In T. P. Monath (Ed.), *St. Louis Encephalitis* (pp. 473–499). Washington, DC: Am. Pub. Hlth. Assoc.

Boyd, M. F. (1941). An historical sketch of the prevalence of malaria in North America. *The American Journal of Tropical Medicine and Hygiene, 21*, 223–244.

Boyd, M. F. (Ed.). (1949). *Malariology: A comprehensive survey of all aspects of this group of diseases from a global standpoint*. Philadelphia: Saunders, 1,643 p.

Bradley, T. J. (1987). Physiology of osmoregulation in mosquitoes. *Annual Review of Entomology, 32*, 439–462.

Brasil, et al. (2017). Outbreak of human malaria caused by *Plasmodium simium* in the Atlantic forest in Rio de Janeiro: A molecular epidemiological investigation. *Lancet Global Health, 5*, e1038–e1046.

Brown, M. R., & Lea, A. O. (1990). Neuroendocrine and midgut endocrine systems in the adult mosquito. *Advances in Disease Vector Research, 6*, 29–58.

Bruce-Chwatt, L. J. (1980). *Essential malariology*. London: William Heinemann Medical Books Ltd., 367 p.

Burke, D. S., & Leake, C. J. (1988). Japanese encephalitis. In T. P. Monath (Ed.), *The Arboviruses: Epidemiology and ecology* (Vol. 3, pp. 63–92). Boca Raton, FL: CRC Press.

Burkett-Cadena, N. D. (2013). *Mosquitoes of the southeastern United States*. Tuscaloosa: University of Alabama Press, 188 p.

Burkot, T. R., & Graves, P. M. (1994). Human malaria transmission: Reconciling field and laboratory data. *Advances in Disease Vector Research, 10*, 149–182.

Burt, A. (2014). Heritable strategies for controlling insect vectors of disease. *Philosophical Transactions of the Royal Society B, 369*, 20130432. https://doi.org/10.1098/rstb.2013.0432.

Calisher, C. H., & Karabatsos, N. (1988). Arbovirus serogroups: Definition and geographic distribution. In T. P. Monath (Ed.), *The Arboviruses: Epidemiology and ecology* (Vol. 1, pp. 19–57). Boca Raton, FL: CRC Press.

Calisher, C. H., & Thompson, W. H. (Eds.). (1983). *California serogroup viruses: Proceedings of an international Symposium, held in Cleveland, Ohio, November 12 and 13, 1982*. New York: A. R. Liss.

Cano, J., Maria P Rebollo, M. P., Golding, N., Pullan, R. L., Crellen, T., Soler, A., et al. (2014). The global distribution and transmission limits of lymphatic filariasis: Past and present. *Parasites and Vectors, 7*, 466.

Cardé, R. T., & Gibson, G. (2010). Host finding by female mosquitoes: Mechanisms of orientation to odours and other host cues. In W. Takken, & B. G. J. Knols (Eds.), *Ecology and control of vector-borne diseases* (Vol. 2, pp. 115–141). The Netherlands: Wageningen Academic Publishers.

Carlson, J., Olson, K., Higgs, S., & Beaty, B. (1995). Molecular genetic manipulation of mosquito vectors. *Annual Review of Entomology, 40*, 359–388.

Carpenter, S. J. (1941). *The mosquitoes of Arkansas*. Little Rock, Arkansas: Arkansas State Board of Health, 87 p.

Carpenter, S. J., & LaCasse, W. J. (1955). *Mosquitoes of North America (North of Mexico)*. Berkeley, California: Univ. California Press, 360 p.

Centers for Disease Control. (1977). *Mosquitoes of public health importance and their control*. Hhs Publ. No. (Centers for disease control) 82-8140. Atlanta, GA: U.S. Dept. of Health & Human Services, 55 p.

Chapman, H. C. (1966). *The mosquitoes of Nevada*. Carson City, Nevada: U. S. Dept. Agric. & Univ. Nevada, 43 p.

Chapman, H. C. (Ed.). (1985). *Biological control of mosquitoes*. Fresno, California: American Mosquito Control Association. Bull. No. 6.

Christophers, S. R. (1911). Development of the egg follicle in Anophelines. *Paludism, 2*, 73−88.

Christophers, S. R. (1960). *Aedes aegypti (L.). The yellow fever mosquito. Its life history, bionomics and structure*. London: Cambridge Univ. Press, 739 p.

Clements, A. N. (1963). *The physiology of mosquitoes*. New York: Pergamon, 393 p.

Clements, A. N. (1992). *The biology of mosquitoes. Vol. 1. Development, nutrition and reproduction*. New York: Chapman & Hall, 509 p.

Clements, A. N. (1999). *The biology of mosquitoes. Vol. 2. Sensory reception and Behaviour*. Wallingford, U.K: CABI Publ., 740 p.

Clements, A. N. (2011). *The biology of mosquitoes. Vol. 3. Transmission of viruses and interactions with bacteria*. Wallingford, U.K: CABI Publ., 592 p.

Coatney, G. R., Collins, W. E., Warren, Mc, W., & Contacos, P. G. (1971). *The primate malarias*. Washington, DC: US Govt. Printing Office, 366 p.

Collins, W. E., & Aikawa, M. (1993). Plasmodia of nonhuman primates. In J. P. Kreier (Ed.), *Parasitic Protozoa* (2nd ed., Vol. 5, pp. 105−134). NY: Academic Press.

Collins, W. E. (2012). *Plasmodium knowlesi*: A malaria parasite of monkeys and humans. *Annual Review of Entomology, 57*, 107−121.

Corbet, P. S., Williams, M. C., & Gillett, J. D. (1961). O'nyong nyong fever: An epidemic virus disease in East Africa: IV. Vector studies at epidemic sites. *Transactions of the Royal Society of Tropical Medicine and Hygiene, 55*, 463.

Cox, F. E. G. (1993). Plasmodia of rodents. In J. P. Kreier (Ed.), *Parasitic protozoa* (2nd ed., Vol. 5, pp. 49−104). NY: Academic Press.

Cox, G. W. (1944). *The mosquitoes of Texas*. Austin, Texas: Texas State Health Dept., 100 p.

Curtis, C. F. (Ed.). (1990). *Appropriate technology in vector control*. Boca Raton, Florida: CRC Press, 233 p.

Dale, P. E. R., & Hulsman, K. (1990). A critical review of salt marsh management methods for mosquito control. *Aquatic Sciences, 3*, 281−311.

Darsie, R. F., Jr. (1951). Pupae of the culicine mosquitoes of the northeastern United States (Diptera, Culicidae, Culicini). *Cornell University Agricultural Experiment Station, 304*, 1−67.

Darsie, R. F., Jr. (1989). Keys to the genera, and to the species of five minor genera, of mosquito pupae occurring in the Nearctic region (Diptera: Culicidae). *Mosquito Systematics, 21*, 1−10.

Darsie, R. F., Jr., & Morris, C. D. (1998). *Keys to the adult females and fourth instar larvae of the mosquitoes of Florida (Diptera, Culicidae)*. Bull. Florida Mosq. Control Assoc., No. 1. Vero Beach: Florida Med. Entomol. Lab., IFAS, Univ. Florida, 156 pp.

Darsie, R. F., Jr., & Ward, R. A. (1981). Identification and geographical distribution of the mosquitoes of North America, north of Mexico. *Mosquito Systematics Supplement, 1*, 1−313.

Darsie, R. F., Jr., & Ward, R. A. (2004). *Identification and geographical distribution of the mosquitoes of North America, North of Mexico*. Gainesville: Univ. Press of Florida, 384 p.

Davidson, E. W., & Becker, N. (1996). Microbial control of vectors. In B. J. Beaty, & W. C. Marquardt (Eds.), *The biology of disease vectors* (pp. 549−663). Niwot: Univ. Press of Colorado.

de Barjac, H., & Sutherland, D. J. (Eds.). (1989). *Bacterial control of mosquitoes and black flies*. New Brunswick: Rutgers Univ. Press, 350 p.

Debenham, M. L. (Ed.). (1987a). *Culicidae of the Australasian region. Vol. 4. Nomenclature, synonymy, literature, distribution, biology and relation to Disease: Genus* Aedes, subgenera Scutomyia, Stegomyia, Verrallina. Canberra: Australian Gov. Publ. Service. Entomology monograph No. 2 (in part) 324 p.

Debenham, M. L. (Ed.). (1987b). *Culicidae of the Australasian region. Vol. 5. Nomenclature, synonymy, literature, distribution, biology and relation to Disease: Genus* Anopheles, subgenera Anopheles, Cellia. Canberra: Australian Gov. Publ. Service. Entomology monograph No. 2 (in part) 315 p.

Debenham, M. L. (Ed.). (1988a). *Culicidae of the Australasian region. Vol. 6. Nomenclature, synonymy, literature, distribution, biology and relation to Disease: Genera* Armigeres, Bironella and Coquillettidia. Canberra: Australian Gov. Publ. Service. Entomology monograph No. 2 (in part), 124 p.

Debenham, M. L. (Ed.). (1988b). *Culicidae of the Australasian region. Vol. 9. Nomenclature, synonymy, literature, distribution, biology and relation to Disease: Genus* Culex (*subgenera* Lutzia, Neoculex, *subgenus Undecided), genera* Culiseta, Ficalbia, Heizmannia, Hodgesia, Malaya, Mansonia. Canberra: Australian Gov. Publ. Service. Entomology monograph No. 2 (in part) 162 p.

Debenham, M. L. (Ed.). (1988c). *Culicidae of the Australasian region. Vol. 10. Nomenclature, synonymy, literature, distribution, biology and relation to Disease: Genera* Maorigoeldia, Mimomyia, Opifex, Orthopodomyia, Topomyia, Toxorhynchites. Canberra: Australian Gov. Publ. Service. Entomology monograph No. 2 (in part) 105 p.

Debenham, M. L. (Ed.). (1989a). *Culicidae of the Australasian region. Vol. 7. Nomenclature, synonymy, literature, distribution, biology and relation to disease: Genus* Culex, subgenera Acallyntrum, Culex. Canberra: Australian Gov. Publ. Service. Entomology monograph No. 2 (in part) 281 p.

Debenham, M. L. (Ed.). (1989b). *Culicidae of the Australasian region. Vol. 8. Nomenclature, synonymy, literature, distribution, biology and relation to Disease: Genus Culex, subgenera* Culiciomyia, Eumelanomyia, Lophoceraomyia. Canberra: Australian Gov. Publ. Service. Entomology monograph No. 2 (in part) 171 p.

Debenham, M. L., Hicks, M. M., & Griffiths, M. (Eds.). (1989). *Culicidae of the Australasian region. Vol. 12. Summary of taxonomic changes, revised alphabetical list of species, supplementary bibliography, errata and addenda, geographic guide to species, synopsis of disease relationships, indexes*. Canberra: Australian Gov.Publ. Service. Entomology monograph No. 2 (in part) 217 p.

DeFoliart, G. R., Grimstad, P. R., & Watts, D. M. (1987). Advances in mosquito-borne arbovirus/vector research. *Annual Review of Entomology, 32*, 479−505.

Denlinger, D. L., & Armbruster, P. A. (2014). Mosquito diapause. *Annual Review of Entomology, 59*, 73−93.

Detinova, T. S. (1962). Age-grading methods in Diptera of medical importance. *World Health Organization Monograph Series, 47*, 1−216.

de Zulueta, J. (1994). Malaria and ecosystems: From prehistory to post-eradication. *Parassitologia, 36*, 7−15.

Dickinson, W. E. (1944). The mosquitoes of Wisconsin. *Bulletin of the Public Museum of the City of Milwaukee, 8*, 269−365.

Dixon, R. D., & Brust, R. A. (1972). Mosquitoes of Manitoba. III. Ecology of larvae in the Winnipeg area. *Canadian Entomologist, 104*, 961−968.

Downes, J. A. (1969). The swarming and mating flight of Diptera. *Annual Review of Entomology, 14*, 271−298.

Dye, C. (1992). The analysis of parasite transmission by bloodsucking insects. *Annual Review of Entomology, 37*, 1–19.

Eisen, L., & Eisen, R. J. (2011). Using geographic information systems and decision support systems for the prediction, prevention, and control of vector-borne diseases. *Annual Review of Entomology, 56*, 41–61.

Evenhuis, N. L., & Gon, S. M., III (1989). Family Culicidae. In N. L. Evenhuis (Ed.), *Catalog of the Diptera of the Australasian and Oceanian regions* (pp. 191–218). Honolulu: Bishop Museum Press.

Fauci, A. S., & Morens, D. M. (2016). Zika virus in the Americas—yet another arbovirus threat. *New England Journal of Medicine, 374*, 601–604.

Fauquet, C. M., Mayo, M. A., Maniloff, J., Desselberger, U., & Ball, L. A. (Eds.). (2005). *Virus taxonomy. 8th report of the international committee on taxonomy of viruses*. New York: Academic Press, 1162 p.

Favoretto, S., Araújo, D., Oliveira, D., Duarte, N., Mesquita, F., Zanotto, P., et al. (2016). First detection of Zika virus in neotropical primates in Brazil: A possible new reservoir. *BioRχiv*. https://doi.org/10.1101/049395.

Fine, P. E. M. (1981). Epidemiological principles of vector-mediated transmission. In J. J. McKelvey, B. F. Eldridge, & K. Maramorosch (Eds.), *Vectors of disease agents* (pp. 77–91). New York, NY: Praeger Publishers.

Floore, T. G. (Ed.). (2007). Biorational control of mosquitoes. *Journal of the American Mosquito Control Association, 23*(2), 1–328.

Foote, R. H., & Cook, D. R. (1959). Mosquitoes of medical importance. In *Agric. handbook* (Vol. 152, pp. 1–158). U.S. Dept. Agric.

Forattini, O. P. (1965). *Entomologia medica. Vol. 1: Parte geral., Diptera, Anophelini, 662 pp.; Vol. 2:* Culicini: Culex, Aedes e Psorophora. *Vol. 3:* Culicini*: Haemagogus, Mansonia, Culiseta, Sabethini. Toxorhychitini. Arboviruses. Filariose bancroftiana. Genetica*. Universidade de Sao Paulo, Faculdade de Higiene e Saude Publica.

Foster, W. A. (1995). Mosquito sugar feeding and reproductive energetics. *Annual Review of Entomology, 40*, 443–474.

Fox, A. S., & Brust, R. A. (1994). How do dilatations form in mosquito ovarioles? *Parasitology Today, 10*, 19–23.

Gaffigan, T. V., & Ward, R. A. (1985). Index to the second supplement to "A catalog of the mosquitoes of the world," with corrections and additions. *Mosquito Systematics, 17*, 52–63.

Gartrell, F. E., Cooney, J. C., Chambers, G. P., & Brooks, R. H. (1981). TVA mosquito control 1934-1980—experience and current program tends and developments. *Mosquito News, 41*, 302–322.

Gerhardt, R. W. (1966). Agric. Expt. Sta. Bull. *South Dakota mosquitoes and their control, 531*. Brookings: South Dakota State Univ., 80 p.

Gilles, H. M., & Warrell, D. A. (1994). *Bruce-chwatts essential malariology* (3rd ed.). Boston: Little, Brown, & Co., 340 p.

Gillett, J. D. (1971). *The Mosquito: Its life, activities, and impact on human affairs*. New York: Doubleday, 358 p.

Gillies, M. T. (1988). Anopheline mosquitos: Vector behaviour and bionomics. In W. H. Wernsdorfer, & I. McGregor (Eds.), *Malaria principles and practice of malariology* (Vol. 1, pp. 453–485). Edinburgh: Churchill Livingstone.

Gillies, M. T., & Coetzee, M. (1987). A supplement to the Anophelinae of Africa south of the Sahara (afrotropical region). *Publications of the South African Institute for Medical Research, 55*, 1–143.

Gillies, M. T., & de Meillon, B. (1968). The Anophelinae of Africa south of the Sahara (Ethiopian geographical region). *Publications of the South African Institute for Medical Research, 54*, 1–343.

Gjullin, C. M., Sailer, R. I., Stone, A., & Travis, B. V. (1961). The mosquitoes of Alaska. In *Agric. handbook* (Vol. 182, pp. 1–98). U. S. Dept. Agric.

Gordon, R. M., & Lavoipierre, M. M. J. (1962). *Entomology for students of medicine*. Oxford: Blackwell Science, 353 p.

Grieve, R. B., Lok, J. B., & Glickman, L. T. (1983). Epidemiology of canine heartworm infection. *Epidemiologic Reviews, 5*, 220–246.

Grimstad, P. R. (1988). California group virus disease. In T. P. Monath (Ed.), *The Arboviruses: Epidemiology and ecology* (Vol. 2, pp. 99–136). Boca Raton, FL: CRC Press, Inc.

Grove, D. (1990). *A history of human helminthology*. Wallingford, UK: C.A.B. International, 848 p.

Gubler, D. J. (1988). Dengue. In T. P. Monath (Ed.), *The Arboviruses: Epidemiology and ecology* (Vol. 2, pp. 223–260). Boca Raton, FL: CRC Press.

Gubler, D. J., & Bhattacharya, N. C. (1974). A quantitative approach to the study of bancroftian filariasis. *The American Journal of Tropical Medicine and Hygiene, 23*, 1027–1036.

Gubler, D. J., & Clark, G. G. (1994). Community-based integrated control of *Aedes aegypti*: A brief overview of current programs. *The American Journal of Tropical Medicine and Hygiene, 50*, 50–60.

Gubler, D. J., Ooi, E. E., Vasudevan, S., & Farrar, J. (Eds.). (2014). *Dengue and dengue hemorrhagic fever* (2nd ed.). Wallingford, UK: CAB International, 624 p.

Gutsevich, A. V., Monchadskii, A. S., & Shtakel'berg, A. A. (1971). Mosquitoes, family Culicidae. Fauna of the U.S.S.R.: Diptera. *Academy of Sciences of the USSR, Zoological Institute, Leningrad, 3*(4), 1–408 (English translation 1974, Israel Program for Scientific Translations.).

Gwadz, R., & Collins, F. H. (1996). Anopheline mosquitoes and the agents they transmit. In B. J. Beaty, & W. C. Marquardt (Eds.), *The biology of disease vectors* (pp. 73–84). Niwot: Univ. Press of Colorado.

Gyapong, J. O., Gyapong, M., & Adjei, S. (1996). The epidemiology of acute adenolymphangitis due to lymphatic filariasis in northern Ghana. *The American Journal of Tropical Medicine and Hygiene, 54*, 591–595.

Hagedorn, H. H. (1994). The endocrinology of the adult female mosquito. *Advances in Disease Vector Research, 10*, 109–148.

Hagedorn, H. H. (1996). Physiology of mosquitoes. In B. J. Beaty, & W. C. Marquardt (Eds.), *The biology of disease vectors* (pp. 273–297). Niwot: Univ. Press of Colorado.

Hall, A. B., Basu, S., Jiang, X., Qi, Y., Timoshevskiy, V. A., Biedler, J. K., et al. (2015). A male-determining factor in the mosquito. *Aedes aegypti Science, 348*, 1268–1270.

Harbach, R. E., & Kitching, I. J. (1998). Phylogeny and classification of the Culicidae (Diptera). *Systematic Entomology, 23*, 327–370.

Harbach, R. E., & Knight, K. L. (1980). *Taxonomist's glossary of mosquito anatomy*. Marlton, New Jersey: Plexus Publishing, 415 p.

Harbach, R. E., & Knight, K. L. (1981). Corrections and additions to taxonomists' glossary of mosquito anatomy. *Mosquito Systematics, 13*, 201–217.

Harris, K. F. (Ed.). (1985). *Advances in disease vector research. (formerly Current topics in disease vector research)* (Vol. 1). New York: Springer-Verlag.

Hardy, J. L. (1988). Susceptibility and resistance of vector mosquitoes. In T. P. Monath (Ed.), *The Arboviruses: Epidemiology and ecology* (Vol. 1, pp. 87–126). Boca Raton, FL: CRC Press.

Harrison, G. (1978). *Mosquitoes, malaria and man: A history of the hostilities since 1880*. London: John Murray, 314 p.

Hawking, F., & Worms, M. (1961). Transmission of filarioid nematodes. *Annual Review of Entomology, 6*, 413–432.

Hawley, W. A. (1988). The biology of *Aedes albopictus*. *Journal of the American Mosquito Control Association, 4*(Suppl. 1), 1–40.

Hawley, W. A., Reiter, P., Copeland, R. S., Pumpuni, C. B., & Craig, G. B., Jr. (1987). *Aedes albopictus* in North America: Probable introduction in tires from northern Asia. *Science, 236*, 1114–1116.

Hayes, C. G. (1988). West Nile fever. In T. P. Monath (Ed.), *The Arboviruses: Epidemiology and ecology* (Vol. 5, pp. 59–88). Boca Raton, FL: CRC Press.

Headlee, T. J. (1945). *The mosquitoes of New Jersey and their control*. New Brunswick, NJ: Rutgers Univ. Press, 326 p.

Hearle, E. (1926). *The mosquitoes of the lower Fraser valley, British Columbia, and their control*. National Res. Council Rpt. No. 17, Ottawa.

Hemingway, J. (2005). Biological control of mosquitoes. In W. C. Marquardt (Ed.), *Biology of disease vectors* (2nd ed., pp. 649–660). New York: Elsevier Academic Press.

Hicks, M. M. (Ed.). (1989). *The Culicidae of the Australasian Region: Vol. 11, nomenclature, synonymy, literature, distribution, biology and relation to disease: Genera* Tripteroides, Uranotaenia, Wyeomyia, Zeugnomyia. Canberra: Austalian Govt. Publ. Service. Entomology Monogr. No. 2 306 p.

Higgs, S., & Beaty, B. J. (1996). Rearing and containment of mosquito vectors. In B. J. Beaty, & W. C. Marquardt (Eds.), *The biology of disease vectors* (pp. 595–605). Niwot: Univ. Press of Colorado.

Hoc, T. Q. (1996). Application of the ovarian oil injection and ovariolar separation techniques for age grading hematophagous Diptera. *Journal of Medical Entomology, 33*, 290–296.

Hopkins, C. C., Hollinger, F. B., Johnson, R. F., Dewlett, H. J., Newhouse, V. F., & Chamberlain, R. W. (1975). The epidemiology of St. Louis encephalitis in Dallas, Texas, 1966. *American Journal of Epidemiology, 102*, 1–15.

Hopkins, G. H. E. (1952). *Mosquitoes of the Ethiopian region I. Larval bionomics of mosquitoes and taxonomy of culicine larvae* (2nd. ed). London: British Museum (Nat. Hist.), 355 p.

Horsfall, W. R. (1955). *Mosquitoes. Their bionomics and relation to disease*. New York: Ronald Press, 723 p.

Horsfall, W. R., Fowler, H. W., Jr., Moretti, L. J., & Larsen, J. R. (1973). *Bionomics and embryology of the inland floodwater mosquito Aedes vexans*. Urbana, Illinois: Univ. Illinois Press, 211 p.

Iversson, L. B. (1988). Rocio encephalitis. In T. P. Monath (Ed.), *The arboviruses: Epidemiology and ecology* (Vol. 4, pp. 77–92). Boca Raton, FL: CRC Press.

Johnston, R. E., & Peters, C. J. (1996). Alphaviruses. In B. N. Fields, D. M. Knipe, P. M. Howley, et al. (Eds.), *Fields virology* (3rd ed., pp. 843–898). Philadelphia: Lippincott-Raven Publishers.

Jones, J. C. (1978). The feeding behavior of mosquitoes. *Scientific American, 238*, 138–148.

Jupp, P. G., & McIntosh, B. M. (1988). Chikungunya virus disease. In T. P. Monath (Ed.), *The arboviruses: Epidemiology and ecology* (Vol. 2, pp. 137–158). Boca Raton, Florida: CRC Press.

Kay, B. H., & Aaskov, J. G. (1988). Ross River virus (epidemic polyarthritis). In T. P. Monath (Ed.), *The arboviruses: Epidemiology and ecology* (Vol. 4, pp. 93–112). Boca Raton, Florida: CRC Press.

Kent, B. J. (2009). Molecular methods for arthropod bloodmeal identification and applications to ecological and vector-borne disease studies. *Molecular Ecology Resources, 9*, 4–18.

Kettle, D. S. (1995). *Medical and veterinary entomology* (2nd ed.). Wallingford, UK: CAB International, 725 p.

King, W. V., Bradley, G. H., Smith, C. N., & McDuffie, W. C. (1960). A handbook of the mosquitoes of the southeastern United States. In *Agric. handbook* (Vol. 173, pp. 1–188). U.S. Dept. Agric.

Kistler, K. E., Vosshall, L. B., & Matthews, B. J. (2015). Genome engineering with CRISPR- Cas9 in the mosquito *Aedes aegypti*. *Cell Reports, 11*, 51–60.

Klowden, M. J. (1996). Vector behavior. In B. J. Beaty, & W. C. Marquardt (Eds.), *The biology of disease vectors* (pp. 34–50). Niwot: Univ. Press of Colorado.

Knight, K. L. (1978). Supplement to a catalog of the mosquitoes of the world (Diptera: Culicidae). In *Thomas say found, Suppl. to Vol. VI*. College Park, Maryland: Entomol. Soc. Am., 107 p.

Knight, K. L., & Stone, A. (1977). A catalog of the mosquitoes of the world (Diptera: Culicidae). In *Thomas say found* (2nd ed., Vol. VI). College Park, Maryland: Entomol. Soc. Am., 611 pp. Updates are presented online by the Walter Reed Biosytematics Unit: http://www.mosquitocatalog.org/main.asp.

Knight, K. L., & Wonio, M. (1969). *Mosquitoes of Iowa (Diptera: Culicidae)*. Agric. & Home Econ. Expt. Sta., Special Rpt. No. 61. Ames: Iowa State Univ. Sci. Tech, 79 p.

Koella, J. C. (1991). On the use of mathematical models of malaria transmission. *Acta Tropica, 49*, 1–25.

Kramer, L. D., Styer, L. M., & Ebel, G. D. (2008). A global perspective on the epidemiology of West Nile virus. *Annual Review of Entomology, 53*, 61–81.

LaCasse, W. J., & Yamaguti, S. (1950). *Mosquito fauna of Japan and Korea*. Kyoto: Off. Surgeon, 8th U. S. Army, 268 p.

Lacey, L. A., & Undeen, A. H. (1986). Microbial control of black flies and mosquitoes. *Annual Review of Entomology, 31*, 265–296.

Laird, M. (1988). *The natural history of larval mosquito habitats*. New York: Academic Press, 555 p.

Laird, M., & Miles, J. W. (Eds.). (1983). *Integrated mosquito control methodologies. Vol. 1. Experience and components from conventional chemical control*. New York: Academic Press, 369 p.

Laird, M., & Miles, J. W. (Eds.). (1985). *Integrated mosquito control methodologies. Vol. 2. Biocontrol and other innovative components, and future directions*. New York: Academic Press, 444 p.

Lalremruata, A., Magris, M., Vivas-Martínez, S. V., Koehler, M., Esen, M., Kempaiah, P., et al. (2015). Natural infection of *Plasmodium brasilianum* in humans: Man and monkey share quartan malaria parasites in the Venezuelan Amazon. *EBioMedicine, 2*, 1186–1192.

Lane, J. (1953). *Neotropical Culicidae, 3 vols*. Sao Paulo: University of Sao Paulo, 1112 p.

Laven, H. (1967). Eradication of *Culex pipiens fatigans* through cytoplasmic incompatibility. *Nature, 216*, 383–384.

Lavoipierre, M. M. (1958). Studies on the host-parasite relations of filarial nematodes and their arthropod hosts. II. The arthropod as a host to the nematode; a brief appraisal of our present knowledge, based on a study of the more important literature from 1878 to 1957. *Annals of Tropical Medicine and Parasitology, 52*, 326–345.

Lee, D. J., Hicks, M. M., Griffiths, M., Russell, R. C., & Marks, E. N. (1980). The Culicidae of the Australasian region. In *Entomol. Monogr. No. 2 (in part)* (Vol. 1). Canberra: Austal. Gov. Publ. Service.

Lee, D. J., Hicks, M. M., Griffiths, M., Russell, R. C., & Marks, E. N. (1982). *The Culicidae of the Australasian region. Vol. 2. Nomenclature, synonymy, literature, distribution, biology and relation to disease: Genus* Aedeomyia*, genus* Aedes *(subgenera* Aedes*,* Aedimorphus*,* Chaetocruiomyia*,* Christophersiomyia*,* Edwardsaedes and Finlaya*)*. Canberra: Austral. Gov. Publ. Service. Entomol. Monogr. No. 2 (in part) 286 p.

Lee, D. J., Hicks, M. M., Griffiths, M., Russell, R. C., & Marks, E. N. (1984). *The Culicidae of the Australasian region. Vol. 3. Nomenclature, synonymy, literature, distribution, biology and relation to disease: Genus* Aedes*, subgenera* Geokusea*,* Halaedes*,* Huaedes*,* Leptosomatomyia*,* Levua*,* Lorrainea*,* Macleaya*,* Mucidus*,* Neomelanoconion*,* Nothoskusea*,* Ochlerotatus*,* Paraedes*,* Pseudoskusea*,* Rhinoskusea. Canberra: Austral. Gov. Publ. Service. Entomol. Monogr. No. 2 (in part) 257 p.

Lees, S. R., Knols, B., Bellini, R., Benedict, M. Q., Bheecarry, A., et al. (2014). Review: Improving our knowledge of male mosquito biology in relation to genetic control programmes. *Acta Tropica, 132S*, S2−S11.

Lindsay, S. W., & Gibson, M. E. (1988). Bednets revisited: Old idea, new angle. *Parasitology Today, 4*, 270−272.

Linthicum, K. J. (2012). Summary of the symposium Global Perspective on the *Culex pipiens* Complex in the 21st Century: the interrelationship of *Culex pipiens, quinquefasciatus, molestus* and others. *Journal of the American Mosquito Control Association, 24*(issue 4S), 152−155.

Linthicum, K. J., Britch, S. C., & Anyamba, A. (2016). Rift Valley fever: An emerging mosquito-borne disease. *Annual Review of Entomology, 61*, 395−415.

Lounibos, L. P., Rey, J. R., & Frank, J. H. (Eds.). (1985). *Ecology of mosquitoes: Proceedings of a workshop*. Vero Beach: Florida Med. Entomol. Lab., 579 p.

Liu, N. (2015). Insecticide resistance in mosquitoes: Impact, mechanisms, and research directions. *Annual Review of Entomology, 60*, 537−559.

Lu, B. L., & Su, L. (1987). *A handbook for the identification of Chinese Aedine mosquitoes*. Beijing: Science Press, 160 p. [in Chinese].

Lu, B. L., Chen, B. H., Xu, R., & Ji, S. (1988). *A checklist of Chinese mosquitoes (Diptera: Culicidae)*. Beijing: Guizhu People's Publ. House, 164 p. [In Chinese, English introduction].

Macdonald, G. (1957). *The epidemiology and control of malaria*. London: Oxford University Press, 266 p.

Mail, G. A. (1934). *The mosquitoes of Montana*. Montana State College, Agric. Expt. Sta. Bull. No. 288, Bozeman, Montana. 72 p.

Marquardt, W. C. (Ed.). (2005). *Biology of disease vectors* (2nd ed.). New York: Elsevier Academic Press, 785 p.

Marshall, I. D. (1988). Murray valley and Kunjin encephalitis. In T. P. Monath (Ed.), *The Arboviruses: Epidemiology and ecology* (Vol. 3, pp. 151−190). Boca Raton, Florida: CRC Press.

Marshall, J. F. (1938). *The British mosquitoes*. London: British Museum (Natural History), 341 p.

Matheson, R. (1944). *Handbook of the mosquitoes of North America* (2nd ed.). Ithaca, NY: Comstock Publishing Co., 314 p.

Mattingly, P. F. (1969). *The biology of mosquito-borne disease*. London: Allen & Unwin, 184 p.

Mattingly, P. F. (1971). Contributions to the mosquito fauna of southeast Asia. XII. Illustrated keys to the genera of mosquitoes (Diptera: Culicidae). *Contributions of the American Entomological Institute, 7*, 1−84.

Mattingly, P. F. (1973). Culicidae (mosquitoes). In K. G. V. Smith (Ed.), *Insects and other arthropods of medical importance* (pp. 37−107). London: British Museum (Natural History).

McDonald, J. L., Sluss, T. P., Lang, J. D., & Roan, C. C. (1973). *Mosquitoes of Arizona*. Agric. Expt. Sta., Tech. Bull. No. 205. Tucson: Univ. Arizona, 21 p.

McIver, S. B. (1982). Sensilla of mosquitoes (Diptera: Culicidae). *Journal of Medical Entomology, 19*, 489−535.

McKelvey, J. J., Eldridge, B. F., & Maramorosch, K. (Eds.). (1981). *Vectors of disease agents. Interactions with plants, animals, and man*. New York: Praeger, 243 p.

McKiel, J. A., Hall, R. R., & Newhouse, V. F. (1966). Viruses of the California encephalitis complex in indicator rabbits. *The American Journal of Tropical Medicine and Hygiene, 15*, 98−102.

Means, R. G. (1979). *Mosquitoes of New York. Part I. The genus Aedes Meigen with identification keys to genera of Culicidae, 221 pp. Part II. Genera of Culicidae other than Aedes occurring in New York*. Albany: Univ. State of New York, State Educ. Dept., State Sci. Serv., New York State Museum, 180 p.

Meegan, J. M., & Bailey, C. L. (1988). Rift valley fever. In T. P. Monath (Ed.), *The Arboviruses: Epidemiology an ecology* (Vol. 4, pp. 51−76). Boca Raton, Florida: CRC Press.

Meola, R., & Readio, J. (1988). Juvenile hormone regulation of biting behavior and egg development in mosquitoes. *Advances in Disease Vector Research, 5*, 1−24.

Merritt, R. W., Dadd, R. H., & Walker, E. D. (1992). Feeding behavior, natural food, and nutritional relationships of larval mosquitoes. *Annual Review of Entomology, 37*, 349−376.

Minar, J. (1991). Family Culicidae. In A. Soos, & L. Papp (Eds.), *Catalogue of Palearctic Diptera: Psychodidae—Chironomidae* (Vol. 2, pp. 73−113). Amsterdam: Elsevier.

Mitchell, C. J. (1977). Arthropod-borne encephalitis viruses and water resource developments. *Cahiers Office de la Recherche Scientifique et Technique Outre-Mer Sèrie Entomologie Mèdicale et Parasitologie, 15*, 241−250.

Mitchell, C. J. (1983). Mosquito vector competence and arboviruses. *Current Topics in Vector Research, 1*, 63−92.

Molineaux, L., & Gramiccia, G. (1980). *The Garki project*. Geneva: World Health Organization, 311 p.

Monath, T. P. (Ed.). (1980). *St. Louis encephalitis*. Washington, DC: Amer. Pub. Hlth. Assoc.

Monath, T. P. (1988). Yellow fever. In T. P. Monath (Ed.), *The Arboviruses: Epidemiology and ecology* (Vol. 3, pp. 139−231). Boca Raton, FL: CRC Press.

Monath, T. P., & Heinz, F. X. (1996). Flaviviruses. In B. N. Fields, D. M. Knipe, P. M. Howley, et al. (Eds.), *Fields virology* (3rd ed., pp. 961−1034). Philadelphia: Lippincott-Raven Publishers.

Moore, C. G., & Gage, K. L. (1996). Collection methods for vector surveillance. In B. J. Beaty, & W. C. Marquardt (Eds.), *Biology of disease vectors* (pp. 471−491). Niwot, Colorado: University Press of Colorado.

Moore, C. G., McLean, R. G., Mitchell, C. J., Nasci, R. S., Tsai, T. F., Calisher, C. H., et al. (1993). *Guidelines for arbovirus surveillance in the United States*. Fort Collins, Colorado: Centers for Disease Control and Prevention, U. S. Dept. of Health and Human Services.

Morens, D. M., & Fauci, A. S. (2014). Chikungunya at the door — déjà vu all over again? *New England Journal of Medicine, 371*, 885−887.

Morris, C. D. (1988). Eastern equine encephalomyelitis. In T. P. Monath (Ed.), *The Arboviruses: Epidemiology and ecology* (Vol. III, pp. 1–20). Boca Raton, Florida: CRC Press, Inc.

Morris, C. D., Baker, R. H., & Opp, W. R. (1992). *H. T. Evans Florida mosquito control handbook*. Florida Mosq. Control Assoc.

Muirhead-Thomson, R. C. (1951). *Mosquito behaviour in relation to mosquito transmission and control in the tropics*. London: Arnold, 219 p.

Muirhead-Thomson, R. C. (1968). *Ecology of insect vector populations*. New York: Academic Press, 174 p.

Muirhead-Thomson, R. C. (1982). *Behaviour patterns of blood-sucking flies*. Oxford: Pergamon Press, 224 p.

Nasci, R. S., & Miller, B. R. (1996). Culicine mosquitoes and the agents they transmit. In B. J. Beaty, & W. C. Marquardt (Eds.), *The biology of disease vectors* (pp. 85–97). Niwot: Univ. Press of Colorado.

Nayar, J. K. (1982). *Bionomics and physiology of Culex nigripalpus (Diptera: Culicidae) of Florida: An important vector of diseases*. Florida Agr. Exp. Sta., Bull. No. 827. 73 p.

Florida Agr. Exp. Sta., Bull. No. 852. 148 p. In Nayar, J. K. (Ed.), *Bionomics and physiology of* Aedes taeniorhynchus *and* Aedes sollicitans*, the salt marsh mosquitoes of Florida*.

Neafsey, D. E., Waterhouse, R. M., Abai, M. R., Aganezov, S. S., Alekseyev, M. A., et al. (2015). Mosquito genomics. Highly evolvable malaria vectors: The genomes of 16 *Anopheles* mosquitoes. *Science, 347*, 1258522. https://doi.org/10.1126/science.1258522.

Nedelman, J. (1990). Gametocytemia and infectiousness in falciparum malaria: Observations and models. *Advances in Disease Vector Research, 6*, 59–89.

Nyasembe, V. O., & Torto, B. (2013). Volatile phytochemicals as mosquito semiochemicals. *Phytochemistry Letters, 8*, 196–201.

O'Meara, G. F. (1985). Ecology of autogeny in mosquitoes. In E. P. Lounibos, J. R. Rey, & J. H. Frank (Eds.), *Ecology of mosquitoes: Proceedings of a Workshop* (pp. 459–471). Vero Beach: Florida Med. Entomol. Lab.

Onori, E., & Grab, B. (1980). Indicators for the forecasting of malaria epidemics. *Bulletin World Health Organization, 58*, 91–98.

Ottesen, E. A., & Ramachandran, C. P. (1995). Lymphatic filariasis infection and disease: Control strategies. *Parasitology Today, 11*, 129–131.

Owen, W. B., & Gerhardt, R. W. (1957). *The mosquitoes of Wyoming* (Vol. 21, pp. 71–141). Laramie, Wyoming: Univ. Wyoming Publ. (3).

Pampana, E. (1963). *A textbook of malaria eradication*. Oxford, UK: Oxford Univ. Press.

Pan American Health Organization. (1972). *Venezuelan encephalitis*. PAHO Scientific Publication No. 243, Washington, D.C.

Peters, W. (1985). The problem of drug resistance in malaria. *Parasitology, 90*, 705–715.

Pittaway, A. R. (1992). *Arthropods of medical and veterinary importance: A Checklist of preferred names and allied terms*. CAB International.

Powers, A. M., & Logue, C. H. (2007). Changing patterns of chikungunya virus: Re-emergence of a zoonotic arbovirus. *Journal of General Virology, 88*, 2363–2377.

Pratt, H. D., Barnes, R. C., & Littig, K. S. (1963). *Mosquitoes of public health importance and their control*. Publ. Health Service, Publ. No. 772, Washington, D.C. 64 p.

Rai, K. S. (1991). *Aedes albopictus* in the Americas. *Annual Review of Entomology, 36*, 459–484.

Rai, K. S. (1996). Genetic control of vectors. In B. J. Beaty, & W. C. Marquardt (Eds.), *Biology of disease vectors* (pp. 564–574). Niwot, Colorado: University Press of Colorado.

Raikhel, A. S. (1992). Vitellogenesis in mosquitoes. *Advances in Disease Vector Research, 9*, 1–39.

Rayner, J. C. (2015). *Plasmodium malariae* malaria: From monkey to man? *EBioMedicine, 2*, 1023–1024.

Reeves, W. C. (Ed.). (1990). *Epidemiology and control of mosquito-borne arboviruses in California, 1943-1987*. Sacramento: California Mosquito and Vector Control Association.

Reinert, J. F., Kaiser, P. E., & Seawright, J. A. (1997). Analysis of the *Anopheles (Anopheles) quadrimaculatus* complex of sibling species (Diptera: Culicidae) using morphological, cytological, molecular, genetic, biochemical, and ecological techniques in an integrated approach. *Journal of the American Mosquito Control Association, 13*(Suppl.), 1–102.

Reisen, W. K., & Monath, T. P. (1988). Western equine encephalomyelitis. In T. P. Monath (Ed.), *The Arboviruses: Epidemiology and ecology* (Vol. 4, pp. 89–137). Boca Raton, FL: CRC Press, Inc.

Rempel, J. G. (1950). A guide to the mosquito larvae of western Canada. *Canadian Journal of Research, Section D, Zoological Sciences, 28*, 207–248.

Rempel, J. G. (1953). The mosquitoes of Saskatchewan. *Canadian Journal of Research, Section D, Zoological Sciences, 31*, 433–509.

Restifo, R. A. (1982). *Illustrated key to the mosquitoes of Ohio [adapted from Stojanovich (1960, 1961)]*. Ohio Biol. Survey, Biol. Notes No. 17, Columbus, Ohio. 56 p.

Ribeiro, J. M. C. (1987). Role of saliva in blood-feeding by arthropods. *Annual Review of Entomology, 32*, 463–478.

Ross, E. S., & Roberts, H. R. (1943). *Mosquito Atlas. Part I: The Nearctic Anopheles, important malaria vectors of the Americas and Aedes aegypti, Culex quinquefasciatus. 44 p. Part II: Eighteen old world anophelines important to malaria*, 44 p. Philadelphia: Am. Entomol. Soc., Acad. Nat. Sci.

Ross, H. H. (1947). The mosquitoes of Illinois (Diptera, Culicidae). In *Bull. Illinois Nat. Hist.Surv* (Vol. 24, pp. 1–96).

Ross, H. H., & Horsfall, W. R. (1965). A synopsis of the mosquitoes of Illinois (Diptera: Culicidae). In *Ill. Nat. Hist. Surv., Biol. Notes No. 52*, 50 p.

Ross, R. (1911). *The prevention of malaria*. London: Murray.

Rozeboom, L. E. (1942). The mosquitoes of Oklahoma. In *Tech. Bull. No, T-16*. Okla. Agric. Expt. Sta., 56 p.

Russell, R. C. (2002). Ross river virus: Ecology and distribution. *Annual Review of Entomology, 47*, 1–31.

Russell, P. F. (1955). *Man's mastery of malaria*. Oxford: Oxford Univ. Press, 308 p.

Russell, P. F., Rozeboom, L. E., & Stone, A. (1943). *Keys to the anopheline mosquitoes of the world, with notes on their identification, distribution, biology, and relation to malaria*. Philadelphia: Am. Entomol. Soc., Acad. Nat. Sci., 152 p.

Rutschky, C. W., Mooney, T. C., Jr., & Vanderberg, J. P. (1958). *Mosquitoes of Pennsylvania. An illustrated key to species with accompanying notes on biology and control*. Penn. State Univ., Agric. Expt. Sta. Bull. No. 630. Pennsylvania: University Park, 26 p.

Sasa, M. (1976). *Human filariasis: A global survey of epidemiology and control*. Tokyo, Japan: Tokyo University Press.

Service, M. W. (Ed.). (1988). *Biosystematics of haematophagous insects*. Oxford: Clarendon Press, 363 p.

Service, M. W. (1989). *Demography and vector-borne diseases*. Boca Raton, FL: CRC Press, 402 p.

Service, M. W. (1990). *Handbook of the afrotropical toxorhynchitine and culicine mosquitoes, excepting* Aedes and Culex. London: British Museum (Natural History), 207 p.

Service, M. W. (1993a). *Mosquito Ecology: Field sampling methods* (2nd ed.). New York: Elsevier Applied Science, 988 p.

Service, M. W. (1993b). Mosquitoes (Culicidae). In R. P. Lane, & R. W. Crosskey (Eds.), *Medical insects and arachnids* (pp. 120–240). New York: Chapman & Hall.

Service, M. W. (1997). Mosquito (Diptera: Culicidae) dispersal—the long and short of it. *Journal of Medical Entomology, 34*, 579–588.

Severson, D. W., & Behura, S. K. (2012). Mosquito genomics: Progress and challenges. *Annual Review of Entomology, 57*, 143–166.

Shuaib, W., Hashim Stanazai, H., Abazid, A. G., & Mattar, A. A. (2016). Re-emergence of Zika virus: A review on pathogenesis, clinical manifestations, diagnosis, treatment, and prevention. *American Journal of Medicine, 129*, 879.e7–879.e12.

Silver, J. B. (2008). *Mosquito ecology: Field sampling methods* (3rd ed.). Springer, 1494 p.

Siverly, R. E. (1972). *Mosquitoes of Indiana*. Indianapolis: Indiana State Board Health, 126 p.

Smallegange, R. C., & Takken, W. (2010). Host-seeking behaviour of mosquitoes: Responses to olfactory stimuli in the laboratory. In W. Takken, & B. G. J. Knols (Eds.), *Ecology and control of vector-borne diseases* (Vol. 2, pp. 143–180). The Netherlands: Wageningen Academic Publishers.

Smart, J. (1948). *Insects of medical importance*. London: British Museum (Natural History).

Snodgrass, R. E. (1959). *The anatomical life of the mosquito*. Smithsonian Misc. Publ., Vol. 139, No. 8. Baltimore, Maryland: Baltimore Press, 87 p.

Snow, K. R. (1990). Mosquitoes. In *Naturalists' handbooks* (Vol. 14). Slough, United Kingdom: Richmond Publ., 66 p.

Sokolova, M. I. (1994). A redescription of the morphology of mosquito (Diptera: Culicidae) ovarioles during vitellogenesis. *Bulletin of the Society for Vector Ecology, 19*, 53–68.

Soper, F. L. (1963). The elimination of urban yellow fever in the Americas through the eradication of *Aedes aegypti*. *American Journal of Public Health, 53*, 7–16.

Soper, F. L., & Wilson, D. B. (1943). *Anopheles gambiae in Brazil 1933-1940*. New York: Rockefeller Foundation.

Stage, H. H., Gjullin, C. M., & Yates, W. W. (1952). Mosquitoes of the northwestern states. In *U. S. Dept. Agric. Handbook* (Vol. 46). Washington, DC. 95 p.

Steffan, W. A., & Evenhuis, N. L. (1981). Biology of *Toxorhynchites*. *Annual Review of Entomology, 26*, 159–181.

Stojanovich, C. J. (1960). *Illustrated key to common mosquitoes of southeastern United States*. Atlanta, Georgia: Cullom & Ghertner Co., 36 p.

Stojanovich, C. J. (1961). *Illustrated key to common mosquitoes of northeastern North America*. Atlanta, Georgia: Emory Univ. Branch, Cullom & Ghertner Co., 49 p.

Stone, A. (1981). Culicidae. In J. F. McAlpine, et al. (Eds.), *Manual of nearctic Diptera* (Vol. 1, pp. 341–350). Chapt. 25.

Stone, A., & Delfinado, M. D. (1973). Family Culicidae. In M. D. Delfinado, & D. E. Hardy (Eds.), *A catalog of the Diptera of the Oriental region. Suborder Nematocera* (Vol. 1, pp. 266–343). Honolulu: Univ. Press of Hawaii.

Strickland, G. T. (Ed.). (1991). *Hunter's tropical medicine* (7th ed.). Philadelphia: W. B. Saunders Co.

Tabachnick, W. J. (1994). Genetics of insect vector competence for arboviruses. *Advances in Disease Vector Research, 10*, 93–108.

Takken, W., & Verhulst, N. O. (2013). Host preferences of blood-feeding mosquitoes. *Annual Review of Entomology, 58*, 433–453.

Tate, H. D., & Gates, D. B. (1944). *The mosquitoes of Nebraska*. Agric. Expt. Sta. Res. Bull. No. 133. Univ. Nebraska, 27 p.

Telford, S. R., Jr. (1994). Plasmodia of reptiles. In J. P. Kreier (Ed.), *Parasitic protozoa* (2nd ed., Vol. 7, pp. 1–72). New York: Academic Press.

Tempelis, C. H. (1975). Host-feeding patterns of mosquitoes, with a review of advances in analysis of blood meals by serology. *Journal of Medical Entomology, 11*, 635–653.

Tsai, T. F., & Mitchell, C. J. (1988). St. Louis encephalitis. In T. P. Monath (Ed.), *The Arboviruses: Epidemiology and ecology* (Vol. 4, pp. 113–143). Boca Raton, FL: CRC Press.

Tulloch, G. S. (1939). A key to the mosquitoes of Massachusetts. *Psyche, 46*, 113–136.

Turell, M. J. (1988). Horizontal and vertical transmission of viruses by insect and tick vectors. In T. P. Monath (Ed.), *The Arboviruses: Epidemiology and ecology* (Vol. I, pp. 127–152). Boca Raton, FL: CRC Press.

United States Department of Agriculture, Animal and Plant Health Inspection Service. (1973). *The origin and spread of venezuelan equine encephalomyelitis* (pp. 91–100). APHIS.

United States Public Health Service and Tennessee Valley Authority. (1947). *Malaria control on impounded water*. Washington, D.C: U.S. Government Printing Office.

Van Dine, D. L. (1922). *Impounding water in a Bayou to control breeding of malaria mosquitoes*. Bull. No. 1098. Washington, D.C: USDA, U.S. Govt. Printing Office, 22 p.

Van Riper, C., III, et al. (1994). Plasmodia of birds. In J. P. Kreier (Ed.), *Parasitic protozoa* (2nd ed., Vol. 7, pp. 73–140). New York: Academic Press.

Wahid, B., Amjad Ali, A., Rafique, S., & Muhammad Idrees, M. (2017). Global expansion of Chikungunya virus: Mapping the 64-year history. *International Journal of Infectious Diseases, 58*, 69–76.

Wallis, R. C. (1960). *Mosquitoes in Connecticut*. Conn. Agric. Expt. Sta., Bull. No. 632. New Haven, Connecticut, 30 p.

Walton, T. E., & Grayson, M. A. (1988). Venezuelan equine encephalomyelitis. In T. P. Monath (Ed.), *The Arboviruses: Epidemiology and ecology* (Vol. 4, pp. 203–231). Boca Raton, FL: CRC Press.

Ward, R. A. (1984). Second supplement to "a catalog of the mosquitoes of the world (Diptera: Culicidae)." *Mosquito Systematics, 16*, 227–270.

Ward, R. A. (1992). Third supplement to "a catalog of the mosquitoes of the world (Diptera: Culicidae)." *Mosquito Systematics, 24*, 177–230.

Ward, R. A., & Darsie, R. F. (1982). Corrections and additions to the publication, identification and geographical distribution of the mosquitoes of North America north of Mexico. *Mosquito Systematics, 14*, 209–219.

Washino, R. K., & Tempelis, C. H. (1983). Mosquito host bloodmeal identification: Methodology and data analysis. *Annual Review of Entomology, 28*, 179–201.

Watts, D. M., Pantuwatana, S., DeFoliart, G. R., Yuill, T. M., & Thompson, W. H. (1973). Transovarial transmission of LaCrosse virus (*California encephalitis* group) in the mosquito, *Aedes triseriatus*. *Science, 182*, 1140–1141.

Weaver, S. C., Ferro, C., Barrera, R., Boshell, J., & Navarro, J.-C. (2004). Venezuelan equine encephalitis. *Annual Review of Entomology, 49*, 141–174.

Weaver, S. C., Federico Costa, F., Garcia-Blanco, M. A., Ko, A. I., Ribeiro, G. S., Saade, G., et al. (2016). Zika virus: History, emergence, biology, and prospects for control. *Antiviral Research, 130*, 69–80.

Wernsdorfer, W. H., & McGregor, I. (Eds.). (1988). *Malaria: Principles and practice of malariology, 2 vol*. New York: Churchill Livingstone, 1818 p.

White, G. B. (1980). Family Culicidae. In R. W. Crosskey (Ed.), *Catalogue of the Diptera of the Afrotropical region* (pp. 114–148). London: British Museum (Natural History).

Wilkerson, R. C., & Strickman, D. (1990). Illustrated key of the female anopheline mosquitoes of Central America and Mexico. *Journal of the American Mosquito Control Association, 6*, 7–34.

Wilkerson, R. C., Linton, Y. M., Fonseca, D. M., Schultz, T. R., Price, D. C., & Strickman, D. A. (2015). Making mosquito taxonomy useful. A stable classification of the Aedini that balances utility with current knowledge of evolutionary relationships. *PLoS One*. https://doi.org/10.1371/journal.pone.0133602.

Wood, R. J. (2005). Genetic control of vectors. In W. C. Marquardt (Ed.), *Biology of disease vectors* (2nd ed., pp. 661–669). New York: Elsevier Academic Press.

Wood, D. M., & Borkent, A. (1989). Phylogeny and classification of the Nematocera. In J. F. McAlpine (Ed.), *Vol. 3. Manual of Nearctic Diptera* (pp. 1333–1370). Res. Branch, Agriculture Canada, Monogr. No. 32.

Wood, D. M., Dang, P. T., & Ellis, R. A. (1979). *The insects and arachnids of Canada. Part 6. The mosquitoes of Canada. Diptera: Culicidae*. Hull, Quebec: Canad. Gov. Publ. Centre, 390 pp.

Woodring, J. L., & Davidson, E. W. (1996). Biological control of mosquitoes. In B. J. Beaty, & W. C. Marquardt (Eds.), *The biology of disease vectors* (pp. 530–548). Niwot: Univ. Press of Colorado.

World Health Organization. (1973). *Manual on larval control operations in malaria programmes*. Offset Publication No. 1. Geneva, Switzerland: World Health Organization.

World Health Organization. (1975). *Manual on practical Entomology in malaria*. Parts I (160 pp.) and II (191 pp.). Offset Publication No. 13. Geneva, Switzerland: World Health Organization.

World Health Organization. (1982). *Manual on environmental management for mosquito control*. Offset Publication No. 66. Geneva, Switzerland: World Health Organization, 283 p.

World Health Organization, Vector Biology and Control Division. (1989). *Geographical distribution of arthropod-borne diseases and their principal vectors*. World Health Organization/VBC Publication 89.967, 134 p.

World Health Organization. (1991). *Prospects for malaria control by genetic manipulation of its vectors*. Unpublished document TDR/BCV/MALENT/91.3. Geneva, Switzerland: World Health Organization.

World Health Organization. (2017). *World malaria report. Licence: CC by-NC-SA 3.0 IGO*, 159 p. www.who.int/malaria

Wright, J. W., & Pal, R. (Eds.). (1967). *Genetics of insect vectors of disease*. New York: Elsevier, 794 p.

Yamaguti, S., & LaCasse, W. J. (1951). *"Mosquito fauna of North America," parts I-V*. Off. Surg., Hq., Japan Logistical Command, APO 343.

Yuan, L., Huang, X.-Y., Liu, Z.-Y., Zhang, F., Zhu, X.-L., et al. (2017). A single mutation in the prM protein of Zika virus contributes to fetal microcephaly. *Science, 358*, 933–936.

This page intentionally left blank

Chapter 16

Horse Flies and Deer Flies (Tabanidae)

Bradley A. Mullens
Department of Entomology, University of California, Riverside, CA, United States

Because of their fairly large size, striking appearance, and diurnal biting habits, horse flies and deer flies (Fig. 16.1) are familiar to most people who have livestock or engage in outdoor activities. Diversity within the family is greatest in the tropics, but moist temperate regions typically have a rich fauna as well. Tabanids are present on every continent except Antarctica and have managed to colonize remote islands such as the Galápagos and the Melanesian Archipelago. Large seasonal populations of some species occur as far as 60°N latitude, but they disappear above the tree line.

The eyes of many species, when alive, are brilliantly patterned with shades of green, yellow, orange, and violet. Some species with strikingly green eyes are commonly called **greenheads**, while some others are called yellow flies due to their yellow bodies. The blue-tail fly of American folk music probably was *Tabanus atratus*, a large black species with a blue cast to the abdomen. Some common names reflect times or places where these biting flies are found (e.g., **March fly**, **May fly**, and **mango fly**), while the sources of some colloquial names are obscure (e.g., **cleg** and **whamefly**). Other common names include **breezefly**, **bulldog**, and **gadfly**, the latter two reflecting the persistent annoyance of tabanids in seeking a bloodmeal.

The term **horse fly** is applied to relatively large species of tabanids, typically 10−30 mm in length. They can be a serious nuisance to livestock and can mechanically transmit several significant animal pathogens, including those that cause surra, anaplasmosis, and equine infectious anemia. Even moderate numbers of flies feeding on livestock can result in significant production losses. A few horse flies readily bite people, examples being the infamous greenheads (*Tabanus nigrovittatus* and *T. simulans*) in the coastal regions of the eastern United States.

The smaller tabanid species called **deer flies** typically are 6−11 mm long. In contrast to horse flies, they frequently attack humans. Fortunately, there are just a few human diseases known to be associated with deer flies. The most important tabanid-transmitted human diseases are loiasis and tularemia. Outdoor activity and tourism suffer in areas where tabanid populations are high, although such losses are hard to quantify.

TAXONOMY

Tabanidae have among the most described species of any family of blood-feeding Diptera. It contains approximately 4,455 species and subspecies in 144 genera worldwide, with maximum diversity in the Neotropics (Baldacchino et al., 2014; Morita et al., 2016). Of these, 335 species in 25 genera are found in the Nearctic Region (Burger, 1995). While the temperate fauna is well known, the tropical fauna has been less studied; this is particularly true for the immature stages. Most pest species in North America are members of the genera *Chrysops*, *Hybomitra*, and *Tabanus*. The oldest blood-feeding Tabanomorpha, which include the Tabanidae and a few related families, probably evolved about 160 million years ago. However, several important hematophagous genera (*Chrysops, Hybomitra, Haematopota*) apparently have radiated mostly in the past 20 million years. With more than 1,350 species, *Tabanus* is one of the largest genera of Diptera, but it is not monophyletic and is likely to undergo some revision.

The Tabanidae are divided into three subfamilies (Mackerras, 1954; Fairchild, 1969) (Table 16.1), although there is evidence the Chrysopsinae may need redefinition (Morita et al., 2016). The subfamily Pangoniinae is regarded as ancestral, containing fascinating but poorly known genera such as *Stonemyia* and *Goniops*, many of which are not known to feed on blood. Most of the economically important tabanids are members of the two other subfamilies. Tabanids in the subfamily Chrysopsinae are called **deer flies**; nearly all are members of the genus *Chrysops*, which includes more than 80 Nearctic species. The term "deer fly" also is applied to members of the genus *Silvius*, a

Medical and Veterinary Entomology. https://doi.org/10.1016/B978-0-12-814043-7.00016-9

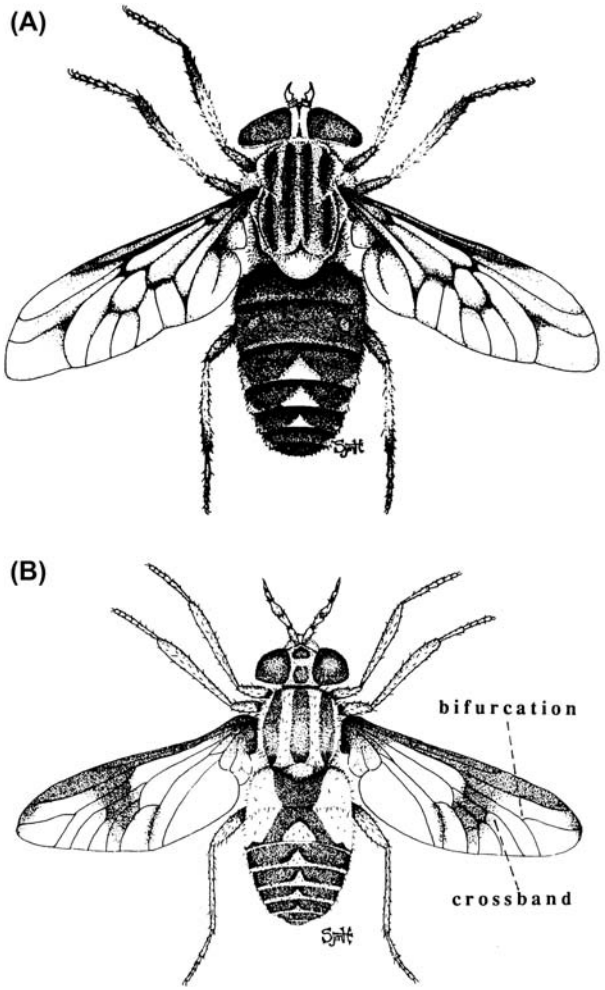


FIGURE 16.1 Adult tabanid flies, females, dorsal view. (A) Horse fly, *Tabanus trimaculatus*; (B) Deer fly, *Chrysops callidus*. Note distinctive wing venation, including bifurcation of vein near wing tip, and the darkened crossband in deer flies. *Original by Susan Milne Hope.*

TABLE 16.1 Subfamilies, Tribes, and Selected Genera of Tabanidae in North America

Taxon	No. of Species
Subfamily Pangoniinae	
Tribe Pangoniini	
Genus *Apatolestes*	13
Genus *Stoneymyia*	6
Tribe Scionini	
Genus *Goniops*	1
Subfamily Chrysopsinae	
Tribe Bouvieromyiini	
Genus *Mercomyia*	2
Tribe Chrysopsini	
Genus *Silvius*	12
Genus *Chrysops*	83
Subfamily Tabaninae	
Tribe Diachlorini	
Genus *Diachlorus*	1
Genus *Chlorotabanus*	1
Genus *Leucotabanus*	2
Tribe Haematopotini	
Genus *Haematopota*	5
Genus *Tabanus*	107
Genus *Atylotus*	14
Genus *Hybomitra*	55

The largest genera in terms of numbers of species are *Chrysops, Tabanus,* and *Hybomitra.*

few species of which can be quite pestiferous on humans and animals in the western United States.

Members of the Tabaninae are the most evolutionarily derived. This subfamily includes the **horse flies**, represented by *Tabanus*, which has 107 Nearctic species, and *Hybomitra*, with 55 Nearctic species. Species of *Haematopota*, together with *Tabanus* and *Hybomitra*, are important pests in the Old World. Only five species of *Haematopota* occur in the Nearctic region, where *H. americana* is the only species known to be a pest of mammals.

Burger (1995) compiled a complete catalog of species of Nearctic Tabanidae. Pechuman and Teskey (1981) provided generic keys to larvae, pupae, and adults of Nearctic tabanids. There are several regional keys for identification of adults of North American tabanids at the species level: Florida (Jones and Anthony, 1964), California (Middlekauf and Lane, 1980), New York (Pechuman, 1981), Illinois (Pechuman et al., 1983), Tennessee (Goodwin et al., 1985), Texas (Goodwin and Drees, 1996), and Canada and Alaska (Teskey, 1990). Most of these works include valuable information on biology and ecology. Immatures of Nearctic tabanids are more difficult to identify than adults. Many North American species remain undescribed, and the immature stages are less likely to be encountered by the casual collector. Immatures of some species can be identified using keys or references found in Burger (1977), Pechuman et al. (1983), Goodwin et al. (1985), and Teskey (1990).

Taxonomic references for other regions include those for Europe (Chvala et al., 1972), Neotropics (Fairchild, 1986; Coscaron and Papavero, 1993; Fairchild and Burger, 1994), Australia (MacKerras, 1954), Australasia (Burger and Chainey, 2000), Mali (Goodwin, 1982), Ethiopian region (Oldroyd, 1954–1957), the former Soviet Union

(Olsufiev, 1977), and Japan (Takahashi, 1962; Hayakawa, 1985). Immature stages of Palearctic Tabanidae are treated by Andreeva (1990).

MORPHOLOGY

Tabanid larvae (Fig. 16.2) are spindle-shaped (fusiform) and generally whitish in color, although some are shades of brown or green. Mature larvae of common species typically measure 15−30 mm in length, but some larger tabanid larvae may be as long as 60 mm. The head capsule is incomplete and partially sclerotized. The mandibles are strong, parallel, and ventrally curved and are used to capture and subdue prey. The larval cuticle has distinctive longitudinal striations and often exhibits species-specific pubescence patterns that give some tabanid larvae a mottled appearance. Abdominal segments have lateral and ventral **pseudopods** for locomotion (three pairs in *Chrysops* spp., four pairs in Tabaninae). Larvae of the more terrestrial species tend to be relatively stocky with short pseudopods. Species adapted to a fully aquatic existence in streams (e.g., *Tabanus fairchildi*) have elongated pseudopods armed with cuticular, recurved, distal hooks. Semiaquatic larvae, represented by the majority of tabanid species, have intermediate characters.

Located in the anterodorsal portion of the anal segment of larvae is a pear-shaped vesicle called **Graber's organ**. It is readily seen through the cuticle only in tabanid larvae; the number of black bodies within it increases for each larval instar. Its function is unknown, although it also exists in some adult tabanids. Two main tracheal trunks run the length of the body, terminating in a dorsally directed respiratory siphon. A terminal spine is present on the siphon of some species.

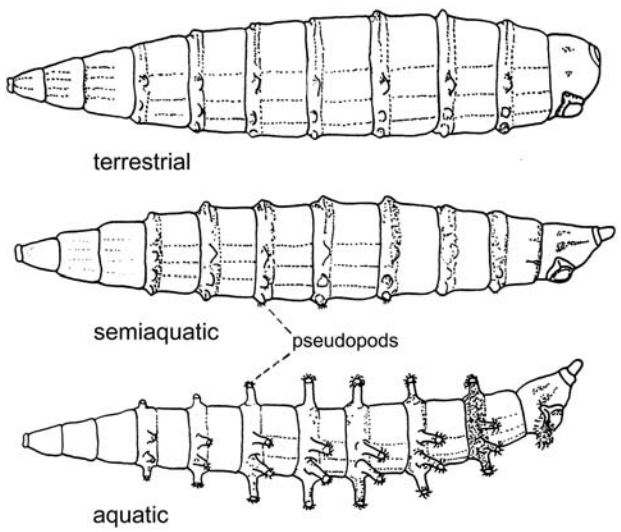


FIGURE 16.2 Three basic morphotypes of tabanid larvae, typical of terrestrial, semiaquatic, and aquatic species, respectively. Locomotory appendages, or pseudopods, are particularly well developed in aquatic larvae. *Redrawn from Andreeva, 1989.*

Tabanid pupae are usually tan or brown, with the eyes, legs, and wing pads visible externally. A fringe of spines on the posterior margin of many abdominal segments and a starlike series of three or four pairs of caudal projections called pupal asters are useful in identification.

Tabanid adults are stout-bodied flies. They generally can be distinguished as horse flies or deer flies based on several morphological characters (Table 16.2). The antennae are prominent and extend anteriorly. The flagellum, with four to eight flagellomeres, usually is enlarged at the base in Tabaninae but only slightly enlarged in *Chrysops* species (Fig. 16.3). The eyes often consist of large ommatidial facets dorsally and smaller facets ventrally. This arrangement is believed to enhance visual acuity in locating potential mates. The male has **holoptic eyes**, which occupy most of the head, touching each other medially. The female has **dichoptic eyes** that are smaller than those of the male and are separated by the frons. The frons of most species is covered by very fine pubescence. Slightly raised, sometimes bare areas of cuticle, called the median callus and basal callus, aid identification.

The remarkable color patterns of the adult eyes are very distinctive and beautiful in many species and sometimes are useful taxonomically. While eye patterns and colors, unfortunately, disappear when a specimen dries, the basic pattern often can be restored by rehydration of the specimen. The Pangoniinae and Chrysopsinae possess well-developed ocelli at the vertex of the frons, whereas the ocelli of Tabaninae are vestigial or absent. *Hybomitra* spp. usually exhibit a raised, denuded ocellar tubercle, which is lacking in *Tabanus* spp.

The maxillary palps are two-segmented and enlarged at the base of the apical palpomere. The proboscis is stout and includes toothed, bladelike mandibles and maxillary laciniae used to lacerate the skin and capillary beds during blood-feeding. The female hypopharynx is rigid, with the salivary duct opening at the tip to introduce saliva into the feeding wound. Blood is drawn up between the labellar lobes into the food canal between the labrum and hypopharynx. This feeding method is known as pool feeding, or telmophagy. Males, which do not feed on blood, lack mandibles, recurved teeth on the laciniae, and a rigid hypopharynx. Like most adults of blood-feeding Diptera, both sexes of tabanids also feed on sugar in the form of nectar and honeydew. It is the only food for the males.

The necessity of using both types of food sources is nicely illustrated by the African tabanid genus *Philoliche*. The mouthparts have evolved spectacularly to feed on nectar from flowers with very deep corollas (Fig. 16.4). The nonretractable labium thus may be up to three times the body length and folds backward parallel to the fly body (and beyond) except when it is being used for nectar feeding. Even the stiffer, well-armed, proximal piercing mouthparts of female *Philoliche* are long by comparison to most tabanids. The nectar-feeding portion again folds back and is out of the way while the piercing structures slice the

TABLE 16.2 Morphological Characters Used to Differentiate Adult Horse Flies and Deer Flies

Character	Horse Flies (e.g., *Tabanus*)	Deer Flies (e.g., *Chrysops*)
Body length	10–30 mm	6–11 mm
Antennae	Short, base of flagellum greatly enlarged	Long, base of flagellum not greatly enlarged
Ocelli	Vestigial or lacking	Present
Wings	Clear, uniformly cloudy, or spotted	Distinctly banded
Apical spurs on hind tibiae	Lacking	Present

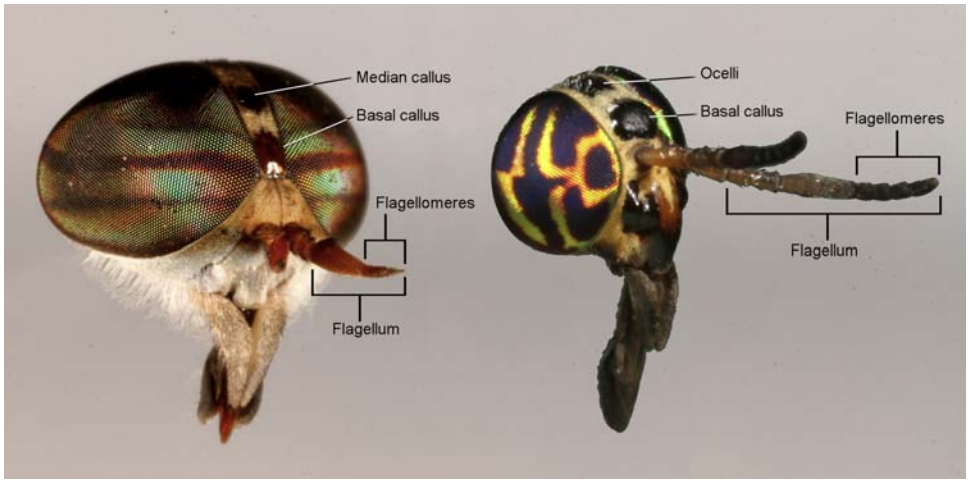


FIGURE 16.3 Morphology of head and antennae of tabanid flies, noting important taxonomic characters. (left) Horse fly, *Tabanus* sp. (Right) Deer fly, *Chrysops* sp.; note the elongate flagellum (terminal segment of the antenna) of *Chrysops*, with multiple flagellomeres, or pseudosegments. *Photograph by Nathan D. Burkett-Cadena.*

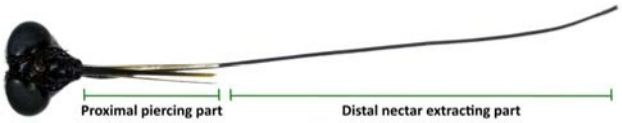


FIGURE 16.4 Remarkably elongated mouthparts in species of *Philoliche* in Africa, adapted for feeding on both blood and sugar. The stiff basal structures, composed of the labrum–epipharynx and well-developed mandibles, are used for blood-feeding, whereas the extremely long labium is extended deep into corollas of flowers for imbibing nectar; the labium is folded back during flight and blood-feeding. *Reproduced with permission of Florian Karolyi.*

skin. They are able to ingest the blood from the tip of the piercing portion.

The thorax is stout with a prominent notopleural lobe and strong flight muscles. Legs are also stout with fairly prominent tibial spurs. Apical spurs are present on the hind tibia of the Pangoniinae and Chrysopsinae but are lacking in the Tabaninae. The wing venation is consistent within the Tabanidae; a key feature is the widely divergent R4 and R5 veins, which fork and enclose the apex of the wing. Wing membranes are clear in some species and variously darkened in others, providing useful taxonomic characters, particularly for *Chrysops* spp. Wings of *Haematopota* species (clegs) are mottled, while those of most other Tabaninae are not. The abdomen of tabanids is as wide as the thorax, slightly compressed dorsoventrally, and often distinctly colored or patterned.

Internally, tabanids have a large crop for water and sugar storage. Blood is directed to the midgut. As with many other blood-feeders, tabanids can rapidly eliminate excess fluids from the bloodmeal via the anus. Genitalia are fairly simple structurally in both sexes and are of little use in routine species identifications.

LIFE HISTORY

Tabanid **eggs** are 1–3 mm long and are deposited in masses (Fig. 16.5). The female usually lays 100–800 eggs in a single mass, the numbers varying substantially with the species and size of the bloodmeal. Some species lay several smaller batches of eggs, particularly in captivity; *Atylotus thoracicus* has been observed to deposit eggs singly on sphagnum moss in the laboratory. Eggs are white when laid

FIGURE 16.5 Female horse fly, *Tabanus imitans*, ovipositing on plant stem. *Photograph by Sturgis McKeever.*

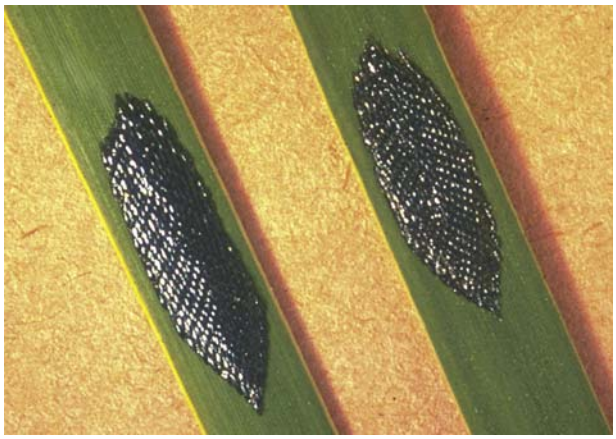


FIGURE 16.6 Two egg masses of *Chrysops callidus*, deposited on vegetation. Note single layer of eggs typical of most deer flies. *Photograph by Laverne L. Pechuman.*

FIGURE 16.7 Egg mass of *Chrysops cincticornis*. Note multiple layers of eggs typical of certain deer flies and most horse flies. *Photograph by Elton J. Hansens.*

but darken to grey, brown, or black within several hours. Egg masses most often are found on leaves or stems of emergent vegetation at the edges of ponds (lentic habitats) or streams (lotic habitats) or on leaves or bark of trees overhanging the water. The larvae of Nearctic spp. of *Chrysops* have been categorized as 65% lentic and 18% lotic, with 13% of the species being found in both lentic and lotic habitats. Stream-dwelling species often deposit their eggs above waterline on stones in the stream where water flow is moderate. Some species can be terrestrial and may be found in fairly dry soil (e.g., *Tabanus abactor*, *T. sulcifrons*, *T. subsimilis*). Terrestrial species usually lay their eggs on vegetation or in leaf litter. *Apatolestes actites* is known to oviposit in crustacean burrows in beach habitats.

Many *Chrysops* spp. deposit eggs in a single layer, such as *C. callidus* (Fig. 16.6), whereas others deposit eggs in tiers, such as *C. cincticornis* (Fig. 16.7). Species of *Tabanus* and *Hybomitra* lay their eggs in tiers, commonly three or four tiers high. Such masses taper in pyramid fashion from base to apex. The exact shape of the egg mass often reflects the oviposition substrate; for example, the same species may deposit an elongate egg mass on a grass stem or a broader one on a deciduous leaf. While the eggs often are easily seen, it is uncommon to observe females in the act of oviposition. Recent DNA fingerprinting efforts have allowed more species-specific descriptions of egg mass morphology and location. Females of an unusual and primitive tabanid, *Goniops chrysocoma*, lay eggs on the underside of a leaf and secure themselves above the mass by using the tarsal claws. They remain with the mass until it hatches, buzzing noisily if disturbed. The female dies soon after the eggs hatch.

Embryogenesis typically requires 5–12 days at temperatures of 21–24°C, and is both temperature and species dependent. Egg hatch can occur within 2–3 days at temperatures of 30–35°C. First-instar larvae are equipped with an **egg burster**, a projection on the head capsule with which they split the chorion and then drop to the water or moist substrate below. They molt once, apparently without feeding, before beginning to move about in the substrate.

Tabanid larvae are found in a wide variety of aquatic and semiaquatic habitats. These include mud and water-saturated vegetation in marshes or near pond or creek margins, under stones in and along streams, and in

terrestrial habitats such as under forest litter. Some species are common in a variety of semiaquatic habitats (e.g., *Tabanus punctifer*). Others are very specific; larvae of *Leucotabanus annulatus*, for example, are found only in rotten tree stumps. Tabanid larvae are general predators that feed on a variety of invertebrates such as larvae of chironomid midges and crane flies, or annelids. Horse-fly larvae also are cannibalistic, a factor that influences their densities and distribution and complicates efforts to rear them in the laboratory. It is unusual to find high densities of tabanine larvae in nature. Nonetheless, densities of larvae in flooded hardwood forest in Louisiana (USA) have been estimated at about 10 per m^2, leading to high adult populations. In contrast to tabanine larvae, *Chrysops* larvae apparently are not as cannibalistic and may be found at high field densities. *Chrysops* larvae probably are predaceous and feed primarily on invertebrates, although some authors have speculated that they feed on detritus.

Tabanids undergo 6−13 larval molts and overwinter as larvae. Most temperate species have one generation per year (univoltine), whereas others may produce two or more generations (multivoltine). Adult tabanid abundance from 1 year to the next probably reflects local weather patterns. These include generally negative impacts of high summer/fall air temperatures the prior year and positive effects of higher fall and spring precipitation, although species vary in their responses. Particularly large species, such as *Tabanus atratus* and *T. calens*, may spend 2 or 3 years as larvae. Development may be prolonged for 3 years or longer in very cold, seasonally dry, or otherwise unfavorable conditions. In the spring, the larvae leave the water-saturated soil to pupate above waterline. Pupal periods vary with species and temperature but typically last 4−21 days.

Most temperate species have very distinctive seasonal flight periods that vary little from year to year. In the eastern United States and Canada, for example, *Hybomitra lasiophthalma* is one of the first pestiferous horse flies to emerge. It begins activity as early as March in the southern United States or as late as June in southern Canada and is abundant for 3−6 weeks, and then adults disappear until the following spring. In contrast, *Tabanus subsimilis* has been collected in other parts of the United States from mid May through early October. Some tabanids can develop from egg to adult in as little as 6 weeks, and a few species have multiple broods within a season. A prolonged period of adult emergence, or the presence of unrecognized sibling species, can contribute to what appears to be a long adult flight period.

Many tabanids are **anautogenous** and require a single large bloodmeal in order to develop a batch of eggs. Bloodmeal size varies from 20 to 25 mg for many *Chrysops* spp. to almost 700 mg for *Tabanus atratus*. Other species, such as the salt marsh greenhead *T. nigrovittatus*, are obligately **autogenous** in the first gonotrophic cycle and must seek a bloodmeal for each subsequent cycle. Microsatellite studies of *T. nigrovittatus* indicated a high degree of local relatedness that perhaps results from autogenous adult oviposition near the emergence sites. Still others seem to be facultatively autogenous, probably reflecting genetic variability and the carry-over of available nutrients from the larval stage. Following a bloodmeal, egg development is believed to be typically 3−4 days. Time to oviposition may be several days longer for some species, especially under laboratory conditions where the full range of oviposition cues may be lacking. One California species, *Apatolestes actites*, apparently undergoes two autogenous gonotrophic cycles, a rarity among families of hematophagous Diptera.

BEHAVIOR AND ECOLOGY

The biology and behavior of tabanid larvae are generally poorly known. They are laborious to rear in the laboratory due to their long developmental times and predaceous and cannibalistic habits. No tabanid species has been successfully colonized. As soil dwellers they are difficult to observe and sample. Tabanid larvae are rarely free-swimming in nature, but some, such as *Tabanus punctifer*, are buoyant and can swim effectively by repeatedly flipping the rear half of the body and propelling themselves in short, gliding spurts.

On contacting a prey item, a tabanid larva will strike, often with an audible "click," seizing the prey with its mandibles. Tabanid larvae are capable of capturing prey larger than themselves, and prey struggles usually cease very quickly. A toxin likely is involved in prey capture, but this has not been conclusively demonstrated.

Prior to pupation, larvae generally seek out drier soil, such as above waterline at the edge of ponds or streams. Pupation occurs near the soil surface with the head end oriented upward. Larvae of a few species, including *T. atratus* in the New World and several other *Tabanus* spp. in the Old World, construct a mud cylinder above waterline. They spiral downward 5−13 cm to delineate the perimeter of the 3- to 9-cm diameter cylinder and then burrow into the center to form a pupation tunnel. This unusual behavior may preserve the structural integrity of the pupation tunnel and facilitate future emergence or yield a drier pupation site in periodically flooded habitats. Mortality during tabanid egg and larval stages is high. A production ratio of only three pupae per egg mass has been calculated for *Hybomitra bimaculata* in Swiss bog-meadow habitats.

The biology and behavior of tabanid adults are better understood than those of the immatures. The sex ratio at emergence is approximately 1:1, and emergence of males precedes that of females by 1 to a few days. An important activity for both sexes is **carbohydrate feeding**, which

provides energy for general body maintenance, flight, and mating. Sugars are obtained at floral or extrafloral nectaries or other natural plant-sugar deposits. *Tabanus nigrovittatus* and some *Chrysops* spp. adults are known to feed on honeydew from plant-sucking insects, such as aphids and scales. Males, in particular, engage in "dipping" behavior, touching the surface of pools of water with the mouthparts while in flight. This may serve to fill the crop with enough water to allow flies to regurgitate on honeydew deposits. The ingested sugars replenish energy reserves expended during daily flight and mating activities.

Tabanid **mating** occurs in flight, especially in the morning, but has never been induced in the laboratory. This is a key barrier to colonization. Most observations are of individuals or small groups of males (Tabaninae) hovering within 3 m of the ground along forest roads, ecotone areas, or above natural features (e.g., plant clumps) that serve as markers. Some species apparently hover above treetops or forest canopies as high as 90−100 m above ground level. Males of other species, such as *Chrysops fuliginosus* and *Hybomitra illota*, perch on vegetation and other objects. In both case, males detect and pursue passing females. They also chase conspecific males and other passing insects. Males of *Hybomitra hinei* have been observed to pursue 8-mm beads shot past them at speeds of 27−30 m/s.

Males of some species are thought to exhibit territoriality. However, what may appear to be agonistic interactions between males may actually be normal pursuit behaviors directed toward any appropriate-sized object moving through their response zone. Individual males do not necessarily use a particular site continuously over time. The occurrence of **male aggregations** at the tops of hills, known as "hilltopping" behavior, is common for some species. The larger eyes of males reflect a mating strategy dominated by visual cues. The larger, dorsal ommatidia may be more sensitive to ultraviolet light, allowing the male to detect a fast-moving female against the sky and the smaller, ventral ommatidia may be used to resolve visual details. While pheromones are suspected for a few tabanid species, this has yet to be proved.

Adult feeding activity is typically diurnal but occasionally crepuscular or nocturnal. It is affected by changes in environmental conditions, particularly temperature and barometric pressure. Species that feed diurnally generally attack hosts throughout the daytime, with discernible periods of higher activity. Females of *Tabanus wilsoni* and *T. pallidescens* tend to feed near midday, whereas *T. abactor* feeds more frequently in late afternoon or early evening. Species that feed during crepuscular periods, particularly dusk, include *Chlorotabanus crepuscularis*, *Leucotabanus annulatus*, *Tabanus moderator*, and *T. equalis*. Some crepuscular species feed into the early night.

FIGURE 16.8 *Tabanus sulcifrons* (late form) feeding on cow. Note blood droplet from prior bite wound. *Photograph by Bradley A. Mullens.*

Tabanid females usually mate before they seek a vertebrate host. Males do not feed on blood. Most species, particularly the Tabaninae, feed on large mammals such as cattle, horses, and deer (Fig. 16.8). Deer flies often attack large mammals, including humans, but there also are records of *Chrysops* spp. feeding on ravens, crows, ducks, and robins. Reptiles such as turtles also may be attacked. Occasionally tabanids may even be rather host-specific, as appears to be the case with *Phorcotabanus cinereus* in Brazil, which feeds preferentially on ducks, attacking exposed, fleshy skin at the base of the bill.

Tabanids are strong fliers and readily disperse several kilometers in short-term flights. Adult **dispersal** probably is influenced by host availability. Mark−release−recapture studies with tabanids can yield 3%−6% recovery, which is very high for insects. This suggests that local populations tend to remain in a given area, with dispersal occurring in a series of short flights. Marked *Tabanus abactor* females have been shown to return to a host at the same site where they had obtained a bloodmeal 3−4 days earlier. Malaise traps hung above waterways in the Brazilian Amazon collected very large numbers of adults, suggesting they may use such waterways for dispersal in those generally forested habitats.

The attack rates by *Chrysops* spp., and to a lesser extent by *Tabanus* and *Hybomitra*, vary substantially in different habitats. Many *Chrysops* spp., for example, tend to frequent forest edges or ecotones, attacking in large numbers a host entering these specific areas from adjacent open fields. Dark-colored hosts, or even dark areas on a black-and-white animal such as a Holstein cow, are often favored for attack.

Many tabanids are selective in attacking specific body regions of their hosts (Fig. 16.9), regardless of color.

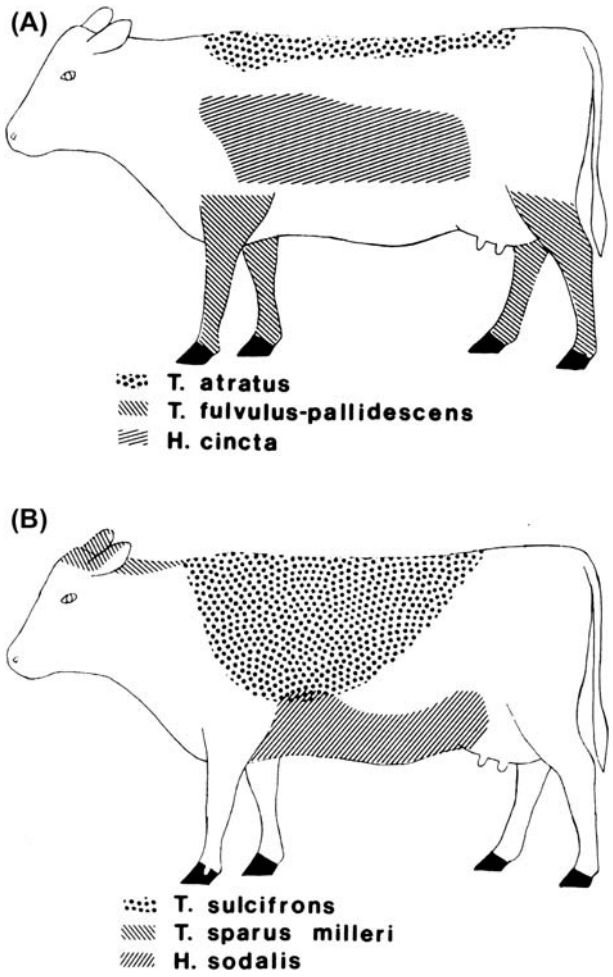


FIGURE 16.9 Feeding sites on cattle characteristic of individual tabanid species. H, *Hybomitra*; T, *Tabanus*. *From Mullens and Gerhardt, 1979.*

Species-specific attack behaviors probably contribute to the disproportionate collections of certain tabanid species in traps. Deer flies usually feed high on the body, especially on the head or shoulders, and are poorly represented in canopy or box traps that require entry from below. Horse flies feed in various regions depending on the species. Legs are favored feeding sites for many horse fly species that attack livestock. Feeding-site selection appears to reflect resource partitioning in both time and space, thereby reducing competition for hosts among tabanid species. Competition both within and among species is mediated by intensified host defensive behaviors such as kicking. In fact, daily animal movement patterns and herd structure may be substantially influenced by fly attack. Animals in larger groups, especially individuals distant from the herd perimeter, tend to suffer fewer bites.

Field studies have documented the importance of persistent feeding behavior in mechanical transmission of disease agents. It is difficult to imagine a better group of potential mechanical vectors than horse flies. They are large, painful biters that are frequently interrupted in the act of feeding. If disturbed or dislodged, they will return to the same host or one nearby within seconds. Their success in initial feeding attempts often is poor. It has been estimated that only 10% of horse flies successfully feed to repletion during the initial attempt on cattle, though this varies among species. Smaller tabanids, versus larger species, tend to be more successful at full engorgement in initial feeding attempts, probably because of less pain from their bites. Vertebrate populations vary in susceptibility over time due to levels of population immunity, and mechanically transmitted tabanid disease outbreaks tend to be periodic, such as every 3–5 years.

The propensity of horse flies to return to the same host animal or transfer to another depends on the fly species and the distance between animals. Larger tabanids are more likely to transfer. It has been estimated that almost 90% of certain horse flies that attack horses will return to the original horse if other hosts are more than 35–50 m away. The moderate-sized tabanid *T. fuscicostatus* has an average of 10 nL of blood residue on its mouthparts after feeding. Biting rates and interrupted feeding are important factors that influence the transmission of disease agents.

Tabanids respond to volatile compounds that serve as **chemical host cues**. The best known and most effective attractant is carbon dioxide. Species vary in their response to CO_2, but even a low release rate of 100 mL/min can result in a 2- to 4-fold increase in collections of tabanid females in traps. Compounds such as 1-octen-3-ol, ammonia, and phenols, or complex mixtures of chemicals in animal urine are detected by tabanid antennae and have been shown to be attractive to certain tabanids. They may act synergistically with each other or with CO_2.

In addition, **visual cues** such as shape, color, and movement of potential hosts are very important. Shades of blue, black, or red are particularly attractive to tabanids, while contrast with the background and reflectance also play a role in the orientation of tabanids to their hosts. Many imaginative traps have been designed to collect tabanids based on their visual response and orientation behavior. Among the more widely used devices are box traps (Fig. 16.10A), Nzi traps using bright blue colors (Fig. 16.10D), and variations on canopy traps (Fig. 16.10B), all of which collect primarily host-seeking females. The effectiveness of canopy traps is enhanced by incorporating movement in the form of a suspended, reflective, black sphere which responds to air movements. The distinctive eye patterns of many species are due to corneal structures acting as interference filters. Older, host-seeking females of some species have been shown to be relatively more sensitive to green wavelengths. This presumably aids in discriminating hosts against a background of green vegetation.

FIGURE 16.10 Traps used for collecting, monitoring, or sometimes suppressing local populations of Tabanidae. (A) Box trap, used on the U.S. eastern seaboard against *Tabanus nigrovittatus*. (B) Canopy trap with black-ball target to enhance attraction. (C) Author standing next to H trap, with container for holding dry ice (CO_2). (D) Nzi trap. *(A) Photograph by Elton J. Hansens. (B) Photograph by Bradley A. Mullens. (C) Photograph by Teresa R. Mullens. (D) Photograph by Steve Mihok.*

Recent research has focused on the role of horizontally polarized light, as might be reflected from a water surface, or less-directional polarized light reflected from different animal coat colors or from a shiny black ball in a trap, in attracting both male and female tabanids. This has implications for understanding their biology, improving monitoring, and possibly contributing to control efforts. It also has been suggested, with some compelling experimental evidence, that zebra stripes function to reduce biting fly attack by reducing this positive polarotaxis, although alternative hypotheses have been proposed, stimulating much interesting discussion among ecologists.

Once tabanid females have located a suitable host, they alight and begin probing. Initial **blood-feeding** attempts are painful and often result in vigorous host response and attempts to dislodge the fly. Flies frequently persist and attack repeatedly. Once blood flow begins, tabanids resist being dislodged, even to the point of sustaining direct

strikes by an animal's tail, feet, or head. An unusually diverse array of chemicals in tabanid saliva maintain blood flow (Fig. 16.8), sometimes for several minutes, via several pathways, including vasodilation, platelet aggregation inhibition, and fibrinolytic enzymes. Tabanids were even used in ancient Eastern medicine to inhibit clotting. It is not unusual for other flies, such as the house fly and face fly, to gather around tabanid feeding wounds to imbibe blood flowing from the wound site.

PUBLIC HEALTH IMPORTANCE

In most temperate areas, adult tabanids are primarily nuisance pests of humans. In this regard they can pose economically significant problems for local tourism. The painful bites, sometimes exceeding 10 per minute, can entirely prevent recreational outdoor activity. Horse-fly larvae can be local pests by inflicting painful bites to the feet of people working in rice paddies. If handled carelessly, the larvae will bite defensively, but they rarely can penetrate the skin of human fingers.

Tabanids transmit some pathogens and parasites biologically, in which cases the disease agent replicates and/or develops within the fly for a period of time prior to transmission (e.g., the filarial nematode *Loa loa*). More commonly, however, tabanids transmit pathogens mechanically via contaminated blood on their mouthparts. Although many pathogenic viruses, bacteria, protozoa, and filarial nematodes have been recovered from tabanids, documentation of transmission is relatively uncommon, in part because of the difficulties in working with tabanids in the laboratory. Many of the disease associations, particularly those involving viruses and bacteria recovered from tabanids, need to be viewed cautiously, as they may be relatively insignificant epidemiologically.

Fortunately, there are relatively few human pathogens transmitted regularly by Tabanidae, as reviewed by Krinsky (1976), Foil (1989), and Baldacchino et al. (2014a). The more significant tabanid-associated diseases are shown in Table 16.3.

Loiasis

The most important tabanid-transmitted disease agent of humans is the **African eyeworm**, *Loa loa*, which causes human loiasis. This filarial nematode is biologically transmitted by *Chrysops* spp. in equatorial rain forests of western and central Africa (including Cameroon, Gabon, and Democratic Republic of the Congo). About 30 million people live in a region with an average *L. loa* prevalence of greater than 30%. **Simian loiasis** is caused by a closely related form (*Loa loa papionis*) and also involves *Chrysops* spp. as vectors. As with other filarial nematodes, transmission of *L. loa* is cyclodevelopmental, requiring the fly as an intermediate host. Interestingly, *Chrysops atlanticus* and several other common deer flies in the southeastern United States have been shown to support the development of *L. loa*.

Adult nematodes live in subcutaneous tissues of the vertebrate host, particularly the thorax, scalp, axillary regions, or eyes. They are 2−7 cm long and produce inflammatory responses as they move through these tissues. If they remain in one area for a time, localized enlargements known as **Calabar swellings** occur; these swellings disappear when the nematode leaves. The common name "eyeworm" is due to adult nematodes sometimes moving across the conjunctiva of the eye (Fig. 16.11). There, or just beneath the skin surface, they often are clearly visible and sometimes can be surgically removed. Migrating nematodes can cause considerable pain, in addition to discoloration and bruising of the affected tissues, particularly evident in the eye.

Mature *L. loa* adults mate and produce microfilariae. Females of *Chrysops* spp. ingest the microfilariae with blood when feeding on an infected person. The microfilariae penetrate the midgut and develop in the abdominal fat bodies or sometimes the thorax. There they molt to second-stage larvae (L2) and eventually move to the head and mouthparts as infective third-stage larvae (L3). In the laboratory, infected deer flies may produce 100 or more *L. loa* infective-stage larvae per fly. This process is temperature dependent and requires at least 7−10 days. On a subsequent feeding, the infective larvae escape from the fly mouthparts and enter a new host through the bite wound during blood-feeding.

The primary vectors of *L. loa* were incriminated in a series of studies in the 1950s in the Congo region of equatorial Africa, which includes parts of Zaire, Congo, Gabon, Cameroon, and southern Nigeria (see Krinsky, 1976). *Chrysops silaceus* and *C. dimidiatus* are particularly attracted to people near fire. From 80% to 90% of their bloodmeals are obtained from humans, and in some hyperendemic areas 90% of the people harbor microfilariae or exhibit loiasis symptoms. Infection rates of 0.5%−1.0% of these two *Chrysops* species have been reported in central Africa, although infection rates in parous flies alone may be much higher. In southern Cameroon, *C. dimidiata* is present at densities estimated to be 800−3,700 flies/km^2, and marked flies moved up to 4.5 km. Depending on the geographic location, people can be subjected to as many as 2,000−3,000 bites per year (i.e., several infective bites per month during the rainy season).

Loiasis has a reputation for low pathogenicity relative to many other parasitic infections, although that idea has been challenged due to the cardiac, renal, and pulmonary symptoms that appear in severe cases. Onchocerciasis control programs have been immensely successful through the widespread and persistent use of ivermectin to reduce or

TABLE 16.3 Selected Disease Agents Transmitted by Tabanids

Disease Agent	Vectors	Geographic Occurrence	Transmission
Viruses			
Equine infectious anemia	*Tabanus, Hybomitra, Chrysops* spp.	Worldwide	Mechanical
Bovine leukemia	*Tabanus* spp.	Worldwide	Mechanical
Hog cholera	*Tabanus* spp.	Worldwide; eradicated from North America, Australia, New Zealand, South Africa	Mechanical
Bacteria/Rickettsia			
Anaplasma marginale	*Tabanus* spp.	Worldwide (Tropics, Subtropics)	Mechanical
Francisella tularensis	*Chrysops* spp.	North America, Russia, Japan	Mechanical
Bacillus anthracis	*Tabanus, Haematopota, Chrysops* spp.	Worldwide	Mechanical
Protozoa			
Besnoitia besnoiti	*Tabanus, Atylotus* spp.	South America, Southern Europe, Africa, Asia,	Mechanical
Trypanosoma evansi	*Tabanus, Haematopota, Chrysops* spp.	South America, North Africa, Asia, India	Mechanical
Trypanosoma vivax	*Tabanus* spp.	South America, Africa	Mechanical
Filarial nematodes			
Loa loa	*Chrysops* spp., esp. *C. dimidiatus, C. silaceus*	Central Africa	Biological
Elaeophora schneideri	*Hybomitra, Tabanus* spp.	North America, southern Europe	Biological

eliminate human carriers, and those treatment areas have also experienced lowered incidence of *L. loa* where both parasites are found. Like *Onchocerca volvulus*, the adults of *L. loa* are not killed by ivermectin, which affects mainly adult nematode reproduction and the infective microfilariae. However, serious encephalopathic adverse reactions have occurred in people treated with ivermectin for *O. volvulus* but who have substantial preexisting *L. loa* infections (>30,000 microfilariae/ml of blood). In one Gabon study, 26% of *L. loa* hosts had infections above that threshold. It thus has become necessary to screen potential patients for high *L. loa* infections first and to consider this potential interaction in some regions where ivermectin is used in onchocerciasis programs.

Tularemia

Tularemia, sometimes called "rabbit fever" or "deer fly fever," is a zoonosis caused by the bacterium *Francisella tularensis*. The name **rabbit fever** reflects the fact that a common method of transmission, particularly in past decades, is through cuts on the hands of hunters and other people handling infected wild rabbits. In the central United States where most American tularemia cases occur, transmission is usually via ticks or direct animal contact. Transmission occurs less commonly by ingestion or aerosol exposure to the bacteria. The epidemiological role of tabanids as vectors in the central United States is unknown. However, transmission by tabanids has been well documented in parts of western North America and is suspected in parts of Russia. Periodic outbreaks of tularemia in Utah (USA) since the early 1900s have been linked convincingly to deer fly bites, particularly those of *Chrysops discalis*, hence the name **deer fly fever**.

At the site of bacterial introduction, a distinctive lesion develops with an ulcerated, pinkish pit in the center and a raised, ridgelike wheal around the perimeter (Fig. 16.12). Bacterial septicemia and resultant fever cause severe illness and occasionally death if the person is not adequately treated with antibiotics. Often the initial lesions are found on the head or upper torso where deer flies commonly bite people. The ability of *C. discalis* to acquire and later transmit the bacterium to humans is dependent on the propensity of this deer fly to feed on other animals, particularly rabbits, which serve as pathogen reservoirs. Occasionally other biting flies, including the horse fly

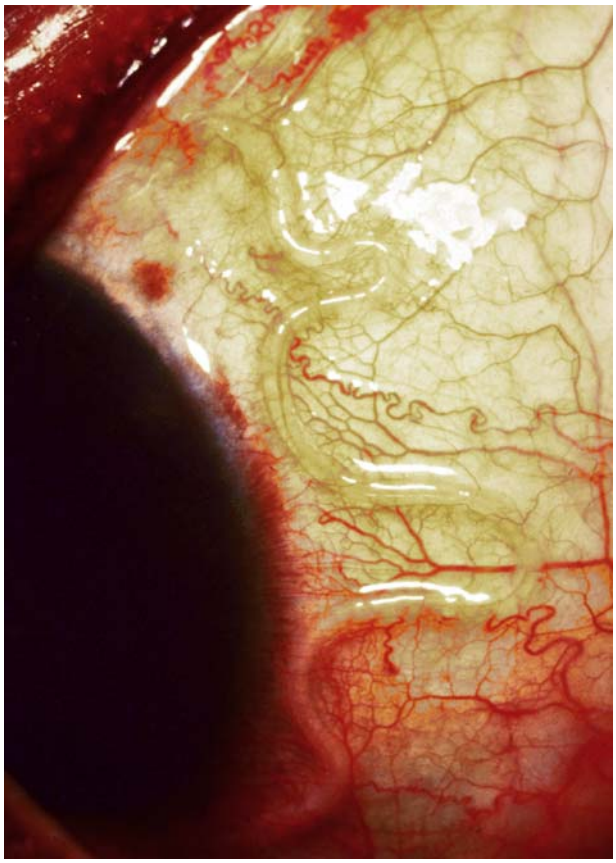


FIGURE 16.11 African eyeworm, *Loa loa*. Adult filarial nematode just beneath conjunctiva of human eye, Cameroon. *Courtesy of U.S. Armed Forces Institute of Pathology, AFIP# 73–6654.*

Tabanus punctifer, also can transmit *F. tularensis*. Such transmission is likely to be mechanical, with the bacterium being introduced into a bite wound via contaminated mouthparts. Feces of deer flies that are fed the bacterium experimentally have been shown to be infective when the feces is rubbed into abraded skin.

Other Tabanid-Transmitted Human Pathogens

Anthrax, a potentially dangerous disease of humans, still occurs worldwide, including localized areas in the United States. Although tabanids are capable of mechanically transmitting the causal bacterium, *Bacillus anthracis*, this mode of transmission is minor in the epidemiology of the disease. Tabanids also were implicated in the 1980s as possible vectors of *Borrelia burgdorferi*, the spirochete that causes **Lyme disease**. The vast majority of transmission is accomplished by ixodid ticks. Tabanids are suspected of contributing to transmission of *B. burgdorferi* in some parts of Europe and North America, but this has not been well documented and requires further study.

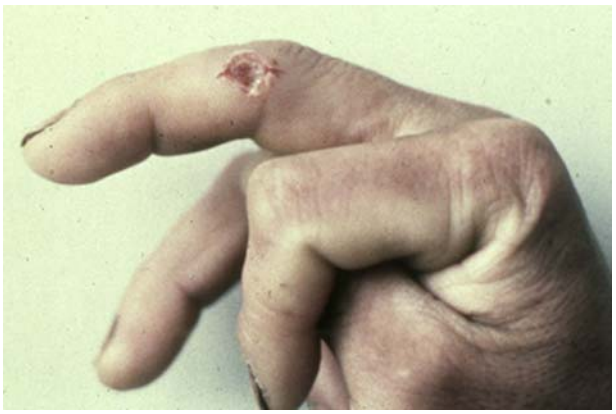


FIGURE 16.12 Tularemia lesion on middle finger of human hand, caused by bacterium *Franciscella tularensis*. *Courtesy of Yoshiro Ohara.*

VETERINARY IMPORTANCE

Owing to their painful, persistent biting behavior, tabanids are significant pests of livestock, particularly cattle and horses, and are extremely bothersome to many wildlife species. Heavy attack by tabanids can cause direct reductions in weight gains of beef cattle, reduced milk yield, reduced feed-utilization efficiencies, and hide damage from the feeding punctures. Cattle protected from tabanid attack in screened enclosures have been shown to gain up to 0.1 kg/day more than control animals exposed to tabanids. Such direct losses can be increased by a concomitant reduction in feed-utilization efficiency of up to 17%. A daily loss of 200 mL of blood per animal may be common during periods of intense tabanid attack.

Tabanids serve as vectors of a number of disease agents of animals, including viruses, bacteria, protozoans, and nematodes (Table 16.3). For thorough overviews of these pathogens, see Krinsky (1976), Foil (1989), or Baldaccino et al. (2014a).

Surra and Related Trypanosomiases

One of the more serious disease agents of livestock transmitted by tabanids is *Trypanosoma evansi*, the causal agent of **surra**. It is morphologically indistinguishable from *T. brucei*. Unlike tsetse fly (*Glossina* sp.) transmission of several other *Trypanosoma* species, including *T. brucei*, tabanid transmission of *T. evansi* is mechanical. The pathogen can be transmitted via contaminated blood in various ways, including direct transmission vertebrate-to-vertebrate under some circumstances. Vampire bats may serve as a long-term reservoir and transmit it biologically (via infected saliva) in parts of Central and South America, where the disease is known as **murrina**. Surra is the most widely distributed of the vector-borne trypanosome diseases and affects a variety of wild and domestic mammals

in northern Africa, southern Asia, the Philippines and Indonesia, and parts of Central and South America. It was apparently introduced into the Western Hemisphere by Spaniards in the 16th century via infected horses. Untreated infections are usually 100% fatal in horses, elephants, and dogs. The disease can be serious and chronic in camels, which are thought to be the original hosts. Cattle and buffalo, in contrast, are not severely affected and may remain asymptomatic for months. Relatively resistant animals can serve as reservoirs of infection. Symptoms of infection are similar to other trypanosomiases. Premodern peoples sometimes had correctly ascertained arthropod transmission before it was scientifically proved; an old Arab name for surra, dating from the early 1900s or before, is **mard el debab**, which means "sickness of the gadflies." In the Punjam region of India, it is called **makhi ki bimari** ("horse fly disease").

Particularly in northern South America, mechanical transmission by tabanids of a related pathogen, *T. vivax*, is a serious problem for sheep producers and, to a lesser extent, for cattlemen. *Trypanosoma equinum* infects horses in South America where it causes a disease called **mal de caderas**, with symptoms similar to surra. In parts of Africa, mechanical transmission by tabanids of species such as *T. brucei*, normally transmitted by tsetse flies, may be significant. Research in field cages (to exclude tsetse) demonstrated mechanical transmission of *T. congolense* and *T. vivax* among cattle in Africa by the tabanid *Atylotus agrestis.*

Trypanosoma theileri causes widespread, generally nonpathogenic infections of cattle and wild hosts such as deer. This trypanosome is found commonly in the hindgut (i.e, singular) of tabanids but is absent from the salivary glands. Transmission therefore occurs primarily through feces entering the bite wound or perhaps by crushing or ingestion of infected tabanids by the animal. In the latter case, infection occurs through abrasions and breaks in the skin or through oral mucosa. Research on the epizootiology of this and other trypanosome diseases is complicated by the presence of *Blastocrithidia* spp., nonpathogenic trypanosomes that occur naturally in tabanids and are easily confused with pathogenic trypanosomes.

Equine Infectious Anemia

Equine infectious anemia (EIA), commonly known as **swamp fever**, is a serious viral disease of horses and other equids (Fig. 16.13). It is a febrile illness that causes lethargy, weight loss, and sometimes death in affected animals. Different strains of EIA virus differ in pathogenicity, and infected animals differ significantly in how they are affected. Acutely infected animals nearly always die fairly quickly. Chronically infected animals eventually succumb to complications, and inapparent carriers may live a number of years with few obvious health problems. This disease is found in many areas of the world and is common in the southeastern United States. A series of studies conducted mostly in Louisiana (USA) provides a good model for understanding mechanical transmission of pathogens by tabanids (Foil, 1989), especially EIA and other retroviruses such as bovine or feline leukemia viruses.

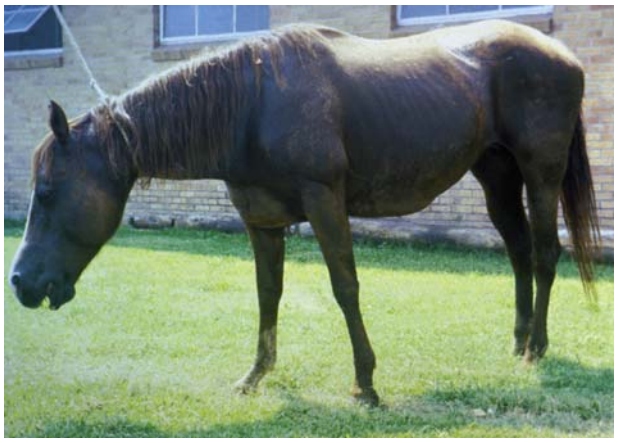


FIGURE 16.13 Horse suffering from equine infectious anemia. *Photograph by W. V. Adams, Jr.*

Because virus infectivity declines rapidly on insect mouthparts, rapid and frequent transfer of vectors between hosts is essential for significant transmission. Even though the amount of blood transferred by an individual fly is low, the potential for transmission is high when multiplied by a large number of persistent flies feeding at a given time. At high pathogen titers (10^6/mL), single tabanid bites can transmit the pathogen.

Anaplasmosis

Ticks and tabanids are regarded as the primary vectors of *Anaplasma marginale*, a rickettsia that causes anaplasmosis in cattle. This disease is most prevalent in the tropics and subtropics, including Africa, Australia, and the Americas. In the United States, the incidence of disease is highest in the southeastern states. While calves seldom are affected severely, adult cattle show marked anemia, fever, and weight loss. Mortality may be as high as 50%.

Ticks transmit the rickettsia biologically and are probably more important vectors than biting flies. Tick transmission is well over two orders of magnitude more efficient than transmission by tabanids or stable flies, and some fly transmission trials have used splenectomized hosts, which are somewhat immunocompromised. Still, mechanical transmission by tabanids up to 2 h postfeeding has been documented in the literature, and there are areas where they

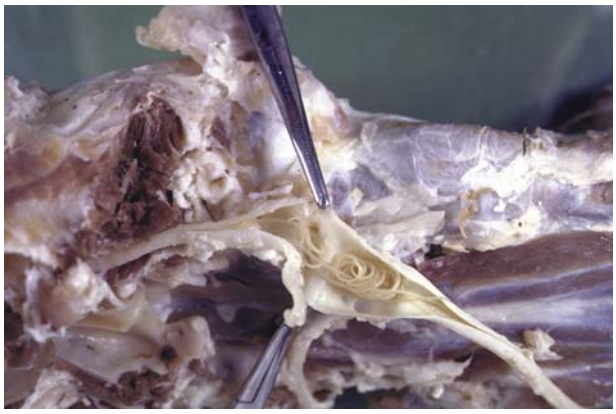


FIGURE 16.14 Arterial worm, *Elaeophora schneideri*. Adult filarial nematode in carotid artery of white-tailed deer. *Courtesy of Southeastern Cooperative Wildlife Disease Study, University of Georgia.*

are thought to be important vectors. Particularly when biting rates are high, horse flies may be significant in transmitting *A. marginale* among susceptible animals during anaplasomosis outbreaks.

Elaeophorosis

A filarial nematode of domestic animals and wild ruminants, *Elaeophora schneideri* (Fig. 16.14), is widely distributed in North America (Pence, 1991). In the Rocky Mountain states (USA), infection rates in mule deer (*Oedocoileus hemionus*) often exceed 50%. Mule deer apparently are the normal reservoir host in western North America and rarely show pathological effects. Compared with mule deer, white-tailed deer (*O. virginianus*) are infected at a lower level (2%−10%) in many regions of the southern United States and sometimes show clinical signs of infection. Domestic sheep and goats may harbor the nematodes but generally do not develop notable pathology. Some wild ruminants, particularly elk and moose, can be severely affected; up to 90% or more of the elk in parts of Arizona and New Mexico (USA) have been shown to harbor the nematode. In susceptible hosts, *E. schneideri* causes obstruction of arterial flow, hence the common name **arterial worm**. Reduced blood flow results in dermatosis, sloughing of distal tissues such as ear or antler tips, blindness, neurological disease, and sometimes death.

At least 16 species of tabanids in the genera *Tabanus*, *Hybomitra*, and *Silvius* have been implicated in the transmission of *E. schneideri*. In the United States, prevalence of nematodes in horse flies captured from hyperendemic areas of New Mexico has been as high as 10%−20%. Prevalence in other states, such as parts of southern Montana and South Carolina, is less than 1% in field-collected flies. In a given region, one or two species, such as *T. laticornis* in New Mexico, seem to be responsible for most of the transmission. Following ingestion of microfilariae by a tabanid, nematode development proceeds in the fat body and hemocoel. Infective larvae (L3) are found in the mouthparts after about 14 days.

Other Pathogens of Veterinary Importance

Several other pathogens are transmitted mechanically by tabanids to livestock and wild animals. Most of them, however, also may be transmitted in other ways. Notable among these are the viruses that cause **bovine leukemia** and **hog cholera** and the sporozoan genus *Besnoitia* that causes **besnoitosis**, which appears to be spreading in Europe. The role of tabanids in transmitting these pathogenic agents is usually very secondary.

There are several other interesting, but economically minor, disease agents transmitted to wild animals by tabanids. A protozoan parasite of turtles, *Haemaproteus metchnikovi*, is transmitted by deer flies (*Chrysops* spp.), and tabanids biologically transmit the filarial nematode *Dirofilaria roemeri*, a pathogen of kangaroos and wallabies.

PREVENTION AND CONTROL

Tabanid control is difficult to achieve. A given area usually has multiple species with different seasonal occurrences and biological characteristics. Typical host contact is only about 4 min per fly during blood feeding, which may occur only once every 3−4 days. Short-term control on livestock for several days may be achieved through the use of insecticides, but insecticide sprays often are not particularly effective. Aerial applications of pyrethroid insecticides can suppress local populations of certain tabanids over a period of several hours to perhaps a few days, but are considered far less effective than similar applications made for mosquito control. Use of insecticides for control of larvae or pupae, which are typically inaccessible in soil, is generally ineffective and can result in environmental damage. Insect repellents applied to human skin or impregnated into clothing may offer temporary relief for humans, but some individuals report poor results when using them.

Providing animals with structures for shelter, or pasturing them away from pasture−forest ecotones or avoiding areas or seasons with known high tabanid activity, can help to reduce tabanid biting intensity. Because tabanids prefer to fly around rather than over vegetation or screen **barriers** over 2 m high, such barriers can help to reduce tabanid access to livestock.

Another approach to tabanid control is water management in areas where drainage or manipulation of water levels is feasible. This must be done carefully, however, because such practices can actually enhance tabanid populations. Larvae of the salt marsh greenhead *Tabanus nigrovittatus*, for example, are more abundant in better

drained sites, and ditching salt marshes can increase its populations. Even in restricted habitats, such as livestock ponds in pastures, water management is not always feasible. Heavy rains and flooding during periods when tabanids are pupating can kill the pupae, resulting in fewer adults a few weeks later. In situations where oviposition sites are limited, properly timed removal of emergent vegetation on which tabanid eggs are deposited can result in a significant reduction in the number of eggs and resultant larvae.

Traps are commonly used to monitor tabanid adults, but they also provide another potential tool for control. Some new designs of large, canopy or curtain-type traps for tabanid control in pastures have been developed and marketed in the United States (Fig. 16.10B). There has been recent, promising research on using modifications of traps such as the Nzi trap (Fig. 16.10D), initially investigated for tsetse in Africa, because they also collect other biting flies, including tabanids. In some coastal areas of the eastern United States, box traps are widely used for suppression of the salt marsh greenheads *Tabanus nigrovittatus* and *T. conterminus* (Fig. 16.10A). These flies are obligately autogenous, and responding females already have laid eggs. Under certain conditions, the traps may distract enough host-seeking adults from humans to result in temporary relief. Similar efforts have been made on a smaller scale using canopy traps, with limited success.

Where large, fairly mobile populations of horse flies occur, even large collections of adults may result in questionable suppression. In a study in the southeastern United States, 95,000 tabanids were captured with 20 CO_2-baited sticky traps from a pasture area over a period of several days, but this action resulted only in very temporary reduction in numbers of tabanids attacking cattle. The development of better attractants or attractant combinations (chemicals, colors, fabrics, decoys, etc.) for adult tabanids is enhancing the control potential of traps. One inventive device even coupled a horizontal, dark reflective surface (reflecting polarized light) to a solar-powered motor driving a constantly spinning wire. This destroyed substantial numbers of male and female tabanids coming during the day to the target.

The use of **biological control agents** also offers some potential for reducing tabanid populations. All stages of tabanids are subject to mortality by predators, including ladybird beetle larvae preying on eggs, wading birds feeding on larvae, and dragonflies and certain solitary wasps attacking adults. A few species of bembicine wasps (Sphecidae, Bembicinae) called **horse guards** specialize in capturing adult tabanids, on which they rear their young. They tend to fly around pastured animals, where they seize tabanids as they attempt to feed. Two species of horse guards in the southeastern United States are *Strictia carolina* and *Bembix texana*. Cannibalism among horse-fly larvae also may be important in biological control.

Tabanid eggs are parasitized by wasps in the families Trichogrammatidae and Scelionidae, which can sometimes cause high egg mortality (>50%). Tabanid larvae are parasitized by flies in the Tachinidae and Bombyliidae, and pupae are parasitized by wasps in the Diapriidae and Pteromalidae. Tabanid larvae also are subject to mortality from nematode parasites in the family Mermithidae. At least one mermithid species, *Pheromermis myopis*, parasitizes other invertebrates that are likely to be eaten by tabanid larvae, thereafter killing the tabanid larvae while completing its development. A number of fungal, bacterial, and protozoan pathogens also are known from tabanids.

REFERENCES AND FURTHER READING

Akotet, M. K. B., Owano-Medang, M., Mawili-Mbouma, D. P., Moussavou-Boussougou, M. N., Afene, S. N., Kendjo, E., et al. (2016). The relationship between microfilaremic and amicrofilaremic loiasis involving co-infection with *Mansonella perstans* and clinical symptoms in an exposed population from Gabon. *Journal of Helminthology, 90*, 469–475.

Allan, S. A., Day, J. F., & Edman, J. D. (1987). Visual ecology of biting flies. *Annual Review of Entomology, 32*, 297–316.

Anderson, J. F. (1985). The control of horse flies and deer flies (Diptera: Tabanidae). *Myia, 3*, 547–598.

Andreeva, R. V. (1984). *The ecology of horse fly larvae and their parasitoses*. Kiev: Naukova Dumka, 170 pp.

Andreeva, R. V. (1989). The morphological adaptations of horse fly larvae (Diptera: Tabanidae) to developmental sites in the Palearctic region and their relationship to the evolution and distribution of the family. *Canadian Journal of Zoology, 67*, 2286–2293.

Andreeva, R. V. (1990). *Identification of the larvae of horse flies of the European part of the USSR, Caucasus and central Asia*. Kiev: Naukova Dumka, 170 pp.

Auroi, C. (1983). The life cycle of *Hybomitra bimaculata* (Marqu.) (Diptera: Tabanidae). III. Pupation, emergence, blood meal and oogenesis. *Mitteilungen der Schweizerischen Entomologischen Gesellschaft, 56*, 343–359.

Baldacchino, F., Duquesnes, M., Mihok, S., Foil, L. D., Duvallet, G., & Jittapalapong, S. (2014a). Tabanids: Neglected subjects of research, but important vector of disease agents! *Infection, Genetics and Evolution, 28*, 596–615.

Baldacchino, F., Manon, S., Puech, L., Buatois, B., Dormont, L., & Jay-Robert, P. (2014b). Olfactory and behavioural responses of tabanid horseflies to octenol, phenols and aged horse urine. *Medical and Veterinary Entomology, 28*, 201–209.

Barros, A. T. M., & Foil, L. D. (2007). The influence of distance on movement of tabanids (Diptera: Tabanidae) between horses. *Veterinary Parasitology, 144*, 380–384.

Blaho, M., Egri, A., Barta, A., Antoni, G., Kriska, G., & Horvath, G. (2012). How can horseflies be captured from solar panels? A new concept of tabanid traps using light polarization and electricity produced by photovoltaics. *Veterinary Parasitology, 189*, 353–365.

Burger, J. F. (1977). The biosystematics of immature Arizona Tabanidae (Diptera). *Transactions of the American Entomological Society, 103*, 145−158.

Burger, J. F. (1995). Catalog of Tabanidae (Diptera) of North America North of Mexico. In *Contributions on entomology, international* (Vol. 1, pp. 1−10).

Burger, J. F., & Chainey, J. E. (2000). Revision of the Oriental and Australasian species of *Chrysops* (Diptera: Tabanidae). *Invertebrate Taxonomy, 14*, 607−654.

Burger, J. F., Lake, D. J., & McKay, M. L. (1981). The larval habitats and rearing of some common *Chrysops* species (Diptera: Tabanidae) in New Hampshire. *Proceedings of the Entomological Society of Washington, 83*, 373−389.

Chippaux, J.-P., Bouchité, B., Demanov, M., Morlais, I., & LeGoff, G. (2000). Density and dispersal of the loiasis vector *Chrysops dimidiata* in southern Cameroon. *Medical and Veterinary Entomology, 14*, 339−344.

Chvala, M., Lyneborg, L., & Moucha, J. (1972). *The horse flies of Europe (Diptera, Tabanidae)*. Denmark: Entomological Society of Copenhagen, 499 pp.

Cooksey, L. M., & Wright, R. E. (1989). Population estimation of the horse fly, *Tabanus abactor* (Diptera: Tabanidae) in north central Oklahoma. *Environmental Entomology, 16*, 211−217.

Coscaron, S., & Papavero, N. (1993). *An illustrated manual for the identification of the neotropical genera and subgenera of Tabanidae (Diptera)*. Belem, Brazil: Museu Paraense Emilio Goeldi.

Dukes, J. C., Edwards, T. D., & Axtell, R. C. (1974). Distribution of larval Tabanidae (Diptera) in a *Spartina alterniflora* salt marsh. *Journal of Medical Entomology, 11*, 79−83.

Desquesnes, M., Dargantes, A., Hua, D.-H., Lun, Z.-R., Holzmuller, P., & Jittapalapong, S. (2013). *Trypanosoma evansi* and surra: A review and perspectives on transmission, epidemiology and control, impact and zoonotic aspects. *BioMed Research International*. https://doi.org/10.1155/2013/321237.

Desquesnes, M., & Dia, M. L. (2003). Mechanical transmission of *Trypanosoma congolense* in cattle by the African tabanid *Atylotus agrestis*. *Experimental Parasitology, 105*, 226−231.

Egri, A., Blaho, M., Kriska, G., Farkas, R., Gyurkovsky, M., Akesson, S., et al. (2012). Polarotactic tabanids find striped patterns with brightness and/or polarization least attractive: An advantage of zebra stripes. *Journal of Experimental Biology, 215*, 736−745.

Fairchild, G. B. (1969). Climate and the phylogeny and distribution of Tabanidae. *Bulletin of the Entomological Society of America, 15*, 7−11.

Fairchild, G. B. (1986). Tabanidae of Panama. In *Contributions of the American Entomological Institute 22* (pp. 1−13).

Fairchild, G. B., & Burger, J. F. (1994). A catalog of the Tabanidae (Diptera) of the Americas south of the United States. In *Memoirs of the American Entomological Institute No. 55*, 249 pp.

Ferreira-Keppler, R. L., Rafael, J. A., & Guerrero, J. C. H. (2010). Seasonality and landscape use by Tabanidae species in the Central Amazon, Brazil. *Neotropical Entomology, 39*, 645−654.

Foil, L. D. (1989). Tabanids as vectors of disease agents. *Parasitology Today, 5*, 88−96.

Goodwin, J. T. (1982). The Tabanidae (Diptera) of Mali. In *Miscellaneous Publications of the Entomological Society of America 13* (pp. 1−14).

Goodwin, J. T., & Drees, B. M. (1996). The horse flies and deer flies (Diptera: Tabanidae) of Texas. *Southwestern Entomologist, 20*(Suppl.).

Goodwin, J. T., Mullens, B. A., & Gerhardt, R. R. (1985). The Tabanidae of Tennessee. In *Tennessee Agricultural Experiment Station Bulletin 642*, 73 pp.

Hayakawa, H. (1985). A key to the females of Japanese tabanid flies with a checklist of all species and subspecies (Diptera, Tabanidae). *Japanese Journal of Sanitary Zoology, 36*, 15−23.

Hollander, A. L., & Wright, R. E. (1980). Impact of tabanids on cattle: Blood meal size and preferred feeding sites. *Journal of Economic Entomology, 73*, 431−433.

Husseneder, C., Delatte, J. R., Krumbolt, J., & Foil, L. D. (2015). Development of microsatellites for population genetic analyses of *Tabanus nigrovittatus* (Diptera: Tabanidae). *Journal of Medical Entomology, 51*, 114−118.

Iranpour, M., Shurko, A. M., Klassen, G. R., & Galloway, T. D. (2004). DNA fingerprinting of tabanids (Diptera: Tabanidae) and their respective egg masses using PCR-restriction fragment profiling. *The Canadian Entomologist, 136*, 605−619.

Jones, C. M., & Anthony, D. W. (1964). *The Tabanidae of Florida*. USDA Technical Bulletin 1295 (p. 85).

Karolyi, F., Colville, J. F., Handschuh, S., Metscher, B. D., & Krenn, H. W. (2014). One proboscis, two tasks: Adaptations to blood-feeding and nectar-extracting in long-proboscid horse flies (Tabanidae, *Philoliche*). *Arthropod Structure & Development, 43*, 403−413.

Krinsky, W. L. (1976). Animal disease agents transmitted by horse flies and deer flies (Diptera: Tabanidae). *Journal of Medical Entomology, 13*, 225−275.

Krcmar, S., Mikuska, A., & Merdic, E. (2006). Response of Tabanidae (Diptera) to different natural attractants. *Journal of Vector Ecology, 31*, 262−265.

Lane, R. S., Anderson, J. R., & Philip, C. B. (1983). Biology of autogenous horse flies native to coastal California: *Apatolestes actites* (Diptera: Tabanidae). *Annals of the Entomological Society of America, 76*, 559−571.

LePrince, D. J., & Lewis, D. J. (1986). Sperm presence and sugar feeding patterns in nulliparous and parous *Tabanus quinquevittatus* Wiedemann (Diptera: Tabanidae) in southwestern Quebec. *Annals of the Entomological Society of America, 79*, 912−917.

Ma, D. Y., Wang, Y. P., Yang, H. L., Wu, J., An, S., Gao, L., et al. (2009). Anti-thrombosis repertoire of blood-feeding horsefly salivary glands. *Molecular & Cellular Proteomics, 8*, 2071−2079.

Mackerras, I. M. (1954). The classification and distribution of Tabanidae (Diptera). I. General review. *Australian Journal of Zoology, 2*, 431−454.

McElligott, P. E. K., & Lewis, D. J. (1996). Distribution and abundance of immature Tabanidae (Diptera) in a subarctic Labrador peatland. *Canadian Journal of Zoology, 74*, 1364−1369.

McKeever, S., & French, F. E. (1997). Fascinating, beautiful blood feeders−deer flies and horse flies, the Tabanidae. *American Entomologist, 43*, 217−226.

McMahon, M. J., & Gaugler, R. (1993). Effect of salt marsh drainage on the distribution of *Tabanus nigrovittatus* (Diptera: Tabanidae). *Journal of Medical Entomology, 30*, 474−476.

Metzger, W. G., & Mordmuller, B. (2014). *Loa loa* − does it deserve to be neglected? *The Lancet Infectious Diseases, 14*, 353−357.

Middlekauf, W. W., & Lane, R. S. (1980). Adult and immature Tabanidae (Diptera) of California. *Bulletin of the California Insect Survey, 22*, 99.

Mihok, S., & Carlson, D. A. (2007). Performance of plywood and cloth Nzi traps relative to Manitoba and greenhead traps for tabanids and stable flies. *Journal of Economic Entomology, 100*, 613–618.

Mihok, S., & Lange, K. (2012). Synergism between ammonia and phenols for *Hybomitra* tabanids in northern and temperate Canada. *Medical and Veterinary Entomology, 26*, 282–290.

Mikuska, A., Krcmar, S., Radovic, A., & Mikuska, T. (2012). The influence of temperature, precipitation and floods on the development of horse fly populations (Tabanidae) in the alluvial habitats of the Danube River in Croatia. *Polish Journal of Ecology, 60*, 395–406.

Morita, S. I., Bayless, K. M., Yeates, D. K., & Wiegmann, B. M. (2016). Molecular phylogeny of the horse flies: A framework for renewing tabanid taxonomy. *Systematic Entomology, 41*, 56–72.

Mullens, B. A., & Gerhardt, R. R. (1979). Feeding behavior of some Tennessee Tabanidae. *Environmental Entomology, 8*, 1047–1051.

Oldroyd, H. (1954–1957). *Horseflies of the Ethiopian region, I–III*. London: British Museum of Natural History.

Olsufiev, N. G. (1977). Horse flies. Family Tabanidae. In *Fauna of the USSR. New series. No. 113, insects, Diptera, 7(2)*. Leningrad: Izdatelistvo Nauka, 434 [+2] pp.

Pechuman, L. L. (1981). *The horse flies and deer flies of New York (Diptera: Tabanidae)*. Search Agriculture, Cornell University Agricultural Experiment Station 18, 68 pp.

Pechuman, L. L., & Teskey, H. J. (1981). Tabanidae. In J. F. McAlpine (Ed.), *Manual of Nearctic Diptera* (Vol. 1, pp. 463–478). Agriculture Canada Research Branch, Monograph 27, 674 pp.

Pechuman, L. L., Webb, D. W., & Teskey, H. J. (1983). The Diptera, or true flies, of Illinois. I. Tabanidae. In *Illinois natural history survey bulletin 33*, 122 pp.

Pence, D. B. (1991). Elaeophorosis in wild ruminants. *Bulletin of the Society for Vector Ecology, 16*, 149–160.

Perich, M. J., Wright, R. E., & Lusby, K. S. (1986). Impact of horse flies (Diptera: Tabanidae) on beef cattle. *Journal of Economic Entomology, 79*, 128–131.

Poinar, G. O., Jr. (1985). Nematode parasites and infectious diseases of Tabanidae (Diptera). *Myia, 3*, 599–616.

Schutz, S. J., & Gaugler, R. (1989). Honeydew-feeding behavior of salt marsh horse flies (Diptera: Tabanidae). *Journal of Medical Entomology, 26*, 471–473.

Scoles, G. A., Miller, J. A., & Foil, L. D. (2008). Comparison of the efficiency of biological transmission of *Anaplasma marginale* (Rickettsiales: Anaplasmataceae) by *Dermacentor andersoni* Stiles (Acari: Ixodidae) with mechanical transmission by the horse fly, *Tabanus fuscicostatus* Hine (Diptera: Tabanidae). *Journal of Medical Entomology, 45*, 109–114.

Smith, S. M., Turnbull, D. A., & Taylor, P. D. (1994). Assembly, mating, and energetics of *Hybomitra arpadi* (Diptera: Tabanidae) at Churchill, Manitoba. *Journal of Insect Behavior, 7*, 355–383.

Sutton, B. D., & Carlson, D. A. (1997). Cuticular hydrocarbon variation in the Tabanidae (Diptera): *Tabanus nigrovittatus* complex of the North American Atlantic coast. *Annals of the Entomological Society of America, 90*, 542–549.

Takahasi, H. (1962). *Fauna Japonica. Tabanidae (Insecta: Diptera)*. Biogeographical Society of Japan, National Science Museum, 143 pp.

Teskey, H. J. (1990). *The insects and arachnids of Canada, Part 16. The horse flies and deer flies of Canada and Alaska (Diptera: Tabanidae)*. Agriculture Canada Publication 1838, 381 pp.

Waage, J. K., & Davies, C. R. (1986). Host-mediated competition in a bloodsucking insect community. *Journal of Animal Ecology, 55*, 171–180.

Wiegmann, B. M., Yeates, D. K., Thorne, J. L., & Kishino, H. (2003). Time flies – a new molecular time scale for brachyceran fly evolution without a clock. *Systematic Biology, 52*, 745–756.

Wilkerson, R. C., Butler, J. F., & Pechuman, L. L. (1985). Swarming, hovering, and mating behavior of male horse flies and deer flies (Diptera: Tabanidae). *Myia, 3*, 515–546.

Wilson, B. H. (1968). Reduction of tabanid populations on cattle with sticky traps baited with dry ice. *Journal of Economic Entomology, 61*, 827–829.

This page intentionally left blank

Chapter 17

Muscid Flies (Muscidae)

Roger D. Moon
Department of Entomology, University of Minnesota, St. Paul, MN, United States

The family Muscidae includes significant blood-feeding parasites, vectors of disease agents, and species that annoy humans and domesticated animals. These flies and others in related families are often called **synanthropic flies**, species that exploit foods and habitats created by agriculture and other human activities. Muscid flies and their relatives can be grouped according to their habitat affinities. There are **filth flies**, such as the house fly, whose adults and immatures occur in a variety of filthy organic substrates, including latrines, household garbage, manure, and manure-soiled animal bedding. A subset of filth flies are **dung flies**, such as the horn fly, whose immatures occur exclusively in cattle droppings. Another group is **sweat flies**, whose adults feed persistently on perspiration. Finally, larvae in two genera are obligate blood feeders that cause myiasis in nestling chicks of raptors and passerine birds.

Muscid flies also can be grouped by the nature of their mouthparts. Nonbiting muscid flies have sponging mouthparts used to ingest liquids from inanimate substrates and animal tissues. These mouthparts are soft, fleshy, and incapable of penetrating skin. In contrast, biting muscid flies have piercing-sucking mouthparts that can pierce skin to obtain blood.

Useful reviews of literature on muscid flies include a two-volume treatise on the biology and disease associations of synanthropic flies (Greenberg, 1971, 1973) and a monograph on the identification and biology of immature muscid flies (Skidmore, 1985). A comprehensive review of veterinary effects and control of muscid flies and other arthropods on livestock is provided by Drummond et al. (1988). Additional reviews and bibliographies concentrate on selected species and their close relatives: house fly (Thomas and Skoda, 1993; West, 1951; West and Peters, 1973); stable fly (Morgan et al., 1983a; Petersen and Greene, 1989; Thomas and Skoda, 1993; Zumpt, 1973); horn fly (Bruce, 1964; Morgan and Thomas, 1974, 1977); and face fly (Morgan et al., 1983b; Pickens and Miller, 1980; Krafsur and Moon, 1997).

TAXONOMY

The Muscidae include approximately 5,000 species in 200 genera. The North American fauna includes ca 700 species in 46 genera. Only a few of these genera are of medical or veterinary concern. Important North American taxa are listed in Table 17.1. Five of them have been introduced from the Old World through human commerce. Outside North America, the same species, or close relatives with similar life cycles, habits, and ecology, may be encountered.

Important muscid flies (Table 17.2) occur in two subfamilies: the Muscinae and the Fanniinae. Important nonbiting Muscinae are the house fly, garbage flies, false stable fly and relatives, face fly, and sweat flies. Important biting Muscinae are the stable fly and horn fly. A third biting species in North America, the moose fly (*Haematobosca alcis*), occurs exclusively on moose (*Alces alces*). The second subfamily, the Fanniinae are represented by the nonbiting little house fly and its relatives (*Fannia* spp.). Although current authors consider the Fanniinae to be a separate family (Fanniidae), it is treated here as a subfamily of Muscidae.

Adults and larvae of North American Diptera can be identified to family using keys in McAlpine et al. (1981); adult Muscidae can be keyed to genus using McAlpine et al. (1987) and references therein. The Muscidae of North America are cataloged in Stone et al. (1965). Other aids for identification are Skidmore's (1985) keys and descriptions of larvae and pupal cases (puparia), and James' (1947) classic treatment of adults and larvae of Muscidae and other families that cause myiasis.

Medical and Veterinary Entomology. https://doi.org/10.1016/B978-0-12-814043-7.00017-0

TABLE 17.1 Important Muscid Pests of Humans and Domesticated Animals in North America

Ecological Group	Common Name	Scientific Name	Hosts for Adults
Filth flies	House fly[a]	*Musca domestica*	None required
	Stable fly[a]	*Stomoxys calcitrans*	Cow, horse, dog, Humans and others
	Garbage flies	*Hydrotaea* (= *Ophyra*[b]), *Hydrotaea aenescens*, *Hydrotaea ignava*[a] (= *Ophyra leucostoma*)	None required
	False stable fly and relative	*Muscina stabulans*, *Muscina levida* (= *assimilis*[c])	None required
	Little house fly	*Fannia canicularis*	None required
	Latrine fly, and relative	*Fannia scalaris*, *Fannia femoralis*	
Dung flies	Horn fly[a]	*Haematobia irritans irritans*	Cow, bison, horse
	Face fly[a]	*Musca autumnalis*	Cow, bison, horse
Sweat flies	Sweat flies	*Hydrotaea meteorica*, *Hydrotaea scambus*, and others	Large mammals

[a]*Species introduced to North America from Old World; others are cosmopolitan or native to New World.*
[b]*According to Huckett and Vockeroth (1987).*
[c]*According to Skidmore (1985).*

TABLE 17.2 Life-History Attributes of Important North American Muscid Flies

Common Name	Days From Egg to Adult, Minimum	Days From Egg to Adult, Normal	Eggs per Cycle	Generations per Year	Overwintering Stages(s)
House fly	7	10–21	120–150	Multiple	All?
Stable fly	12	15–30	80–100	Multiple	All?
Garbage flies	9	14–25	70–190	Multiple	Larva
False stable fly and relatives	10	14–40	98–150	Multiple	Adult in diapause
Little house fly and relatives	22	25–50	58–72	Multiple	Larva?
Horn fly	9	10–20	20–32	Multiple	Pupa in diapause
Face fly	7	12–28	16–42	Multiple	Adult in diapause
Sweat flies	25	35–300	17–150	1–2	Larva

Compiled From Skidmore (1985) and Other **Sources**.

MORPHOLOGY

Life stages of a typical muscid fly consist of egg, larva, pupa, and adult (Fig. 17.1). The egg is similar to those of closely related families and may occur singly or in groups. The egg is generally creamy in color, 0.8–2.0 mm long, elongate-ovate in shape, and concave dorsally where two ribs form hatching pleats (Fig. 17.2).

The larva of muscid flies and related families is known as a maggot, and there are three instars in all the important species. The body is tapered, with the head and mouth hooks at the pointed end and the anus and spiracles at the blunt end (Fig. 17.1). The head is greatly reduced; it lacks eyes and has minute antennae that resemble papillae. The thorax is legless and has a pair of lateral **prothoracic spiracles**. There are eight segments of the abdomen, and each is marked ventrally with transverse rows of spines forming **creeping welts**.

Although the head lacks a sclerotized capsule, it is supported internally by a sclerotized cephalopharyngeal skeleton (Fig. 17.3). This complex structure is partially visible through the integument of a live larva but is best visualized in a cleared, slide-mounted specimen. The size,

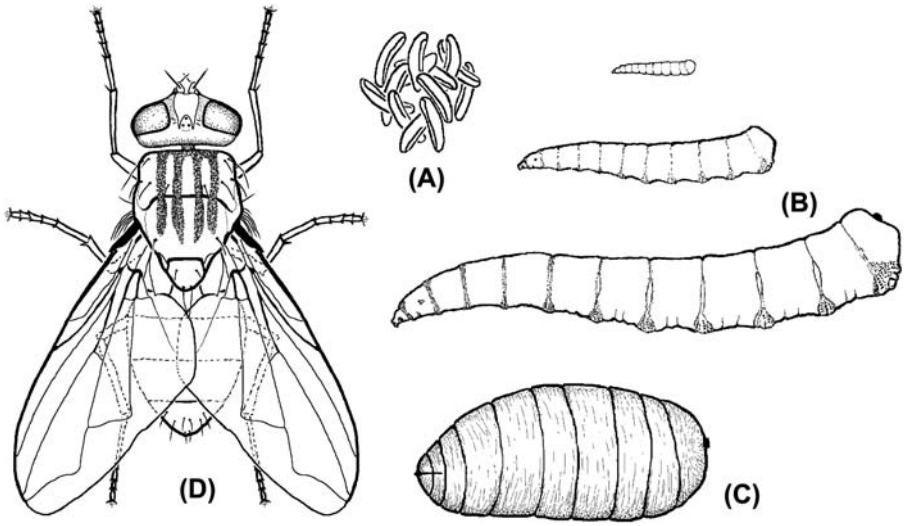


FIGURE 17.1 Life cycle of house fly (*Musca domestica*). (A) Eggs. (B) Larvae, three instars. (C) Pupa. (D) Adult. *Traced from drawing by Fritz Gregor in Greenberg (1971).*

shape, and arrangement of elements of the cephalopharyngeal skeleton are useful in the identification of larvae. Paired mouth hooks, reduced from ancestral mandibles, can be extended and retracted from the oral cavity. They help in crawling, burrowing, and tearing into food and other substrates. Internal dental sclerites, accessory sclerites, and pharyngeal sclerites make up the rest of the cephalopharyngeal skeleton and are variously modified for muscle attachment and feeding.

Most muscid larvae occur in wet substrates where they filter particles of food from the substrate. These filter feeders possess a porous, ventral sieve mechanism between their pharyngeal sclerites (Fig. 17.3A). Exceptions are *Muscina* spp. and *Hydrotaea* spp., whose final instars are facultative or obligate predators. The sieving mechanism is absent in these predatory forms (Fig. 17.3B).

Characters of the spiracles (Figs. 17.4 and 17.5) are useful for determining species and instar of muscid larvae. Paired spiracles occur on the prothorax and at the end of the abdomen. Prothoracic spiracles are absent on first instars, whereas they are present on second and third instars. The shape and number of spiracular tubercles (= spiracular lobes) vary considerably in different species.

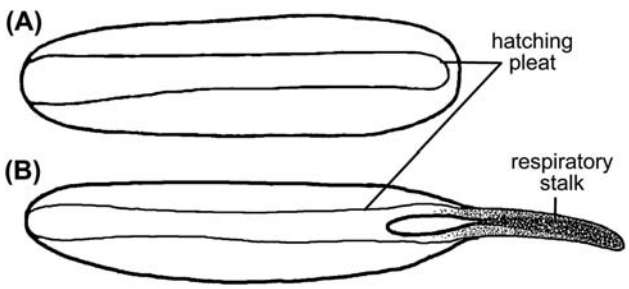


FIGURE 17.2 Eggs of muscid flies, showing hatching pleat. (A) House fly (*Musca domestica*). (B) Face fly (*Musca autumnalis*), with distinctive respiratory stalk.

Structures associated with the paired caudal spiracles on the abdomen are of greatest taxonomic value. Each spiracular plate (Fig. 17.4) consists of a peritreme forming the plate's perimeter, two or three slits that are openings for gas exchange, and a scar that is a remnant from a previous molt. First and second instars have two slits, whereas third instars have three. An exception is the horn fly, in which both the second and third instars have three slits. Peritreme shape, position of the scar, and shape and orientation of the slits are all useful in identifying muscid larvae (Fig. 17.5).

The muscid pupa (Fig. 17.1C) occurs in a case, the **puparium**, which forms during a process called **pupariation**, involving contraction and hardening of the third-instar integument within. A space forms around the pupa during subsequent apolysis, or separation of the new pupal integument inside the puparium. The larval cephalopharyngeal skeleton remains attached inside the puparium's cephalic cap, and prothoracic and posterior spiracles remain embedded in the wall of the puparium. Thus, when an adult specimen has been associated with its puparium, distinguishing traits of the adult, the puparium, and the mature larva can be discerned and used to identify the species from a specimen in any of the three stages.

Adult muscid flies (Fig. 17.1) are 4–12 mm long, with wings longer than the abdomen. Integument colors vary from brownish gray to black, often with dark longitudinal stripes on the thorax, called vittae, and dark spots or blotches on the abdomen. The head (Fig. 17.6) has three ocelli and a prominent pair of compound eyes, which in males are holoptic (i.e., nearly meeting at the dorsal

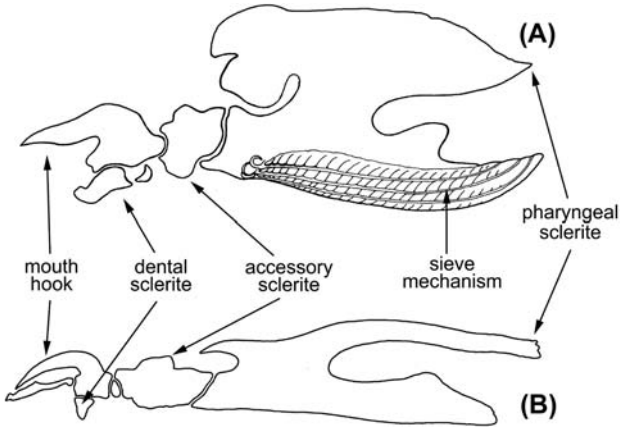


FIGURE 17.3 Cephalopharyngeal skeletons of muscid fly larvae. (A) Filter-feeding larva, characterized by sievelike structures. (B) Predatory larva, with pair of strong mouth hooks. *Redrawn from Skidmore (1985).*

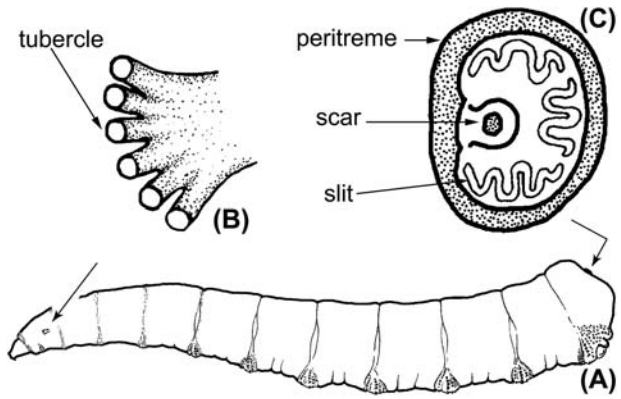


FIGURE 17.4 Respiratory spiracles of house fly (*Musca domestica*), third-instar larva. (A) Larva, showing location of anterior pair of spiracles on first thoracic segment and posterior pair of spiracular plates on caudal segment (*arrows*). (B) Anterior spiracle, enlargement showing multiple openings to trachea on individual tubercles. (C) Posterior spiracular plate, showing sclerotized ring (peritreme) surrounding spiracular area, three sinuous spiracular slits, and scar left by previous molt. *Redrawn from Skidmore (1985).*

midline); in females they are dichoptic, or more widely separated. Each antenna consists of a scape, pedicel, and arista. The arista arises from the pedicel as an undivided flagellum homologous to the flagellum of nematocerous flies. In most muscid flies, the arista is a single hair with fine setae along its shaft. The ptilinal suture, encircling the bases of the antennae, is a remnant of the **ptilinum**, an eversible sac used by the emerging adult to break open and exit through the cephalic cap of the puparium. In most muscid flies, a pair of strong bristles called oral vibrissae project ventrally from the lower edge of the face toward the mouthparts.

Mouthparts of adults vary considerably among species. Generally they consist of a proboscis with a basal rostrum, a slender haustellum, and a terminal labellum (Fig. 17.6).

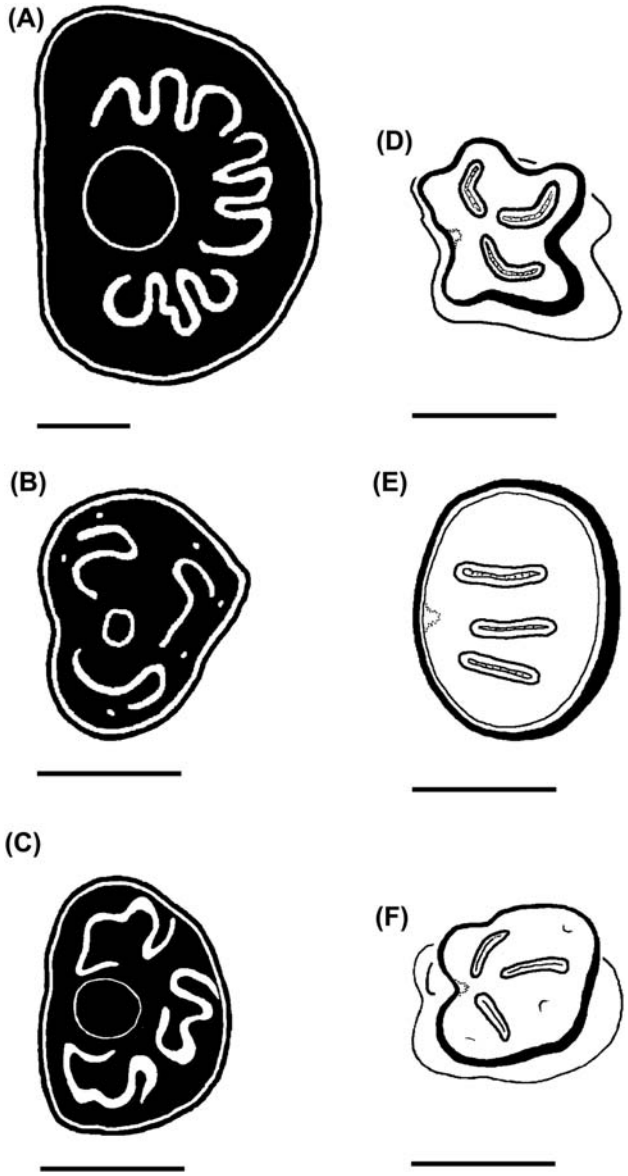


FIGURE 17.5 Caudal spiracular plates of third-instar larvae of six important muscid flies; only right plate of each pair is shown. (A) Face fly (*Musca autumnalis*). (B) Stable fly (*Stomoxys calcitrans*). (C) Horn fly (*Haematobia irritans*). (D) False stable fly (*Muscina stabulans*). (E) Black garbage fly (*Hydrotaea ignava*). (F) *Hydrotaea* sp. Scale bar, 0.2 mm. *Redrawn from Skidmore (1985).*

The maxillary palps arise from the rostrum and appear to be 1-segmented. The haustellum is formed by three structures held in union (Fig. 17.6C): an anterior labrum, a slender hypopharynx, and a posterior labium. The labium encloses both the labrum and hypopharynx and terminates in a two-lobed labellum. Saliva flows from the salivary glands through the salivary canal in the hypopharynx that terminates in the prestomum, or preoral cavity, at the center of the labellum. A cibarial pump inside the head creates suction that draws liquids through the prestomum and up through the food canal between the labrum and labium.

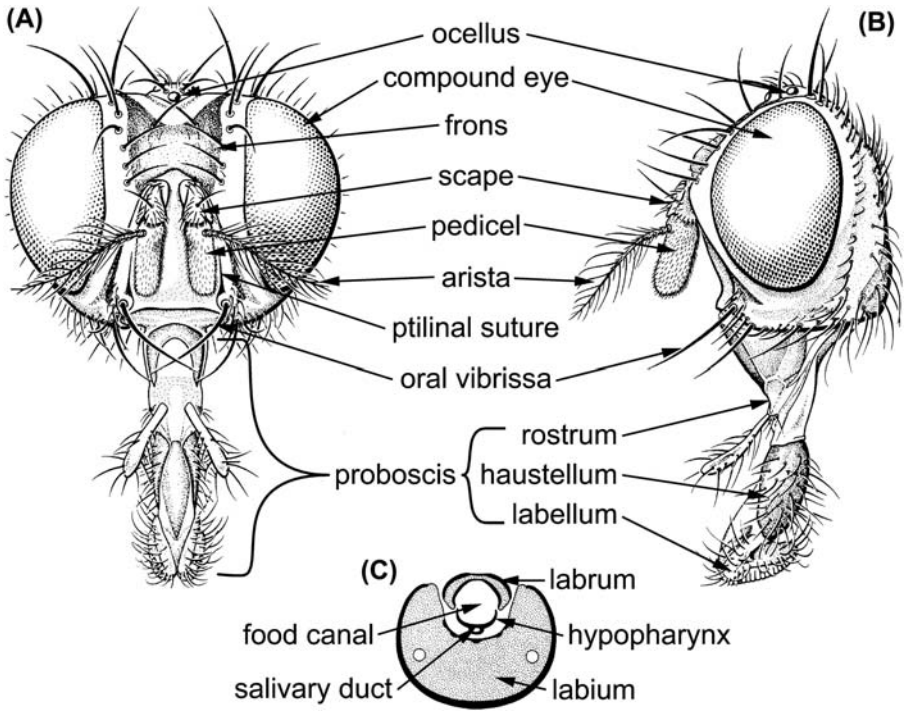


FIGURE 17.6 Head and mouthparts of a nonbiting muscid fly, adult. (A) Anterior view. (B) Lateral view. (C) Cross section of haustellum, showing relationship of individual mouthparts, food canal, and salivary duct. *Adapted from original drawings by R. Idema, McAlpine et al. 1981. Reproduced with permission of Minister of Public Works and Government Services, Canada.*

Important structural differences distinguish the labellae of nonbiting and biting muscid flies. Among nonbiting species, the labellum is an enlarged fleshy, two-lobed structure (Figs. 17.6 and 17.7A). On the mesal surface of each lobe are **pseudotracheae**, rows of fine setae used to scrape food and direct fluid toward the prestomum. The labellum also houses mechanoreceptors, chemoreceptors, and prestomal teeth. These teeth may be short, or elongate and dentate in some *Musca* and *Hydrotaea*. In contrast, mouthparts of biting species (Figs. 17.7B and 17.8) are adapted for piercing or tearing into host skin to obtain blood. Their labellar lobes are comparatively small, but their prestomal teeth are sharp, sclerotized, and greatly enlarged.

Other morphological characters of the thorax and wings are typical of muscid flies. On the thorax (Fig. 17.9), the

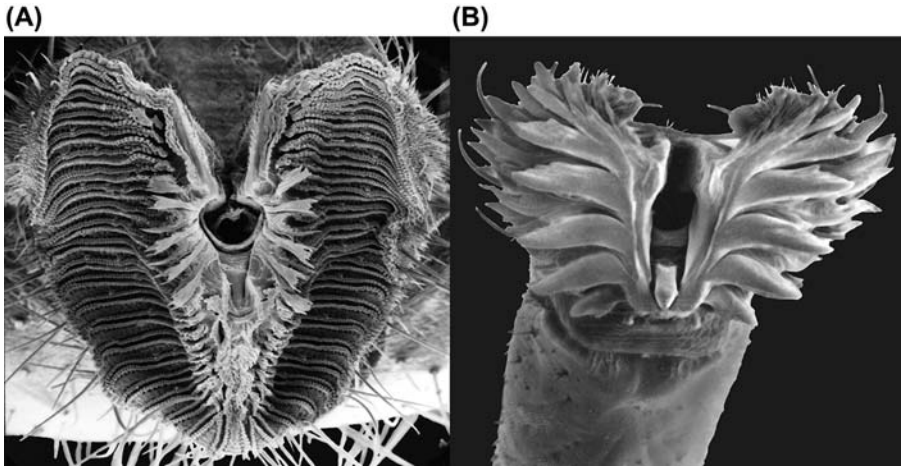


FIGURE 17.7 Scanning electron micrographs of everted pair of labellae at tip of proboscis of two muscid flies, adults. (A) Nonbiting fly (face fly, *Musca autumnalis*), showing parallel arrangement of feeding channels (pseudotracheae) and relatively small prestomal teeth surrounding opening to food canal (prestomum). (B) Biting fly (stable fly, *Stomoxys calcitrans*), lacking pseudotracheae and with enlarged prestomal teeth used to cut through skin of hosts to feed. *Courtesy of Alberto B. Broce.*

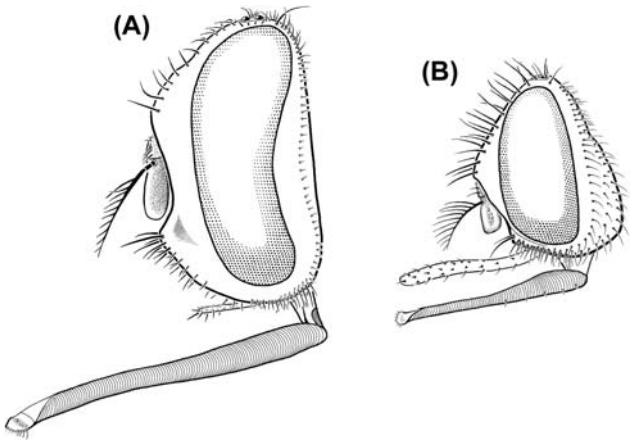


FIGURE 17.8 Heads of two muscid flies with biting mouthparts; in both cases mouthparts are held in a horizontal position beneath head when not feeding. (A) Stable fly (*Stomoxys calcitrans*). (B) Horn fly (*Haematobia irritans*). *Redrawn from Edwards et al. (1939). © The Natural History Museum, London.*

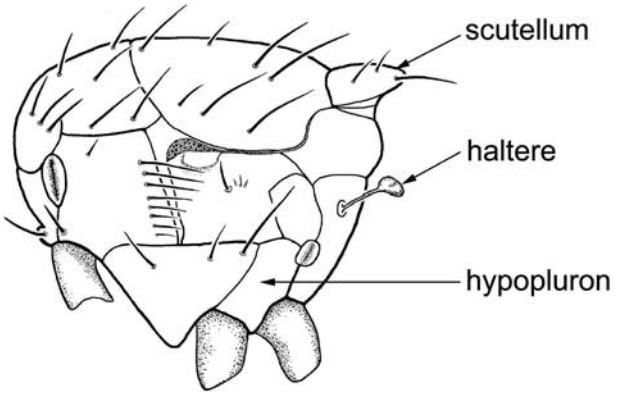


FIGURE 17.9 Lateral view of thorax of adult muscid fly, with wings removed; anterior to left; showing typical sclerites, scutellum, hindwing reduced to form a haltere, and a taxonomically important sclerite, the hypopleuron. *Redrawn from Borror et al. (1989), with permission of Wadsworth, an imprint of the Wadsworth Group, a division of Thomson Learning.*

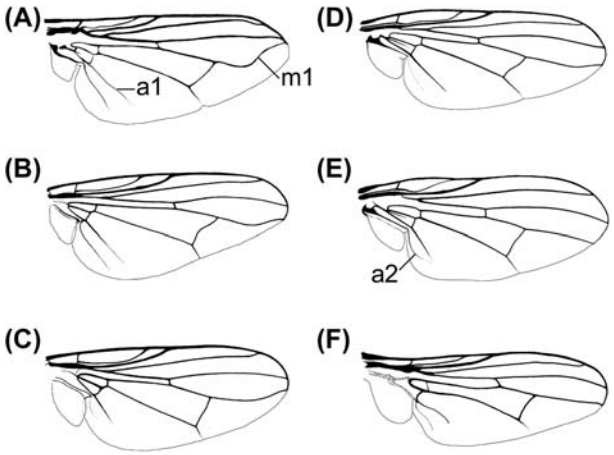


FIGURE 17.10 Wings and wing venation of important muscid flies; right wing shown in each case. (A) House fly (*Musca domestica*). (B) Stable fly (*Stomoxys calcitrans*). (C) Horn fly (*Haematobia irritans*). (D) False stable fly (*Muscina stabulans*). (E) Little house fly (*Fannia canicularis*). (F) Black garbage fly (*Hydrotaea ignava*). a1 and a2, anal wing veins 1 and 2; m1, medial wing vein 1. *(A), (B), and (F) redrawn from Axtell (1986), all rights reserved, copyright © Novartis. (C) Redrawn from Lane and Crosskey (1993) © The Natural History Museum, London. (D) and (E) redrawn from McAlpine et al. (1987), with permission of Minister of Public Works and Government Services, Canada.*

hypopleuron is bare, entirely lacking bristles and setae. The first anal wing vein (vein A1, Fig. 17.10), including a faint trailing fold if present, vanishes before it reaches the wing margin. The combination of these characters, along with aristate antennae, a ptilinal suture, and usually robust oral vibrissae distinguish members of the Muscidae from all other flies.

The abdomen is reduced to five visible segments in both sexes, with succeeding segments 6–12 modified into eversible reproductive terminalia. The male possesses an aedeagus, or intromittent organ, which when at rest is rotated 180 degrees and is partially enclosed in a genital pouch. In the female, the terminalia are modified into a tubular, telescoping ovipositor that is extended to lay eggs. Chemoreceptors and mechanoreceptors occur on the terminalia, and also on the antennae, labellum, and tarsi.

The digestive system of adult muscid flies (Fig. 17.11) is much like that of other dipterans. Saliva is produced in the salivary glands, which extend posteriorly from the head into the abdomen. Ingested foods that are nutrient rich, such as blood and dung fluids, are routed via the proventriculus of the foregut to the midgut where digestion occurs. Fluids that are dilute, such as plant nectar and milk, are shunted for temporary storage to the diverticulum, typically a ventral, saclike extension of the esophagus. Contents of the diverticulum are regurgitated through the proboscis onto the feeding substrate. Waste products and undigested food ultimately pass through the hindgut and out the anus as drops of liquid fly feces. The brown fly specks that collect on feeding and resting substrates consist of droplets of two substances: fly vomit and feces.

LIFE HISTORY

All the important North American muscid species are oviparous, meaning that females deposit fertilized eggs in the environment before they hatch. Some members of the Muscidae in the Old World are larviparous. Oviposition substrates vary among the different species. Filth flies deposit eggs in organic debris that is wet enough to support aerobic microbial fermentation. Common substrates are human feces and garbage and decomposing organic matter such as rotting algal mats, piles of lawn clippings, and

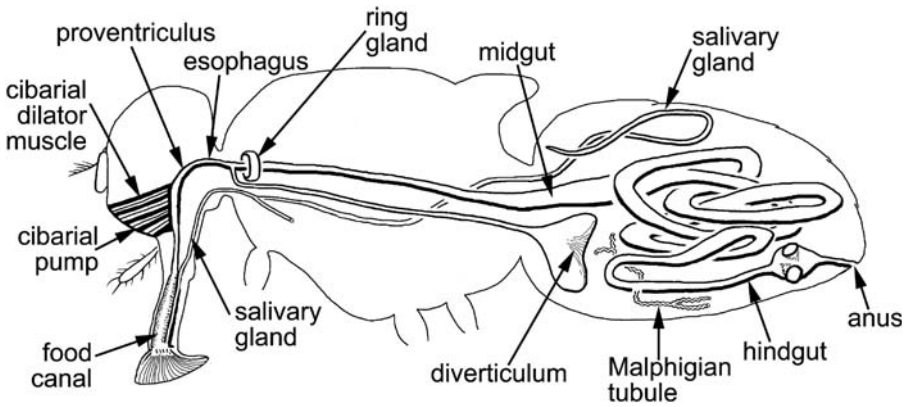


FIGURE 17.11 Digestive system of adult muscid fly; left salivary gland and distal ends of Malphigian tubules omitted for clarity. *Redrawn from Patton (1929).*

food-processing wastes. Where livestock and poultry are confined, attractive substrates include manure (a mixture of aging feces and urine), soiled bedding (bedding + manure + feed), and wet, rotting feeds such as hay, silage, and grain. Filth flies commonly exploit the kinds of wastes that accumulate around human habitations and animal-confinement facilities.

In contrast to filth flies, important muscid dung flies and sweat flies lay their eggs in a much narrower range of substrates. Dung flies oviposit on or into cattle dung pats within minutes to a day or so after an animal defecates. Sweat flies oviposit in plant litter and decomposing dung in grasslands and forests.

Larvae of all of the important muscid flies burrow, feed, and develop in their respective ovipositional substrates. However, larval feeding differs among species and even among instars of the same species. Most muscid flies are saprophages, feeding by filtering bacteria, yeasts, and other small organic particles suspended in their semiliquid habitat. All three instars of the house fly, stable fly, *Fannia* spp., horn fly, and face fly are saprophages. In contrast, the third instars of garbage flies (*Muscina* spp.) and the sweat flies are facultative predators. These larvae can mature as saprophages, but they will switch to predation and consume other soft-bodied insects if available. These facultative predators contribute to natural biological control of other flies that occur in the same habitats.

Once mature, third-instar muscid larvae cease feeding, defecate to empty their alimentary tracts, and enter a wandering phase before they pupate. Depending on the species and substrate moisture, they may disperse to adjacent drier locations or pupate directly in their larval medium.

Developmental times from egg to adult among muscid flies range from 1 to 6 weeks during the summer (Table 17.2). Most of the species develop fastest at temperatures of 27–32°C, and all virtually cease activity at temperatures below 10°C. Sustained temperatures beyond these limits are usually lethal, with exceptions of cold-tolerant species that overwinter as larvae or pupae. Developmental times also can be affected by the food supply (e.g., when crowded or otherwise starved, filth flies will delay pupation to achieve a minimum size). The dung flies have a different strategy. Their larvae sacrifice size and will pupate earlier, resulting in adults that are smaller and less fecund than their better nourished counterparts. Regardless of larval conditions, emerging adults typically occur in an equal sex ratio (1:1).

The reproductive capacity of a muscid fly population is determined in part by fecundity, the number of eggs that the average female produces per batch. The house fly and other muscid filth flies are the most fecund, whereas dung flies produce fewer eggs per batch (Table 17.2). Reproductive capacity is also determined by length of the gonotrophic cycle (i.e., the number of days a female requires to develop and deposit a batch of eggs), and by longevity, which governs how long she will live and how many cycles she can complete. Under summer conditions, muscid flies can develop a new batch of eggs every 2–5 days. The average female probably lives only long enough to produce one or two batches, although longevity is difficult to measure under field conditions.

BEHAVIOR AND ECOLOGY

Adult muscid flies emerge with little stored energy and nutrients, so they must find water, salts, carbohydrates, and proteins if they are to survive and reproduce. The nonbiting species obtain sugars from off-host sources such as plant nectaries and honeydew from sap-sucking aphids and scale insects. Biting species obtain the bulk of their proteins from blood, serum, saliva, mucus, and lachrymal secretions. Both sexes of the biting horn fly and stable fly obtain nearly all their nutrients from blood.

The feeding behavior of a nonbiting fly differs from that of biting species. A nonbiting fly opens its labellum and presses it against the substrate. If the food is not liquid, the fly releases enzyme-laden saliva and repetitively opens and closes the labellum to scrub and dissolve the food into the saliva. The suspension is then drawn along the pseudotracheae into the food canal. Feeding by nonbiting flies can be characterized as a process of salivating, scrubbing, and sucking; they cannot physically penetrate skin. When a biting species feeds (Fig. 17.12), it presses its proboscis against skin and rapidly opens and closes the labellum, directing the prestomal teeth in a downward and outward rasping motion. Once the skin is penetrated, the teeth anchor the proboscis while blood flows into the subsurface lesion and into the food canal.

All the important muscid flies are **anautogenous**, meaning that females require protein to complete their first gonotrophic cycle. Protein is the substrate of yolk synthesis and egg maturation. In the nonbiting species, eggs mature in synchrony 2−5 days after protein is first obtained. Development of a subsequent batch is arrested hormonally until the preceding batch has been laid. There are corresponding cycles of attraction to different substrates, first to sources of protein and then to oviposition sites as eggs mature. In the horn fly and stable fly, eggs develop asynchronously, such that feeding and oviposition are distributed more evenly in time.

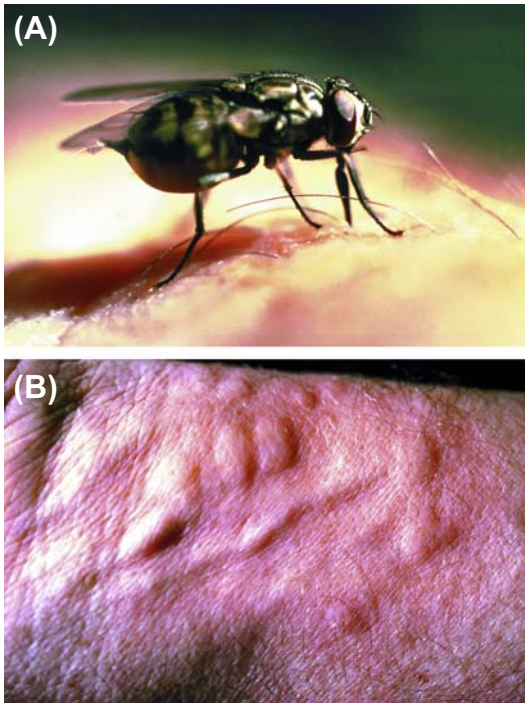


FIGURE 17.12 Stable fly (*Stomoxys calcitrans*) feeding on human. (A) Fully fed female, abdomen distended with blood. (B) Resultant bite reaction in form of welts. *Courtesy of Elton J. Hansens.*

Behavior associated with **mating** differs among species. Males of *Fannia* spp. and some sweat flies and garbage flies hover in swarms, usually in locations shaded by trees or roofs and eves of buildings. These males are attracted to females that fly into the swarms. Once coupled, a mating pair will fall to the ground and complete copulation. Males of the other muscid flies do not form swarms. Instead, they generally perch or rest in sunny locations on substrates such as tree trunks, fence posts and rocks, where the males intercept passing females. Females of the important muscid flies typically store enough sperm from a single mating to fertilize all the eggs they can produce during the remainder of their lives.

Activities and locations of adult muscid flies vary markedly with time of day. All important species are active during daylight hours, and almost all are inactive at night. Activities include flying, host location, feeding, mating, and ovipositing. Sight and olfaction are used to locate hosts and oviposition substrates. Most muscid flies are exophilic, being reluctant to enter buildings. A few species are more endophilic and will enter buildings. Species that feed on animals may be on a host as briefly as a few minutes, just long enough to obtain available foods. The flies leave their hosts when replete, and rest in the surrounding environment while digestion proceeds. Because feeding times are much shorter than digestion times, the adults on a host at any instant are likely to be only a small fraction of the adults present in the host's environment.

Muscid flies tend to prefer certain daytime resting sites, in part according to their needs for thermoregulation. They rest in sunny sites when the air temperature is below about 20°C and in shady sites when temperatures exceed about 30°C.

The flight range of muscid flies is extensive. Detectable numbers of all the important North American species have been collected more than 5 km from known or presumed points of origin. Large numbers of stable flies can appear on beaches 10 or more miles from the nearest likely breeding sites.

The seasonal patterns in abundance and age structure of muscid adult vary among species, years, and locations. In localities with cold winters, populations are typically restricted to a distinct breeding season—the warmer, wetter months of spring, summer and autumn. In these cases, populations grow to a single annual peak of abundance, normally in early autumn. A notable exception is the house fly, which can breed continuously in heated buildings. In warmer climates, the breeding season for most species is longer, and may be continuous year-round. Adults of the house fly, *Fannia* spp., and the horn fly, for example, reproduce throughout the year in the southeastern United States and southern California. Densities of adults have two seasonal peaks, with growth phases in spring and autumn separated by periods of decline during summer and winter.

Most muscid flies of medical-veterinary importance are multivoltine, developing through two or more generations per breeding season (Table 17.2). These generations usually overlap, with continuous recruitment of new adults; eggs, larvae, pupae, and adults of all ages are present simultaneously throughout most of the breeding season. Population growth within the breeding season is influenced by availability of breeding media, by weather and its effects on survival of immature stages, and the fly reproductive rate. Survival of larvae is enhanced if their breeding habitat remains wet enough to support filter feeding, yet dry enough to allow aerobic respiration. Substrate moisture is critical because the saprophagous larvae feed by filtering particles suspended in their medium. Suitable moisture levels are usually 30%–75%.

Muscid flies overwinter in different ways (Table 17.2). The house fly and stable fly breed continuously in frost-free southern regions of North America. Breeding by these flies is restricted to warmer months in more northern latitudes, because the flies lack a stage that can endure temperatures below freezing for much more than a day. It was once thought that these two filth flies, lacking a freeze-tolerant life stage, died out each winter in temperate latitudes and then repopulated each spring from milder regions. However, it is now known that local populations can survive winters in protected, semiheated substrates associated with humans and livestock. Regional repopulation does occur, however, with the bushfly (*M. vetustissima*) in Australia, where adults disperse southward from more northerly latitudes that remain warm during winter.

Other muscid flies of medical-veterinary importance overwinter in diapause, a state of developmental arrest typically associated with a tolerance for freezing. The face fly and *Muscina* spp. overwinter as adults. In autumn, these flies enter hibernacula (e.g., under bark of dead trees and siding on buildings) and emerge the next spring to begin reproduction. The horn fly, in contrast, overwinters in temperate regions as a diapausing pupa. Garbage flies, *Fannia* spp., and sweat flies are thought to overwinter as larvae, but further study is needed to determine if they exhibit a true diapause, or are in a simpler state of cold-tolerant quiescence.

SPECIES OF MEDICAL-VETERINARY IMPORTANCE

Adults and larvae of some important muscid flies can be identified tentatively from external characters and from features of their behavior, habitat, and geographic location.

House Fly (*Musca domestica*)

This nonbiting filth fly occurs on all continents except Antarctica. It is native to the Afrotropical and Oriental regions, and was probably introduced into the Americas by Europeans during colonial times. Adults are gray and black flies, 6–9 mm long, with four black vittae on an otherwise gray thorax (Figs. 17.1 and 17.13A). The wing has a sharp forward bend in vein M1 (Fig. 17.10A). The abdomens of typical females are checkered gray and black at the dorsal midline, and creamy yellow on the sides, which in North America is sufficient to distinguish this species from the face fly. Larvae have large caudal spiracles that resemble back-to-back "D"s, and the slits are sinuous (Fig. 17.4B).

Immatures can be found in a wide variety of decaying organic substrates. Major breeding sites include human garbage dumps, open privies, livestock manure, soiled bedding, poultry manure, and fruit and vegetable processing wastes. Breeding continues year-round in tropical and

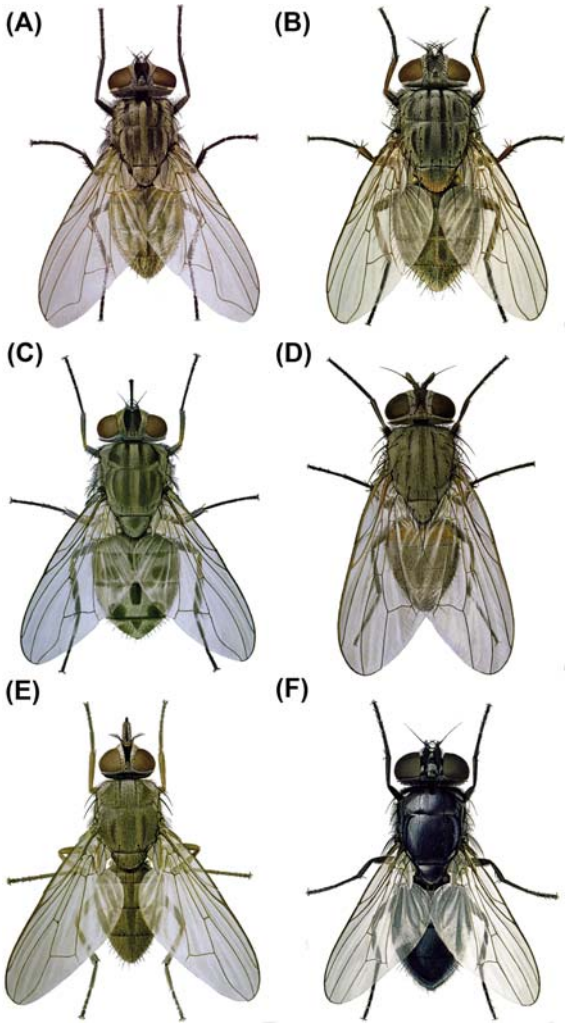


FIGURE 17.13 Muscid flies, adult females. (A) House fly (*Musca domestica*). (B) False stable fly (*Muscina stabulans*). (C) Stable fly (*Stomoxys calcitrans*). (D) Little house fly (*Fannia canicularis*). (E) Horn fly (*Haematobia irritans*). (F) Black garbage fly (*Hydrotaea ignava*). *Original drawings by Fritz Gregor, and published in Greenburg, 1971, reprinted by permission of Princeton University Press.*

subtropical regions, but is interrupted by winter in temperate regions. From a public health standpoint, the house fly is probably most significant as a nuisance and potential vector of enteric pathogens. Although it can be quite abundant where livestock, poultry, and companion animals are housed, its direct, adverse effects on animal health are minor.

Bazaar Fly (*Musca sorbens*)

This nonbiting filth fly is the most abundant synanthropic muscid fly in many parts of the Afrotropical, Oriental, and Pacific regions. It was introduced through commerce into Hawaii and would flourish if introduced elsewhere in tropical latitudes of the Americas. Greenberg's (1971) key provides characters to distinguish the bazaar fly from other *Musca* species in the Afrotropical and Oriental regions.

The species recognized as *M. sorbens* before 1970 apparently consists of a complex of at least three species that are partially distinguishable by the ratio of the width of the male's frons (area between compound eyes) to the width of the head (including eyes). The "broad-frons" form is the bazaar fly (*M. sorbens*), which occurs from Africa east through the Orient and on many Pacific islands. One of the "narrow-frons" form is the bushfly (*M. vetustissima*), a distinct species that occurs in Australia and Papua New Guinea. The second "narrow-frons" form is *Musca biseta* and coexists with the bazaar fly in Africa and eastward. Further study is needed to resolve distinctions between *M. biseta* and the bushfly in southern Asia and the Orient (Pont, 1991).

Adult bazaar flies are strongly exophilic, being far less inclined than the house fly to enter buildings. Larvae have been recorded in unburied human stools and dog feces, and less commonly in feces of other animals, in carrion, and in garbage. The bazaar fly is important to public health as a mechanical vector of enteric pathogens, but is of little or no importance to the health of domesticated animals.

Bush Fly (*Musca vetustissima*)

This nonbiting dung fly occurs in Australia, where it is a major nuisance to humans and livestock. It is closely related to the bazaar fly, and keys out as *M. sorbens* in Greenberg (1971). Adults are attracted to large mammals as sources of fluids for nourishment and feces for oviposition. Several authors have speculated that the bush fly was originally associated with aboriginal encampments and that its abundance increased when domesticated cattle were imported. Larvae have been recorded from the feces of a wide variety of large mammals, but in nature cattle dung pats are overwhelmingly the most productive. Breeding is continuous in subtropical Australia, and southward migrations serve to repopulate temperate Australia and Tasmania each spring (Hughes, 1977).

Face Fly (*Musca autumnalis*)

This nonbiting dung fly is native to Europe and central Asia and was introduced into North America before 1952 (Krafsur and Moon, 1997). It occurs in all southern Canadian provinces and in the United States north of Arizona–Georgia (35°N). The adult resembles the house fly (Figs. 17.1 and 17.13A), is 6–10 mm long, and has four black vittae on an otherwise gray thorax and a sharp forward bend in wing vein M1 (Fig. 17.10A). The male's abdomen has a distinct, black longitudinal band along the midline and bright yellow sides. The female has a characteristic yellow patch on the ventrolateral aspect of the first visible abdominal segment; the remaining segments are gray-black to the ventral midline. The egg has a distinct brown-black respiratory stalk (Fig. 17.2B). Mature larvae are bright yellow with black, D-shaped spiracular plates (Fig. 17.5A), and puparia are white due to calcification.

During the fly breeding season, adult face flies occur around grazing cattle and horses. Their larvae develop exclusively in fresh cattle dung pats. In autumn, newly emerged adults enter diapause, aggregate on buildings (sometimes in the thousands), often getting behind siding, in wall voids, in attics, and occasionally into interior rooms. The face fly was first recognized as a nuisance in European households due to its overwintering habits. It became recognized as a pest of cattle and horses after it was introduced into North America.

Cluster Fly (*Pollenia rudis*)

The cluster fly is discussed here because it often occurs along with the face fly in household infestations. This calliphorid fly is native to Europe and North Africa and was introduced into North America, where it now occurs in all southern provinces of Canada and throughout the United States. The adult is 7–9 mm long; the abdomen is completely black with silvered checking; and there are crinkly golden hairs on the head and thorax. Cluster fly larvae are internal parasites of earthworms (*Allolobophora* spp., Lumbricidae), also introduced from Europe, and produce two to four generations per year. Adults can be a nuisance in households, but they do not affect domesticated animals.

Stable Fly (*Stomoxys calcitrans*)

This biting filth fly (Figs. 17.12 and 17.13C) is native to Africa, Europe, Asia, and the Orient and was probably introduced into the Americas and Australia during colonial times. The stable fly also is commonly known as the **beach fly** because of outbreaks on recreational beaches, **dog fly** because it pesters dogs, and **lawn-mower fly** because larvae have been found in damp, matted grass on the

undersides of lawn mowers. It also is misleadingly called the **biting house fly** because of its superficial resemblance to the house fly.

The adult is 5−7 mm long and has seven circular black spots on an otherwise gray abdomen (Fig. 17.13C) and a forward projecting piercing-sucking proboscis, with maxillary palps less than one-fifth the length of the haustellum (Fig. 17.8A). Larvae and pupae have uniquely shaped, subhexagonal posterior spiracular plates that are far apart; the horizontal space between them is greater than twice a plate's width (Fig. 17.5B). Larvae occur in moist decaying fibrous substrates such as straw bedding, wet hay, algal mats, and wet grass clippings. Other larval habitats include accumulations of manure from dairy and beef cattle, mixtures of soil and partially composted bedding and animal manure, and byproducts of crop processing such as peanut hulls, beet pulp, sugarcane bagasse, and pineapple. Breeding is continuous in tropical and subtropical climates. In temperate regions, the species is thought to overwinter as immatures wherever larval substrates do not freeze. Stable flies are important to public health because they will attack and annoy people, but they are much more important from a veterinary perspective.

Horn Fly (*Haematobia irritans irritans*) and Buffalo Fly (*Haematobia irritans exigua*)

These biting dung flies were once recognized as two separate species: the horn fly (*H. irritans*) and the buffalo fly (*H. exigua*). They are now considered subspecies of *H. irritans*, based mainly on subtle morphological differences and allopatric distributions (Zumpt, 1973). The horn fly is native to northern Africa, Europe, and central Asia and was introduced into North America from Europe in the middle 1880s. It has since spread to all cattle-producing regions in North and South America, including Hawaii. The buffalo fly is native to southern Asia, the Orient, Indonesia, and several Pacific islands. It spread through commerce to New Guinea and Australia before 1840. It is possible that the two subspecies intergrade in parts of Asia.

The adult horn fly (Fig. 17.13E) is 3−5 mm long and has a piercing-sucking proboscis (Fig. 17.8B). The maxillary palps are held appressed to the haustellum and are almost as long as the haustellum. Wing vein M1 is gently curved (Fig. 17.10C). Adults of both subspecies are specific to cattle, bison, and water buffalo; aberrant hosts include horses and other large mammals. The flies occur mainly on the withers, back, and sides but will move to the belly and legs when weather is hot. Once a host is located, the adult remains on its host almost continuously, except when disturbed or laying eggs. Horn flies and buffalo flies feed and oviposit during day and night. Females of both flies lay their eggs exclusively under edges of dung pats, usually within minutes of defecation. For unknown reasons, dung from horses, sheep, and other large mammals is unsuitable. Horn flies occur in far greater numbers on grazing cattle than on animals confined in drylots or indoors. Reproduction is continuous and populations are multivoltine. The horn fly overwinters as a diapausing pupa in temperate latitudes. Neither subspecies poses any threat to human health, but both species are serious economic pests of grazing beef and dairy cattle.

False Stable Fly (*Muscina stabulans*) and Its Relatives

These nonbiting filth flies include the false stable fly, which has been spread worldwide through commerce, and another 10 species that occur mainly in the Holarctic region. The name *Muscina assimilis*, used widely in older literature for a relative of the false stable fly, has been relegated to a synonym of *M. levida* (Skidmore, 1985). The false stable fly (Fig. 17.13B) and its relatives are stout flies, 8−12 mm long, with brown-black bodies and a rounded bend in wing vein M1 (Fig. 17.10D). The tip of the scutellum of the false stable fly is red-orange. The posterior spiracular plates are roughly circular; they are separated by one plate's width, and the slits are bowed and arranged radially (Fig. 17.5D). Third-instar larvae are facultatively predatory, and adults overwinter in a prereproductive diapause. These species can affect public health, but they are not thought to affect the health of domesticated animals.

Little House Fly (*Fannia canicularis*) and Its Relatives

There are about 100 species of these nonbiting filth flies in North America (Chillcott, 1961), and additional ones in Latin America, Africa, Europe, and Asia. The little house fly (*F. canicularis*) and ***latrine fly*** (*F. scalaris*) have spread by commerce throughout the world. *Fannia* species are 5−8 mm long, with dark thoraces and abdomens variously marked with yellow (Fig. 17.13D). The arista lacks setae and the second anal vein (A2) curves toward the first anal vein (A1) (Fig. 17.10E). Larvae and puparia have characteristic lateral and dorsal processes (Fig. 17.14) whose function is unknown. Males form mating swarms in shady

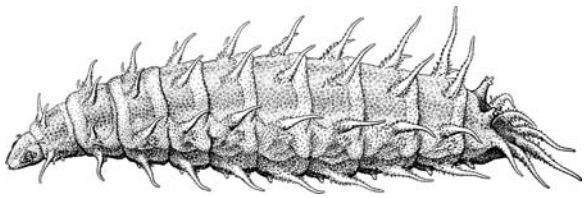


FIGURE 17.14 Little house fly (*Fannia canicularis*), third-instar larva. *From McAlpine et al. (1981). Reproduced with permission of Minister of Public Works and Government Services, Canada.*

locations, and it is this swarming behavior that most often brings them into contact with people. The little house fly is probably the most endophilic and commonly encountered species of this genus in North America. The latrine fly is more exophilic. Although these flies are most noticeable where domesticated and zoo animals are confined, they are not otherwise important to public and veterinary health.

Garbage Flies (*Hydrotaea* spp.)

There are seven known species of garbage flies, with at least one species in every biogeographic region. This group of nonbiting filth flies, once placed in the genus *Ophyra*, has been merged into *Hydrotaea* (Huckett and Vockeroth 1987). Accordingly, the scientific names of the common species have changed. The **black garbage fly**, known in older literature as *Ophyra leucostoma*, is now named *Hydrotaea ignava*. It is native to the Old World and has been introduced into North America (Skidmore, 1985). The **black dump fly**, formerly *Ophyra aenescens*, is now *Hydrotaea aenescens*. It is native to the New World, occurs in the eastern Pacific Islands including Hawaii, and has been introduced into Europe.

Garbage flies are 4−7 mm long with shiny black thoraces and abdomens (Fig. 17.13F). Wing vein M1 is virtually straight (Fig. 17.10F). Posterior spiracles of mature larvae and puparia are roughly circular; they are separated by less than a plate's width, and have slightly curved slits that barely diverge from a faint scar (Fig. 17.5E). Adults are strongly exophilic. Larvae have been recorded in a great variety of filthy substrates, including carrion. Third instars are facultative predators, and will consume larvae of other flies that cohabit their breeding medium. These filth flies pose a modest threat to public health, but they are not known to harm domesticated animals.

Sweat Flies (*Hydrotaea* spp.)

About 50 species of sweat flies occur in the Palearctic region, and fewer species occur in the remaining biogeographic regions. Sweat flies are gray to black, 3−8 mm long, with an arista that lacks setae. Although females have no simple distinguishing characters, males have a ventral notch or depression at the distal end of the fore femur. Third-instar larvae are facultative predators. Their spiracular plates are stalked, with radially arranged slits (Fig. 17.5F).

Females of six of the 24 North American species, including *Hydrotaea meteorica* and *H. scambus*, are persistent in their attempts to imbibe perspiration and secretions from the eyes, nostrils, lips, and other parts of mammalian hosts. The remaining North American species apparently are not attracted to animals (Huckett, 1954). In Europe, the **sheep headfly** (*H. irritans*) is a primary pest of sheep, cattle, and deer.

Bird Nest Parasites (*Passeromyia* spp., *Philornis* spp., and *Protocalliphora* spp.)

Five species of *Passeromyia* occur in the Old World (Pont, 1975), and about 50 species of *Philornis* occur in the Neotropics and north into the United States (Florida and southern Texas) (Couri, 1999). *Passeromyia* and *Philornis* are sister taxa whose larvae pupate within cocoons in nests of host birds. *Protocalliphora* (Calliphoridae) consists of 65 species in the Old World and 25 species in North America (Sabrosky et al., 1989). Larvae of *Protocalliphora* do not form cocoons.

Adults in these three genera are free-living flies that are believed to feed on fruits and other sources of plant sugars. Larvae and pupae inhabit nests of birds whose chicks are immature and helpless (altricial), such as hawks, falcons, thrushes, and other passerine birds. A few species feed on feces (i.e., are coprophagous), but most are parasites that feed on chick blood (i.e., are sanguiniverous). Sanguinivory is believed to have evolved from coprophagy.

Females of the parasitic species lay eggs on or nearby chicks. Larvae either attack chicks as intermittent ectoparasites, or they burrow into the chicks' skin and develop as endoparasites in their hosts' nares, auditory canals, or skin (Fig. 17.22). Mature larvae spin cocoons and then pupariate in the nest.

PUBLIC HEALTH IMPORTANCE

Muscid flies affect people most frequently as nuisances, occasionally as vectors of pathogenic organisms, and rarely as agents of human myiasis. The cosmopolitan house fly and stable fly are of greatest medical significance. Other notable examples are the bazaar fly in Africa, Asia, and Pacific islands including Hawaii; bushfly in Australia; and two relatives of stable fly, *Stomoxys nigra* and *S. sitiens*, in Africa and Asia.

Filth flies pose particular risks as mechanical vectors of pathogens that cause enteric disease in humans. Among the 2.39 million cases of notifiable infectious diseases reported in 2014 to the US Centers for Disease Control and Prevention, approximately 103,000 (4.3%) were enteric infections causing diarrhea or dysentery. These diseases arise from direct or indirect fecal contamination of food and water, either at points of consumption and preparation, or earlier at points of production and distribution. Globally, the World Health Organization reports that diarrhea and dysentery account for more childhood deaths and morbidity than any other infectious diseases.

Enteric diseases are caused by certain bacteria, viruses, and protozoa. The bacteria include *Escherichia coli*,

Salmonella spp., and *Shigella* spp.; the viruses include Coxsackievirus, Enterovirus 72 (= Hepatitis A), and Enteric cytopathogenic human orphan (= ECHO) virus; and the protozoa include *Chilomastix*, *Cryptosporidium*, *Entamoebae*, and *Giardia* spp. Infections range in severity from benign to fatal, being most severe among children, the elderly, and the infirm. Common sources of enteric pathogens are food and water contaminated with feces from infected people or animals or indirectly via hands, utensils, and filth flies.

Greenberg (1971, 1973) summarized the extensive literature on pathogens associated with muscid flies. Evidence is strong that filth flies in particular are mechanical vectors. Mouthparts, tarsi and gastrointestinal tracts become contaminated when the flies feed on contaminated substrates. Upon dispersal, the flies can inoculate new substrates with contaminated tarsi, mouthparts, fly vomit, and feces.

The medical significance of filth flies at a given time and place depends on which flies and people are involved, and on circumstances in which flies and people come into contact. Filth flies present relatively few problems where people live and work in urban and suburban settings in which indoor and outdoor environments are essentially free of fly breeding media. In rural settings, however, lacking adequate sanitation systems or neighboring mismanaged livestock and poultry production facilities, they can pose health concerns. Intolerance for flies is, in part, the basis for municipal nuisance and health codes used to enforce proper management of organic wastes on the affected premises. Sanitary standards established by the mid-1900s have dramatically reduced the epidemiological importance of filth flies in many parts of the developed world. Too often, however, basic sanitation and filth-fly management are unsatisfactory due to poverty, famine, or war. Under these circumstances, filth flies can reach tremendous densities, breeding in and around accumulated human waste and carrion.

The following muscid flies warrant attention with regard to public health.

House Fly (*Musca domestica*)

The house fly is the most common cause of fly annoyance in North America. Adults aggregate around garbage, compost piles, and other food sources, and they readily enter buildings. House flies are conspicuous when alighting directly on people, crawling on human food, or resting on walls, windows and ceilings. These substrates become soiled with fly specks, dried droplets of fly vomit and feces.

In a classic pair of experiments, Watt and Lindsay (1948) and Lindsay et al. (1953) provided strong evidence that the house fly is a significant vector of enteric pathogens. They controlled filth flies with residual insecticides in selected towns in southern Texas and southern Georgia (USA) and left neighboring towns untreated as controls. Fly surveillance in the treated and untreated towns showed that treatments greatly reduced the densities of house flies and other species. Surveillance of the residents in the treated towns showed concurrent declines in the incidence of diarrhea in people of all ages and in isolates of *Shigella* from children under 10 years of age.

Other studies have confirmed the importance of the house fly in the epidemiology of enteric diseases. Intensive trapping to remove flies at two military field bases in Israel caused declines in fly populations at mess tents, concurrent declines in frequencies of diarrhea and shigellosis among base recruits, and declines in rates of seroconversion for antibodies to *Shigella* and enterotoxigenic *E. coli*. (Cohen et al., 1991). Elsewhere, village-wide spraying of six Pakistani villages during two consecutive fly seasons reduced house fly populations by 95% and lowered the incidence of childhood diarrhea by 23% (Chavasse et al., 1999; also see West et al., 2006). In addition, simple screening of caged broiler houses in Denmark caused a substantial decrease in incidence of chicken-borne campylobacteriosis (Hald et al., 2007).

These studies provide strong evidence that house flies can be important routes for spread of fecal-borne pathogens. Prudence dictates that the house fly and other filth flies should be controlled through sanitation in the synanthropic environment, and that they should be prevented from contacting human food at all points of production, distribution, preparation, and consumption.

Bazaar Fly (*Musca sorbens*)

This nonbiting, synanthropic fly is common in Africa, Asia, and many Pacific islands. Adults feed persistently at the eyes, noses, and mouths of people (Fig. 17.15) and other

FIGURE 17.15 Aggregating bazaar flies (*Musca sorbens*) on human hosts. *Photograph by Raymond Lewis and Denys Dawnway, with permission, © Natural History Museum, London.*

large mammals. The species is strongly exophilic and can be conspicuous wherever human food is exposed outdoors. Greenberg (1971, 1973) summarized the extensive literature that associates the bazaar fly and its close relatives with human pathogens. Most notably, these flies are suspected of mechanically transmitting enteric pathogens and causal agents of acute bacterial conjunctivitis and trachoma. A study involving paired villages in The Gambia showed that community spraying, which reduced bazaar fly populations by around 75%, lowered the incidence of trachoma eye disease (caused by *Chlamydia trachomatis*) by 75% and the incidence of childhood diarrhea by 22% (Emerson et al., 1999). However, a study in Tanzania failed to show reduced prevalence of trachoma with fly spraying after the administration of antibiotics in treated communities (West et al., 2006).

Bush Fly (*Musca vetustissima*)

The earliest European travelers in Australia recorded annoyance by bush flies. This nonbiting dung fly, like the closely related bazaar fly, is strongly exophilic and is a potential irritant to humans almost anywhere in Australia. Flies that are attracted to people swarm around the head, feed at eyes and nostrils, and settle on the head, back, and shoulders. Once on hosts, the flies are peculiarly sedentary. More than a casual brush of the face with the hand is required to dislodge them, leading to a hand gesture that is humorously called an "Aussie salute." Larvae are known to occur in human and animal feces, so adults are a potential mechanical vector of enteric pathogens. Furthermore, the propensity of adults to feed at a host's eyes makes the bush fly a prime suspect in transmission of eye pathogens (Greenberg, 1971).

Face Fly (*Musca autumnalis*) and Cluster Fly (*Pollenia rudis*)

The face fly and cluster fly are two of several species of flies that can be a nuisance in households during winter and early spring. Other species include various blow flies (Calliphoridae), *Muscina* spp., and *Ceroxys latiusculus* (Otitidae). Overwintering flies in cold buildings can be activated by heaters or warm weather and become attracted by light to inhabited rooms. Often people first take notice when live and dead specimens occur at sunny windows. Dead flies that have accumulated over years in an infested building can attract dermestid beetles. Although the flies and beetles can be a source of allergens, these insects do not pose any other known medical threat in indoor environments.

The face fly is a developmental host for species of mammalian eyeworms in the genus *Thelazia*. Definitive hosts of these nematodes are a wide range of wild and domesticated mammals, including dogs, cats, rabbits, camels, cattle, horses, and sheep. Rare zoonotic infections in people have been recorded, involving *T. californiensis* and *T. gulosa* in the United States and *T. callipaeda* in Europe and Asia (Bradbury et al., 2018). Female *Thelazia* spp. live in their host's lachrymal ducts, where they cause mild irritation and ophthalmia (Soulsby, 1982). Eggs are shed and hatch in eye fluids. First-stage larvae are ingested by eye-feeding flies, penetrate the midgut, and develop further in the fly's hemocoel. After 2−4 weeks extrinsic incubation in the fly, infectious third-stage nematodes exit the fly's mouthparts when the vector feeds on another definitive host.

Stable Fly (*Stomoxys calcitrans*)

The stable fly is an important nuisance in outdoor environments throughout the Americas, Eurasia, and the Afrotropical Region. This fly will readily attack people, usually on the lower legs, causing a searing pain with each probe of its bayonet-like proboscis (Fig. 17.12A). It does not take many stable flies to disrupt activities of sunbathers, anglers, and others seeking outdoor leisure. Outbreaks have been recorded in the United States at tourist spots in the Great Lakes area, the Atlantic seaboard, and the Gulf Coast. Annoyance by stable flies is not confined to resorts and beaches; the flies can occur wherever people, fly breeding sites, and favorable weather coincide.

Adoption of the U.S. Declaration of Independence on July 4, 1776, by delegates to the Continental Congress may have been hastened by stable flies. According to Fuller (1913), debate on the Declaration drafted by Thomas Jefferson and his committee might have lasted much longer were it not for torment from stable flies. Jefferson noted the weather was oppressively warm that day in Philadelphia, and the meeting room was next to "… a stable, whence the hungry flies swarmed thick and fierce, alighting on their legs and biting hard through their thin silk stockings. Treason was preferable to discomfort." Clearly Jefferson had a wit, but he also knew enough entomology to infer that the nearby stable was the source of the flies.

As with any blood-feeding arthropod, stable flies provide an opportunity for transmission of blood-borne human pathogens. Experimental evidence suggests that the fly can acquire animal pathogens as mouthpart contaminants. Ingested particles can remain viable in the lumen of a fly's gut and diverticulum for hours to several days. However, none of these pathogens infect the fly, so only mechanical transmission is possible. Experimental evidence using animal disease models in realistic settings suggest that the stable fly is not a vector of any consequence to human health.

False Stable Fly (*Muscina stabulans*) and Its Relatives

The false stable fly and its relatives are common around filthy habitats, including latrines, household wastes, and accumulations of animal manure. The adults have feeding habits similar to those of the house fly and present similar risks for mechanical transport of food-borne pathogens. These flies remain outdoors and rarely feed on human food. However, they do feed and defecate on fruit, and serve as potential vectors wherever breeding sites are near open-air markets and roadside fruit stands. Larvae of the false stable fly and *M. levida* have been involved in rare cases of intestinal and urinary myiasis.

Little House Fly (*Fannia canicularis*) and Its Relatives

These nonbiting filth flies can become nuisances when swarms occur inside inhabited buildings. Hovering *Fannia* spp. often occur at head height indoors, where they can be particularly distracting and bothersome.

Adults of both sexes can be contaminated with pathogenic microbes from filthy larval breeding sites such as latrines, rotting garbage, and poultry manure. It is important, therefore, to exclude *Fannia* from areas where human food is prepared or consumed. On balance, *Fannia* spp. generally pose less of a health hazard than house flies because *Fannia* rarely land and feed on human food.

In the western United States, *Fannia* spp. in the *benjamini* group are commonly attracted to human sweat and mucus. One species in this group, *F. thelaziae*, is a developmental vector for the mammalian eyeworm *Thelazia californiensis*.

The little house fly and latrine fly have been involved in cases of intestinal, aural, and urinary myiasis of people. Most of the cases are thought to have arisen from eggs laid on clothing or bedding soiled with human feces.

Garbage Flies (*Hydrotaea* spp.)

Garbage flies and their larvae are common around municipal garbage dumps, compost sites, poultry houses, and dairies. As occurs with other flies from these kinds of environments, garbage flies can be contaminated with microbial pathogens. However, garbage flies are more sedentary than house flies and are far less inclined to enter buildings and contaminate human food.

Sweat Flies (*Hydrotaea* spp.)

Very little is known about the medical importance of sweat flies in North America. Females of six North American species, including *Hydrotaea meteorica* and *H. scambus*, feed persistently on perspiration and secretions from eyes, nostrils, lips, and other parts of their hosts. Because sweat flies are exophilic and occur most frequently in wooded areas, they are encountered by people in wooded parks, golf courses, and similar outdoor habitats. Except for their annoyance, sweat flies are not regarded as medically significant.

VETERINARY IMPORTANCE

Muscid flies affect the health and comfort of domesticated and wild animals. Domesticated hosts include cattle, sheep, goats, horses, dogs, pigs, and poultry. Wildlife hosts in North America include bison, elk, deer, moose, and rabbits, and other mammals elsewhere in the world. Muscid flies cause discomfort, injure skin, affect growth and thriftiness, and transmit pathogenic viruses, bacteria, helminths, and cestodes. Repeated feedings on localized areas of skin can lead to secondary infections and scabs on the affected animal's ears, legs, back, and other body regions. Feeding by nonbiting muscid flies can retard the healing of wounds caused by biting arthropods and other agents. Muscid fly larvae also can be involved in cases of secondary myiasis in mammals and primary myiasis in birds.

Animals display a variety of **aversive responses** to attack by biting and nonbiting flies. As examples, horses will stamp hooves and switch tails, dogs will cower under cover, and cattle in a herd will mill together, bunched in a rosette formation with tails outward (Fig. 17.16). Frequencies of these and other aversive behaviors increase with fly density (Mullens et al., 2006). Aversive behaviors can be disruptive and interfere with the handling, working, and showing of animals.

Biting muscid flies cause host vital signs to elevate (Schwinghammer et al., 1986a, 1986b). These changes, if prolonged, may be accompanied by changes in water and nitrogen balance. The net effect can be to reduce the

FIGURE 17.16 Holstein cattle bunching in response to attack by flies. *Photograph by Roger D. Moon.*

amount of metabolic energy available for growth and lactation and to reduce efficiency with which animals convert their feed into animal products. Livestock owners recognize a condition called **fly worry**, where animals appear irritated and generally unthrifty.

Economic effects of muscid flies on livestock and poultry industries of the United States are substantial. Estimates indicate that the stable fly and horn fly alone cause annual losses in excess of $1.3 billion in reduced yields and increased production costs for beef and dairy industries (Drummond, 1987; Taylor et al., 2012). Losses attributable to the face fly in the United States are $123 million annually, resulting from the role of this fly in the epizootiology of bovine **pinkeye**. Costs incurred to manage the house fly and other filth flies around livestock and poultry operations have not been estimated. They are no doubt substantial and will probably increase as suburban development continues to expand into traditionally agricultural lands.

House Fly (*Musca domestica*)

This cosmopolitan fly is often the most abundant insect where livestock, poultry, or companion animals are housed. Adults occur on virtually all substrates surrounding the animals, including feed, feces, vegetation, and walls and ceilings of buildings. Adults also occur directly on animals, where they feed on available blood, sweat, tears, saliva, and other body fluids. In response to fly annoyance, animals flap their ears, shake their heads, and avoid pen locations where flies are particularly abundant. Beyond these behavioral symptoms, however, house flies appear to cause no measurable harm. Even when present in large numbers, house flies cause little or no adverse effects on animal growth or feed conversion in cattle, pigs, and other animals. Thus houseflies have much less impact on those animals than on the health and comfort of people living in their vicinity.

House flies can be significant mechanical vectors of microbial pathogens. Adults feed on feces and manure and foul their environment with **fly specks**. These habits degrade the appearance of facilities and contribute to microbial contamination of eggs (Fig. 17.17) and milk at points of production. House flies also can be important in the spread of animal pathogens and genes for antibiotic resistance among animal production facilities (Ahmad et al., 2007; Hald et al., 2007; Macovei and Zurek, 2006; Rochon et al., 2005; Schurrer et al., 2004).

House flies are also developmental hosts for *Habronema muscae* and *Draschia megastoma*, two spirurid nematodes that cause gastric and cutaneous forms of **habronemiasis** in horses. In gastric infections, female worms invade the mucosa of the horse stomach and lay eggs that eventually pass out in the feces. Fly larvae become infected by ingesting these eggs. First-stage nematode larvae pass through the maggot's midgut into the hemocoel and

FIGURE 17.17 Chicken egg speckled with vomit and feces from house fly (*Musca domestica*). *Photograph by Ralph E. Williams.*

subsequently metamorphose into infective third instars while the maggot metamorphoses into an adult. After the fly emerges, the infective third-stage larvae migrate through the thorax and eventually reach the mouthparts. A new gastric infestation in a horse can arise as nematodes exit the mouthparts of flies that feed around the hosts' mouth or if the horse ingests an infected fly in its feed. The nematodes eventually mature and become established in the mucosa. A new cutaneous infection occurs if an infected fly feeds on the host's skin. Cutaneous infections are a dead end for these nematodes because larvae in skin do not develop to maturity. Bush flies and bazaar flies are also hosts for *H. muscae* and *D. megastoma*. Further details of nematode life cycles and development of habronemiasis can be found in Soulsby (1982).

The house fly is also a developmental host for a **chicken tapeworm**, *Choanotaenia infundibulum*. Prevalence of this tapeworm is greatly reduced where chickens are housed in elevated cages, which prevents birds from eating infected fly larvae and pupae.

House fly larvae have been recorded in cases of secondary wound myiasis. Females attracted to purulent wounds can feed and oviposit, and subsequent larvae feed on wound discharges and retard healing. Cases have been reported from nearly all species of domesticated animals.

Bush Fly (*Musca vetustissima*)

As adults, bush flies aggregate around large mammals, including cattle and horses. These nonbiting dung flies feed on facial and urogenital secretions and on serum and blood from wounds. Irritation by feeding flies can lead to skin lesions around the eyes and vulva, particularly of horses, and can retard wound healing. Annoyance by bush flies can induce animals to bunch and mill about, but their economic consequences are not documented. Studies in Australia in the first half of the 1900s suggested that the bush fly is a developmental host for the equine parasites *Habronema*

muscae and *D. megastoma*. Their extrinsic life cycle in the bush fly is the same as in the house fly.

Face Fly (*Musca autumnalis*)

Adult face flies are conspicuous on cattle, bison, and horses (Fig. 17.18), swarming around their heads and feeding at eyes, faces, and wounds. Hosts respond by blinking their eyes, flapping their ears, shaking their heads, and switching their tails. However, modest numbers of face flies do not appear to affect the thriftiness of grazing dairy and beef cattle. Experimental dairy herds protected with repellents grazed as much, grew as fast, and produced as much milk as unprotected herds. In other experiments with beef cattle, steers in screen cages with populations of face flies consumed as much feed and grew as fast as steers in cages without flies.

The face fly is much more important as a vector of bovine and equine pathogens. Of great concern to North American cattle producers is **pinkeye** (Fig. 17.19). This eye disease, also known as **infectious bovine keratoconjunctivitis** (IBK), is caused by the bacterium *Moraxella bovis*. Symptoms include reddened conjunctiva, excessive tearing, photophobia, opacity, and ulceration of the cornea. Pinkeye may involve one or both eyes and is most frequent among calves of white-faced breeds. Expenses associated with pinkeye involve surveillance and treatment of affected animals and retarded growth and blindness in cases that go undetected.

Face flies are mechanical vectors of *M. bovis*, as evidenced by isolation of the pathogen from face flies collected near infected cattle. In laboratory studies, viable bacteria have been recovered from tarsi, mouthparts, diverticula, and regurgitant of flies exposed to bacterial cultures several hours earlier. Thus, *M. bovis* can acquire the bacterium from cattle, and the bacterium can remain viable for several hours on and in contaminated flies. Face flies also may create avenues of infection when they scarify the host conjunctiva while feeding. Furthermore, by stimulating host bunching, face flies may contribute to direct eye-to-eye spread of the bacterium among herd mates. Although the face fly is potentially important in the spread of *Moraxella*, it is not necessary that they be present to have outbreaks of the disease. Pinkeye was known to occur in the United States at least 50 years before the face fly was introduced.

FIGURE 17.18 Face flies (*Musca autumnalis*) on head of a horse. *Photograph by Gary R. Mullen.*

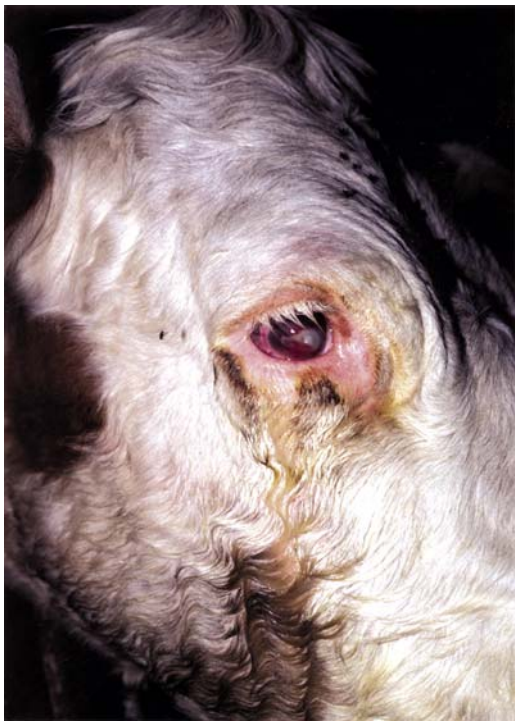


FIGURE 17.19 Pinkeye in a Hereford cow. Note opacity of cornea, reddened conjunctiva, and tearing below eye. *Photograph by Roger D. Moon.*

The face fly is also a developmental host for several spirurid nematodes. Eyeworms in the genus *Thelazia* live in the lachrymal ducts of horses, cattle, and other mammals. In North America, the face fly transmits *T. lacrymalis* among horses and *T. gulosa* and *T. skrjabini* among cattle. Infections are benign in both hosts. The filarial nematode *Parafilaria multipapillosa* infects domesticated and wild equids in southern Europe, northern Africa, the Middle East, central and southern Asia, and South America (Soulsby, 1982). It is transmitted in Russia by *Haematobia atripalpis*, but vectors elsewhere have not been identified.

In Sweden and South Africa, the face fly and closely related species are intermediate hosts of another filarial nematode, *Parafilaria bovicola*. This worm causes **bovine parafilariasis**, also known as **green muscle disease**, named after the appearance of subcutaneous green carcass lesions trimmed at slaughter. After mating, female

nematodes become established under the skin of the back or sides of infected hosts. There they create bleeding points, holes in skin that exude blood and serum that can attract nonbiting flies. Appearance of bleeding points coincides with the presence of vectors in the spring. When microfilariae in exudates are ingested by a fly, they penetrate the fly's midgut and undergo development for 2−3 weeks in the fly's hemocoel. During this extrinsic incubation period, the nematodes metamorphose to infective third stages (L3) and then migrate through the fly's thorax to the proboscis. New hosts become infected when the fly feeds on another animal. In a new host, *P. bovicola* takes about 10 months to reach the host's back, where it matures to the adult. For further details on the biology of this parasite, see Bech-Nielsen et al. (1982).

In South Africa where *P. bovicola* is endemic, the nematodes are transmitted by three endemic *Musca* spp., all in the subgenus *Eumusca*. Four other *Musca* spp. in other subgenera are not competent vectors for reasons that are unclear. *Parafilaria bovicola* was introduced into Sweden and France where the South African vectors are absent. In the nematode's new range, it is transmitted exclusively by the face fly, itself a member of *Eumusca*. If *Parafilaria* were to be imported into North America, its establishment and spread would be almost certain where the face fly is already present.

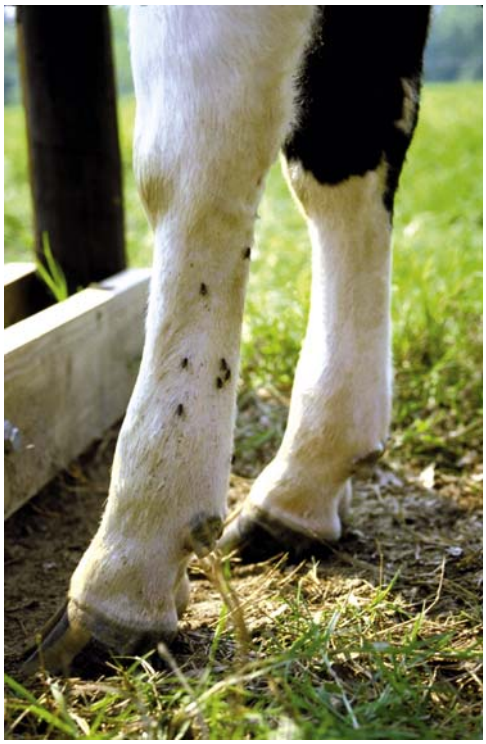


FIGURE 17.20 Stable flies (*Stomoxys calcitrans*) on foreleg of a Holstein calf. *Photograph by Gary R. Mullen.*

Stable Fly (*Stomoxys calcitrans*)

This cosmopolitan species attacks most large mammals, including domesticated cattle, horses, donkeys, dogs, swine, sheep, goats, and camels. Wild hosts include Bovidae, Cervidae, Equidae, Canidae, and Felidae, both in their native ranges and in zoos. The stable fly is often abundant where mammals are confined indoors and outdoors. Confined animals attract and sustain immigrating adults, and organic debris associated with the animals supports local fly breeding.

Stable flies attack large animals on their legs (Fig. 17.20), sides, backs, and bellies, whereas small ruminants and dogs are attacked most frequently on their legs, heads, and ears. Individual stable flies typically feed once per day, and remain on their host for 2−5 min, just long enough to obtain a bloodmeal. Engorged flies can be found on nearby vegetation, fences, and walls. Stable fly bites are painful, and localized wounds can coalesce to form scabs that are slow to heal, especially when aggravated by host scratching and rubbing. Such lesions commonly occur on tips of dogs' ears and elsewhere on other hosts where hair is short or naturally parted.

Behavioral responses to stable fly attack include a variety of aversive behaviors and physiological responses. Attacked animals are likely to stamp and kick their legs, which makes dairy cows difficult to milk and horses difficult to groom and show. Unrestrained cattle, horses, and small ruminants commonly will bunch when attacked. This behavior, combined with leg stamping and tail switching, is a clear indicator of stable fly activity (Mullens et al., 2006).

Experiments with penned beef cattle have shown that irritation by stable flies causes cattle to consume less feed, to grow more slowly, and to convert feed into body mass less efficiently. These effects are greater when weather is hot and humid, presumably because bunching in response to fly attack interferes with the ability of the animals to dissipate excess heat. From an economic perspective, stable flies increase beef production costs because affected cattle require more time and more feed to reach slaughter weight. It is likely that stable flies similarly, affect growing beef calves and lactating dairy cows. As a general guideline, economic losses in feedlots are likely to occur whenever the average number of stable flies per foreleg is three or more (Catangui et al., 1997). Stable fly control is usually warranted when bunching, stamping, and tail switching are excessive.

The stable fly is not an important vector of animal pathogens. Experimental evidence has shown that it is possible for the stable fly to mechanically transmit retroviruses that cause **equine infectious anemia** in horses and **bovine leukosis** in cattle. In nature, however, the role of stable flies as vectors of these agents is negligible. Tabanid

flies are far more important than stable flies in the spread of equine infectious anemia virus. With respect to bovine leukosis virus, transplacental transmission, and transmission during vaccination, tattooing, and rectal palpation are more important as routes of transmission than are biting flies.

The stable fly also is a developmental vector for *Habronema microstoma*, a spirurid nematode that causes gastric and cutaneous forms of **habronemiasis** in horses throughout the world. The gastric form of habaronemiasis is benign, whereas the cutaneous form presents conspicuous granular lesions known as **summer sores**. Onset of summer sores coincides with the stable fly breeding season. These diseases, the parasite that causes them, and the roles played by the stable fly in their transmission are very similar to the situations with *D. megastoma*, *H. muscae*, and the house fly (Soulsby, 1982).

Horn Fly (*Haematobia irritans irritans*) and Buffalo Fly (*Haematobia irritans exigua*)

Of all parasitic arthropods, these two biting dung flies (Fig. 17.21) have the greatest effects on health and productivity of cattle. Both sexes of these two flies feed frequently each day, consuming an average of 10 μL of blood per fly per day (Kuramochi, 1985). At this rate, a cow with an exceptionally large population of 3,000 flies would lose about 30 mL of blood each day. This is a relatively small amount given that the blood volume of an adult cow is about 25 L. Nonetheless, the bites are painful and irritating, and feeding lesions become cosmetic defects in tanned and dyed leather. Feeding by the buffalo fly on zebu cattle in Australia can lead to scabs on their hosts' withers and faces. Infested hosts react to horn flies and buffalo flies by licking their backs, twitching their flanks, switching their tails, and kicking at their bellies with their hindlegs. These defensive behaviors usually suggest that an animal is being attacked by a biting fly of one kind or another. In the case of pastured or rangeland cattle, common culprits are the horn fly or buffalo fly.

FIGURE 17.21 Heavy infestation of horn flies (*Haematobia irritans*) on pastured cow. *Photograph by D. Craig Sheppard, courtesy of University of Georgia.*

Studies in the United States and Canada demonstrate that control of horn flies on mother cows can lead to a substantial increase in average daily growth rates of nursing calves (Drummond, 1987). Similarly, growth rates of yearling stocker cattle and lactation rates of dairy cows may increase following control (Jonsson and Mayer, 1999). However, size of the benefit of horn fly control has varied among studies, presumably due to differences in density of flies, degree of control, and presence of other biting flies and internal parasites. Benefits also can vary with weather, availability of forage, and growth potential determined by cattle genotype, age, and condition when flies are present.

Increases in animal performance following horn fly control make sense in light of animal metabolism, behavior, and energy budgets. When attacked by horn flies, stanchioned steers show elevated heart beats, respiratory rates, rectal temperatures, urine production and urine nitrogen concentration (Schwinghammer et al., 1986b). Pastured steers switch tails more frequently, spend less time grazing, and spend more time walking and resting during the day. These metabolic and behavioral responses suggest that horn flies increase the amount of energy spent by cattle in defending themselves against flies, thereby reducing energy available for growth. With nursing calves, the response to horn fly control is likely to be reflected in increased milk production by their dams; horn flies occur mainly on the cows and only incidentally on their calves.

Progressive management programs for beef cattle usually rely on static thresholds to judge if control of horn flies is economically justified. Measured as an average number of horn flies per animal side, recommended thresholds range from 25 flies per side in Alberta (Canada) to 100 in Nebraska and 200 in Texas (USA). When densities exceed these thresholds, it is likely that increases in calf or steer growth rates in response to fly control during the fly season will more than pay for the cost of treating the cattle, whatever method is used.

The horn fly is a developmental vector for *Stephanofilaria stilesi*, a spirurid nematode that causes **stephanofilariasis** in cattle. This is a form of granular dermatitis that occurs mainly on the belly, scrotum, prepuce, and udder. This nematode is most prevalent in the western United States and Canada but is also recorded from cattle in the Old World. Mature *S. stilesi* occur in the skin. First-stage larvae (L1) are acquired as horn flies feed, following which they enter the fly's haemocoel and metamorphose to L3s. They are introduced into the definitive host when the fly feeds at a later time. Extrinsic incubation is about 3 weeks. Other species of *Stephanofilaria* occur in the Old World where they are thought to be transmitted by other species of muscid flies (Soulsby, 1982).

Horn flies have been incriminated in the transmission of **bovine mastitis** among dairy cows. Damaged teat ends and teat sores caused by horn flies were identified as a contributing factor in *S. aureus* mastitis. This bacterial pathogen has been isolated from field-collected flies, and treatment of cattle with insecticides has reduced disease incidence in treated herds.

Sweat Flies (*Hydrotaea* spp.)

A few species of these flies feed on large mammals. In Europe, the **sheep head fly** (*H. irritans*) swarms and feeds at the faces of sheep, cattle, and deer. Affected sheep develop a condition known as **head fly disease**. Irritated sheep scratch, rub, and open wounds that are further aggravated by the flies. The disease is worst among horned animals of open-faced breeds lacking wool on their faces and among flocks grazing in wooded pastures. Larvae of sheep head fly are sparsely dispersed in soil and decomposing plant litter in grasslands and forests. Larvae of other species have been recorded from undisturbed cattle dung pats (Robinson and Luff, 1979).

The sheep head fly and a complex of other European sweat flies also feed at cow teats. Circumstantial evidence suggests that teat-feeding sweat flies may be mechanical vectors of *Actinomyces* (formerly *Corynebacterium*) *pyogenes*, the putative cause of **summer mastitis** in pastured dairy cattle. It is likely that *Hydrotaea* species are secondary vectors of this pathogen. The importance of sweat flies as vectors of pathogens in North America is not well documented.

Bird Nest Parasites (*Philornis* spp.)

Several species of *Philornis* (Muscidae) are associated with declining populations of birds in the Caribbean and South America. In the Dominican Republic, adults and chicks of critically endangered Ridgway's hawks (*Buteo ridgwayi*) are frequently parasitized by *Philornis pici*. Larvae develop in warbles on their hosts' heads and bodies (Fig. 17.22), and chick health declines with increasing numbers of larvae per chick. In Belize, a second raptor, the orange-breasted falcon (*Falco deiroleucus*), is parasitized by an undetermined *Philornis* species. The young larvae develop in superficial skin lesions, whereas older larvae occur in deeper warbles on heads and bodies of infected chicks In the Galapagos Islands, *Philornis downsi* was introduced from Ecuador in the mid 1960s. The fly now parasitizes native island bird species, including most of Darwin's finches (McNew and Clayton, 2018). Experiments showed that exclusion of *P. downsi* larvae from nests of the medium ground finch (*Geospiza fortis*) increases chick size and fledging success (Koop et al., 2011).

FIGURE 17.22 Head of orange-breasted falcon chick parasitized by seven *Philornis* larvae. Note caudal spiracles of larvae in enlarged warbles around beak, and lateral aspects of one larva in shallower, surface lesion at edge of left eye. *Photograph by Camille Meyers, courtesy of Peregrine Fund.*

PREVENTION AND CONTROL

Three general approaches are used to avoid or reduce problems caused by muscid flies: (1) prevention of breeding; (2) killing adults before they cause harm or produce offspring; and (3) exclusion of adults with screens and other barriers. Prevention of breeding can be either indirect, by making potential media unavailable or unsuitable for survival of preadult stages, or direct by killing immatures before they can develop to adults. A variety of methods can be used to accomplish these objectives (Drummond et al., 1988).

The best approach is to use several methods simultaneously in an integrated pest management program to achieve desired levels of control in poultry houses, stables, and dairies (Axtell, 1986). For example, sanitation and surveillance of adult abundance are commonly used in combination. When densities exceed a set tolerance threshold, sanitation can be increased and adulticides can be applied to bring fly numbers below the tolerance limit. Choices among alternative practices are determined by effectiveness against the target insect, practicality, costs in materials and labor, and environmental acceptability.

Emphasis should be placed on source reduction wherever possible. Housing for people or animals should be designed to limit accumulation of fly breeding media. Particular attention should be given to locations where human and other animal feces, domestic garbage, and rotting animal feed accumulate. The crucial first step to preventing enteric diseases is to prevent filth flies from breeding near human communities. The best defense is a closed sewage system or privy that prevents gravid flies from reaching human excrement. Curtis (1989) has designs of privies that do not require running water.

Facilities should be designed to minimize the labor required to maintain adequate sanitation. In livestock and poultry housing, lanes, alleys, and pens where manure can collect should be easy to scrape. Feed and water should be provided in separate areas, if possible. Straw bedding for animals is particularly difficult to handle and is a notorious source of filth flies, so alternatives such as sawdust, sand, or washable mats should be considered.

In practice, even well-designed facilities have residual places in corners, around feeders, or along fence lines where organic debris can accumulate and fly breeding can occur. These places should be inspected regularly. Waste disposal should involve proper burial, spreading in a thin layer (<3 cm) on open fields, submersion in water, or aerobic composting. Compost piles must be turned frequently to aerate and keep the material in a hot state of active fermentation. If stockpiled materials are not contained in bunkers with vertical sides, then special attention should be given to seepage areas that can form at pile margins.

Many beneficial organisms such as predators, parasites and natural competitors occur in the breeding media of muscid flies. These natural biological control organisms kill and consume developing fly eggs, larvae, and pupae. The faunas in poultry litter and feedlot manure are best known (Rueda and Axtell, 1985; Axtell, 1986). Important groups include nymphs and adults of predatory mites, larvae and adults of predatory beetles, predatory third-instar larvae of *Hydrotaea* spp. and *Muscina* spp., and adults and larvae of parasitic wasps. The latter group, called parasitoids, can be particularly effective. Once a female wasp finds a host, she drills into the host with her ovipositor and deposits one or more eggs (Fig. 17.23). The offspring eventually consume the host and emerge as adults. Pupal parasitoids are species that attack and emerge from host pupae, whereas larval-pupal parasitoids attack larvae and emerge from pupae. The parasitoids most frequently encountered in poultry and cattle manure are pupal parasitoids in the genera *Muscidifurax* and *Spalangia* (Pteromalidae). Other genera and families of parasitic wasps and beetles are prominent in dung pats, and these are mainly larval-pupal parasitoids.

FIGURE 17.23 Parasitoid wasp (*Muscidifurax zaraptor*), female, stinging pupa of house fly. *Photograph by Valerie J. Cervenka, University of Minnesota.*

Populations of beneficial insects and mites can be favored by keeping potential fly breeding media as dry as possible. Soil should be sloped to drain water away from possible breeding areas, and waterers should be kept in good repair. Certain species of parasitoids are available commercially, and these can be released to augment natural populations. However, the cost effectiveness of releasing parasitoids in commercial poultry and livestock facilities has been questioned.

In emergencies, larvicides can be sprayed directly into infested breeding media to kill fly larvae before adults emerge. Alternatively, larvicides can be administered to animals as feed additives or boluses. The active ingredients in these formulations pass through the animal's digestive system to create an insecticidal residue in feces or soiled bedding. Limitations of feed-through larvicides are that they are only effective against flies breeding in feces and bedding but not in other substrates and that some larvicides can disrupt natural biological control. Whatever application method is used, larvicidal residues need to be considered when disposing of treated media.

Management of adult flies is accomplished mainly with traps and adulticides. Inside closed buildings, house flies and *Fannia* spp. can be killed with sticky traps, light traps, sugar- and pheromone-based insecticidal baits, and adulticides formulated as knockdown or residual sprays. The same methods can be effective against adult stable flies, except for baits, which do not attract that blood-feeding species. The use of space sprays of ingredients with short half-lives, such as synergized pyrethrins, can be effective when applied as mists or fogs in closed spaces. These materials have a rapid, knock-down effect on flies contacted directly with the mist droplets.

In contrast, residual sprays of more persistent insecticides such as the pyrethroids, and organophosphates can be applied as coarse sprays to structural surfaces. These formulations provide a more prolonged effect because the residues remain toxic to flies that later walk or rest on the treated surfaces. In outdoor situations, residual sprays should be directed at fly resting sites such as building walls, fence lines, and vegetation where flies seek shelter during hot weather. To limit costs and to retard development of insecticide resistance, residual sprays should be used sparingly and only when necessary.

The use of **traps** is generally effective in closed environments, but if not closed, traps can be overwhelmed by immigration from outside sources. Options in outdoor

FIGURE 17.24 Cow emerging from walk-through trap used to control flies on cattle. Note baffles in side and fabric draped over exiting cow. *Photograph by Hedrick J. Meyer.*

environments are more limited. Walk-through traps (Fig. 17.24) can be used to collect and kill muscid flies from pastured cattle. These traps are most effective against horn flies in situations where host animals are forced to pass through traps on a daily basis. This is accomplished by placing traps in an entryway of a fenced enclosure surrounding water or feed supplement, or in a milking-parlor doorway.

Materials formulated as topical insecticides can be applied directly to animals. A variety of compounds can be applied as sprays, pour-ons, dusts, or wipe-ons. Some of the compounds are formulated into plastic, slow-release ear tags, whereas others can be dispensed in self-applicators such as oilers, back rubbers, and dust bags (Fig. 17.25). To be most effective, self-applicators should be maintained in areas where animals are forced to use them on a daily basis.

Topical insecticides usually are ineffective against house flies, stable flies, and face flies because these species spend so little time directly on animals. In contrast, topical insecticides are more effective against adult horn flies because they remain continuously on their hosts. Insecticidal ear tags were adopted widely in the cattle industry in the 1980s because they were inexpensive, required little labor to apply at turnout for spring grazing, and provided several months of effective horn fly control. However, their success was a mixed blessing. Pyrethroid insecticides were the active ingredients in the first widely used tags. Unfortunately, horn flies developed resistance to those compounds in the first 3–4 years of tag use. In response, manufacturers substituted organophosphate insecticides in later ear tags. Reports of control failures and bioassays indicate that horn flies have evolved resistance to pyrethroid and organophosphate compounds in multiple localities scattered throughout North America and South America.

Chemical repellents can be applied by hand directly to provide temporary relief from muscid flies and other pests. Repellents function mainly by interfering with host-finding behaviors and less so as toxicants. A variety of formulations are marketed mainly for use on horses and companion animals. Effectiveness varies with weather and level of animal activity. Few repellents are effective against muscid flies for more than a few hours.

The most effective way to prevent the house fly and the other filth flies from entering buildings is adult exclusion with door and window screens. Double doorways or positive-pressure air doors can further reduce fly entry into closed structures. These approaches are appropriate at entrances to restaurants, hospitals, and other institutions where flies cannot be tolerated. To prevent household infestations of overwintering adult face flies, cracks and crevices around doors, windows, and eaves should be sealed tightly with caulk. Residual insecticides can be sprayed on the sunny sides of buildings to intercept the flies as they arrive in autumn.

FIGURE 17.25 Dust bag positioned in fence line, used for self-application of topical insecticides for control of flies on cattle. *Photograph by Hedrick J. Meyer.*

REFERENCES AND FURTHER READING

Adams, D. A., Thomas, K. R., Jajosky, R., Foster, L., Sharp, P., Onweh, D. H., et al. (2016). Summary of notifiable infectious diseases and conditions – United States, 2014. *Morbidity Mortality Weekly Report, 2016*(63), 1–15. https://doi.org/10.15585/mmwr.mm6354a1.

Ahmad, A., Nagaraja, T. G., & Zurek, L. (2007). Transmission of *Escherichia coli* O157: H7 to cattle by house flies. *Preventive Veterinary Medicine, 80*, 74–81.

Axtell, R. C. (1986). *Fly control in confined livestock and poultry production*. Ciba-Geigy, Tech. Monogr, 59 pp.

Bech-Nielsen, S., Bornstein, S., Christensson, D., Wallgren, T.-B., Zakrisson, G., & Chirico, J. (1982). *Parafilaria bovicola* (Tubangui 1934) in cattle: Epizootiology-vector studies and experimental transmission of *Parafilaria bovicola* to cattle. *American Journal of Veterinary Research, 43*, 948–954.

Bradbury, R. S., Breen, K., Bonur, E. M., Hoyt, J. W., & Bishop, H. S. (2018). Case report: Conjunctival infestation with *Thelazia gulosa*: A novel agent of human thelaziasis in the United States. *The American Journal of Tropical Medicine and Hygiene*. https://doi.org/10.4269/ajtmh.17-0870.

Bruce, W. G. (1964). The history and biology of the horn fly, *Haematobia irritans* (Linnaeus); with comments on control. In *North Carolina agricultural experiment station technical bulletin no. 157*, 33 pp.

Burger, J. F., & Anderson, J. R. (1974). Taxonomy and life history of the moosefly, *Haematobosca alcis*, and its association with the moose, *Alces alces shirasi*, in Yellowstone National Park. *Annals of the Entomological Society of America, 67*, 204–214.

Catangui, M. A., Campbell, J. B., Thomas, G. D., & Boxler, D. J. (1997). Calculating economic injury levels for stable flies (Diptera: Muscidae) on feeder heifers. *Journal of Economic Entomology, 90*, 6–10.

Chavasse, D. C., Shler, R. P., Murphy, O. A., Huttly, S. R. A., Cousens, S. N., & Akhtar, T. (1999). Impact of fly control on childhood diarrhoea in Pakistan: Community-randomised trial. *Lancet, 353*, 22–25.

Chillcott, J. G. (1961). A revision of the Nearctic species of Fanniinae (Diptera: Muscidae) from north America. *Canadian Entomologist, Supplement, 14*, 1–29.

Cohen, D., Green, M., Block, C., Slepon, R., Ambar, R., Wasserman, S. S., et al. (1991). Reduction of transmission of shigellosis by control of houseflies (*Musca domestica*). *Lancet, 337*, 993–997.

Couri, M. S. (1999). Myiasis caused by obligatory parasites. Ia. *Philornis* Meinert (Muscidae). In J. H. Guimarães, & N. Papavero (Eds.), *Myiasis in man and animals in the Neotropical region. Bibliographic database*. São Paulo, Brazil: Pleiade/FAPESP, 308 pp.

Couri, M. S., Carvalho, C. J. B., & de, Löwenberg-Neto, P. (2007). Phylogeny of *Philornis* Meinert species (Diptera: Muscidae). *Zootaxa, 1530*, 19–26.

Curtis, C. F. (1989). *Appropriate technology in vector control*. Boca Raton, FL: CRC Press, 233 pp.

Drummond, R. O. (1987). Economic aspects of ectoparasites of cattle in North America. In W. H. D. Leaning, & J. Guerrero (Eds.), *Proceedings of a symposium: Vol. XXIII. The economic impact of parasitism in cattle*. Montreal: World Veterinary Congress.

Drummond, R. O., George, J. E., & Kunz, S. E. (1988). *Control of arthropod pests of livestock: A review of technology*. Boca Raton, FL: CRC Press, 245 pp.

Edwards, F. W., Oldroyd, H., & Smart, J. (1939). *British blood-sucking flies*. British Museum (Natural History), 156 pp.

Elzinga, R. J., & Broce, A. B. (1986). Labellar modifications of Muscomorpha flies (Diptera). *Annals of the Entomological Society of America, 79*, 150–209.

Emerson, P. M., Lindsay, S. W., Walraven, G. E. L., Faal, H., Bogh, C., Lowe, K., et al. (1999). Effect of fly control on trachoma and diarrhoea. *Lancet, 353*, 1401–1403.

Frankel, G., & Bhaskaran, G. (1973). Pupariation and pupation in cyclorrhaphous flies (Diptera): Terminology and interpretation. *Annals of the Entomological Society of America, 66*, 418–422.

Fuller, H. B. (May 1913). Myths of American history. *Munsey's Magazine*, 278–284.

Greenberg, B. (1971). *Flies and disease. Vol. I. Ecology, classification and biotic associations*. Princeton, NJ: Princeton University Press, 856 pp.

Greenberg, B. (1973). *Flies and disease. Vol. II. Biology and disease transmission*. Princeton, NJ: Princeton University Press, 447 pp.

Hald, B., Sommer, H. M., & Skovgard, H. (2007). Use of fly screens to reduce *Campylobacter* spp. introduction in broiler houses. *Emerging Infectious Diseases, 13*, 1951–1953.

Hall, R. D. (1984). Relationship of the face fly (Diptera: Muscidae) to pinkeye in cattle: A review and synthesis of the relevant literature. *Journal of Medical Entomology, 21*, 361–365.

Haseyama, K. L. F., Wiegmann, B. M., Almeida, E. A. B., & de Carvalho, C. J. B. (2015). Say goodbye to tribes in the new house fly classification: A new molecular phylogenetic analysis and an updated biogeographical narrative for the Muscidae (Diptera). *Molecular Phylogenetics and Evolution, 89*, 1–12.

Huckett, H. C. (1954). A review of the North American species belonging to the genus *Hydrotaea* Robineay-Desvoidy. *Annals of the Entomological Society of America, 47*, 317–342.

Huckett, H. C., & Vockeroth, J. R. (1987). Muscidae. In J. F. McAlpine (Ed.), *Monograph 28: Vol. 2. Manual of Nearctic Diptera* (pp. 1115–1131). Ottawa: Agriculture Canada (Chapter 105), 658 pp.

Hughes, R. D. (1977). The population dynamics of the bushfly: The elucidation of population events in the field. *Australian Journal of Ecology, 2*, 43–54.

James, M. T. (1947). *The flies that cause myiasis in man*. U.S. Department of Agriculture, Miscellaneous Publication 631, 175 pp.

Jonsson, N. N., & Mayer, D. G. (1999). Estimation of the effects of buffalo fly (*Haematobia irritans exigua*) on the milk production of dairy cattle based on a meta-analysis of literature data. *Medical and Veterinary Entomology, 13*, 372–376.

Koop, J. A. H., Huber, S. K., Laverty, S. M., & Clayton, D. H. (2011). Experimental demonstration of the fitness consequences of an introduced parasite of Darwin's finches. *PLoS One, 6*, e19706.

Krasfur, E. S., & Moon, R. D. (1997). Bionomics of the face fly, *Musca autumnalis*. *Annual Review of Entomology, 42*, 503–523.

Kuramochi, K. (1985). Studies on the reproductive biology of the horn fly, *Haematobia irritans* (L.) (Diptera: Muscidae). II. Effect of temperature on follicle development and blood meal volume of laboratory-reared flies. *Applied Entomology and Zoology, 20*, 264–270.

Kuramochi, K. (2000). Ovipositional behavior of the horn fly (Diptera: Muscidae) in the field. *Journal of Medical Entomology, 370*, 461–466.

Kutty, S. N., Pont, A. C., Meier, R., & Pape, T. (2014). Complete tribal sampling reveals basal split in Muscidae (Diptera), confirms saprophagy as ancestral feeding mode, and reveals an evolutionary correlation between instar numbers and carnivory. *Molecular Phylogenetics and Evolution, 78*, 3349–3364.

Lindsay, D. R., Stewart, W. H., & Watt, J. (1953). Effect of fly control on diarrheal disease in an area of moderate morbidity. *Public Health Reports, 68*, 361–367.

Macovei, L., & Zurek, L. (2006). Ecology of antibiotic resistance genes: Characterization of enterococci from house flies collected in food settings. *Applied and Environmental Microbiology, 72*, 4028–4035.

McAlpine, J. F., Peterson, B. V., Shewell, G. E., Teskey, H. J., Vockeroth, H. J., & Wood, D. M. (1981). Manual of Nearctic Diptera. Volume 1. In *Monograph 27*Ottawa: Agriculture Canada, 674 pp.

McAlpine, J. F., Peterson, B. V., Shewell, G. E., Teskey, H. J., Vockeroth, H. J., & Wood, D. M. (1987). In , *Monograph 28Manual of Nearctic Diptera. Volume 2*. Ottawa: Agriculture Canada, 658 pp.

McNew, S. M., & Clayton, D. H. (2018). Alien invasion: Biology of *Philornis* flies highlighting *Philornis downsi*, an introduced parasite of Galápago birds. *Annual Review of Entomology, 63*, 369−387.

Morgan, C. E., & Thomas, G. D. (1974). *Annotated Bibliography of the Horn Fly, Haematobia irritans (L.), Including References on the Buffalo Fly, H. exigua (de Meijere), and Other Species Belonging to the Genus Haematobia* (pp. 1−134). Agricultural Research Service, Miscellaneous Publication No. 1278.

Morgan, C. E., & Thomas, G. D. (1977). Annotated Bibliography of the Horn Fly, Haematobia irritans (L.), Including References on the Buffalo Fly, H. exigua (de Meijere), and Other Species Belonging to the Genus Haematobia. In *Supplement I* (pp. 1−38). Agricultural Research Service, Miscellaneous Publication No. 1278.

Morgan, C. E., Thomas, G. D., & Hall, R. D. (1983a). Annotated bibliography of the stable fly, *Stomoxys calcitrans* (L.), including references on other species belonging to the genus *Stomoxys*. In *Missouri agricultural experiment station bulletin no. 1049* (pp. 1−190).

Morgan, C. E., Thomas, G. D., & Hall, R. D. (1983b). Annotated bibliography of the face fly, *Musca autumnalis* (Diptera: Muscidae). *Journal of Medical Entomology − Supplement, 4*, 1−25.

Mullens, B. A., Lii, K. S., Mao, Y., Meyer, J. A., & Peterson, N. G. (2006). Behavioral responses of dairy cattle to the stable fly, *Stomoxys calcitrans*, in an open field environment. *Medical and Veterinary Entomology, 20*, 122−137.

Owens, W. E., Oliver, S. P., Gillespie, B. E., Ray, C. H., & Nickerson, S. C. (1998). Role of horn flies (*Haematobia irritans*) in *Staphylococcus aureus*-induced mastitis in dairy heifers. *American Journal of Veterinary Research, 59*, 1122−1124.

Paterson, H. E., & Norris, K. R. (1970). The *Musca sorbens* complex: The relative status of the Australian and two African populations. *Australian Journal of Zoology, 18*, 231−245.

Petersen, J. J., & Greene, G. L. (1989). *Current status of stable fly (Diptera: Muscidae) research* (pp. 1−54). Entomological Society of America, Miscellaneous Publications No. 74.

Pickens, L. G., & Miller, R. W. (1980). Biology and control of the face fly, *Musca autumnalis* (Diptera: Muscidae). *Journal of Medical Entomology, 17*, 195−210.

Pont, A. C. (1975). A revision of the genus *Passeromyia* Rodhain & Villeneuve (Diptera: Muscidae). *Bulletin British Museum of Natural History (Entomology), 30*, 339−372.

Pont, A. C. (1991). A review of the Fanniidae and Muscidae (Diptera) of the Arabian Peninsula. In W. Buttiker, & F. Krupp (Eds.), *Pro Entomologia c/o natural history Museum, Basle, Switzerland: Vol. 12. Fauna of Saudi Arabia* (pp. 312−365), 419 pp.

Robinson, J., & Luff, M. L. (1979). Population estimates and dispersal of *Hydrotaea irritans* Fallen. *Ecological Entomology, 4*, 289−296.

Rochon, K., Lysyk, T. J., & Selinger, L. B. (2005). Retention of *Escherichia coli* by house fly and stable fly (Diptera: Muscidae) during pupal metamorphosis and eclosion. *Journal of Medical Entomology, 42*, 397−403.

Rueda, L. M., & Axtell, R. C. (1985). In *Guide to common species of pupal parasites (Hymenoptera: Pteromalidae) of the house fly and other muscoid flies associated with poultry and livestock manure*. North Carolina Agricultural Research Service, Technical Bulletin No. 278, 88 pp.

Sabrosky, C. W., Bennett, G. F., & Whitworth, T. L. (1989). *Bird blow flies (Protocalliphora) in North America (Diptera: Calliphoridae), with notes on the Palearctic species*. Washington, D.C.: Smithsonian Institution Press, 312 pp.

Schurrer, J. A., Dee, S. A., Moon, R. D., Rossow, K. D., Mahlum, C., Mondaca, E., et al. (2004). Spatial dispersal of porcine reproductive and respiratory syndrome virus-contaminated flies after contact with experimentally infected pigs. *American Journal of Veterinary Research, 65*, 1284−1292.

Schwinghammer, K. A., Knapp, F. W., Boling, J. A., & Schillo, K. K. (1986a). Physiological and nutritional response of beef steers to infestations of the stable fly (Diptera: Muscidae). *Journal of Economic Entomology, 79*, 1294−1298.

Schwinghammer, K. A., Knapp, F. W., Boling, J. A., & Schillo, K. K. (1986b). Physiological and nutritional response of beef steers to infestations of the horn fly (Diptera: Muscidae). *Journal of Economic Entomology, 79*, 1010−1015.

Skidmore, P. (1985). The biology of the Muscidae of the world. In *Dr. W. Junk series entomologica* (Vol. 29, pp. 1−550).

Soulsby, E. J. L. (1982). *Helminths, arthropods and protozoa of domesticated animals* (7th ed.). Philadelphia: Lea and Febiger, 809 pp.

Stone, A., Sabrosky, C. W., Wirth, W. W., Foote, R. H., & Coulson, J. R. (1965). A catalog of the Diptera of America north of Mexico. In *Agriculture handbook no. 276*. Washington, DC: U.S.D.A., 1696 pp.

Taylor, D. B., Moon, R. D., & Mark, D. R. (2012). Economic impact of stable flies (Diptera: Muscidae) on dairy and beef cattle production. *Journal of Medical Entomology, 49*, 198−209.

Thomas, G. D., & Skoda, S. R. (1993). In *Rural flies in the urban environment?*. Lincoln: North Central Regional Research Publication No. 335, Institute of Agriculture and Natural Resources, University of Nebraska, 97 pp.

Watt, J., & Lindsay, D. R. (1948). Diarrheal disease control studies. I. Effect of fly control in a high morbidity area. *Public Health Reports, 63*, 1319−1334.

West, L. S. (1951). *The house fly. Its natural history, medical importance, and control*. Ithaca, NY: Comstock, 584 pp.

West, L. S., & Peters, O. B. (1973). *An annotated bibliography of Musca domestica Linnaeus*. Folkstone, UK: Dawsons, 743 pp.

West, S. K., Emerson, P. M., Mkocha, H., Mchiwa, W., Munoz, B., Bailey, R., et al. (2006). Intensive insecticide spraying for fly control after mass antibiotic treatment for trachoma in a hyperendemic setting: A randomized trial. *Lancet, 368*, 596−600.

Zumpt, F. (1973). *The Stomoxyine biting flies of the world*. Stuttgart: G. Fischer Verlag, 175 pp.

Chapter 18

Tsetse Flies (Glossinidae)

William L. Krinsky
Division of Entomology, Peabody Museum of Natural History, Yale University, New Haven, CT, United States

Tsetse flies (Fig. 18.1) are obligate bloodsucking flies of medical and veterinary importance because they transmit trypanosomes that cause African sleeping sickness in humans and nagana in livestock. Fossil tsetse flies in the Florissant shale of Colorado in the western United States indicate that this family was present in the Western Hemisphere as recently as 26 million years ago. Tsetse flies now occur in over 10 million square kilometers in the tropical and subtropical regions of sub-Sahelian Africa (about 15°N to 26°S). Isolated populations of two species of tsetse flies have been recorded in southwestern Saudi Arabia (Elsen et al., 1990).

Tsetse (pronounced *tsé-tsee*) commonly is used as both a singular and plural term to denote one or more individuals or species of these flies. Although the origin of the name is obscure, it was used as early as the 19th century by the Tswana people living along the edge of the Kalahari Desert. "Tsénse," the Mozambique word for "fly," as well as other similar-sounding African names meaning "fly," are apparently onomatopoetic terms derived from imitations of the unique buzzing sound made by the adult flies (Austen, 1903).

FIGURE 18.1 Adult tsetse fly (*Glossina* sp.) on rabbit. *Courtesy of the Rockefeller Foundation.*

Tsetse generally are considered one of the greatest factors affecting the course of economic and social development in Africa. The morbidity and mortality caused by African sleeping sickness continue to be significant. Nagana, which has stifled agricultural productivity for decades, still stands as a major deterrent to the development of animal agriculture on that continent. African animal trypanosomiasis remains the main hindrance to development of efficient and sustainable livestock production, and the presence of tsetse is the root cause of hunger and poverty.

There is an extensive literature on tsetse, but a few monographic works provide particularly useful introductions to the field. The classic work by Buxton (1955) reviews the natural history of tsetse and provides a detailed historical account of the diseases associated with it. Mulligan (1970) includes a historical perspective in addition to an overview of the biology of tsetse and its parasites, pathology of these parasites in humans and domestic animals, treatment, and control. The epidemiology, pathology, and treatment of the trypanosomiases are reviewed by Maudlin et al. (2004). The historical, social, and economic effects of tsetse in five different African regions are extensively reviewed by Ford (1971), and the impact of tsetse on African rural development is discussed by Jordan (1986). A comprehensive monograph was written by Leak (1999).

TAXONOMY

Tsetse were formerly included in their own subfamily, Glossininae, or the Stomoxyini of the Muscidae because of the resemblance of tsetse to the stable fly and other biting muscids. However, because of their unique antennal structure, tsetse are now placed in their own family, **Glossinidae**. The reproductive and morphological similarities of tsetse to the keds and other hippoboscid flies has led to placement of Glossinidae within the **Hippoboscoidea** (McAlpine, 1989). Glossinidae includes the single genus

Medical and Veterinary Entomology. https://doi.org/10.1016/B978-0-12-814043-7.00018-2

Glossina with 31 species and subspecies (23 species, six of which are further divided into 14 subspecies) (Jordan, 1993). *Glossina* means "tongue fly," in reference to its prominent proboscis. Keys to species and subspecies are included in Jordan (1993). *Glossina* species are arranged into three subgenera (*Austenina*, *Nemorhina*, and *Glossina*) that correspond roughly with groups of species found in different ecological settings. The subgenera often are cited by their group names, each designated by one of the better-known species in each subgenus, i.e., the *fusca* group (=*Austenina*), the *palpalis* group (=*Nemorhina*), and the *morsitans* group. Species in the *fusca* group are most often found in forested habitats, such as rain, swamp, and mangrove forests. Species in the *palpalis* group occur among vegetation around lakes and along rivers and streams. The *morsitans* group, with the exception of the forest-dwelling *Glossina austeni*, occurs in open country and is most often found in dry thickets, scrub vegetation, and areas of savanna woodland (commonly composed of *Berlinia*, *Isoberlinia*, and *Brachystegia* species).

The geographical distributions of members of the three taxonomic groups are shown in Fig. 18.2. The *palpalis* group, which includes *Glossina palpalis*, *Glossina tachinoides*, *Glossina fuscipes*, and two less well-known species, occurs primarily along watercourses in western and central Africa. The *morsitans* group of savanna species, which includes *Glossina morsitans*, *Glossina pallidipes*, *Glossina longipalpis*, *Glossina swynnertoni*, and *G. austeni*, is primarily central and southeastern in distribution. The *fusca* group, which includes *Glossina fusca*, *Glossina tabaniformis*, *Glossina medicorum*, *Glossina longipennis*, *Glossina brevipalpis*, and eight other species, is found in forested areas that overlay most of the western and central African distribution of the *palpalis* group.

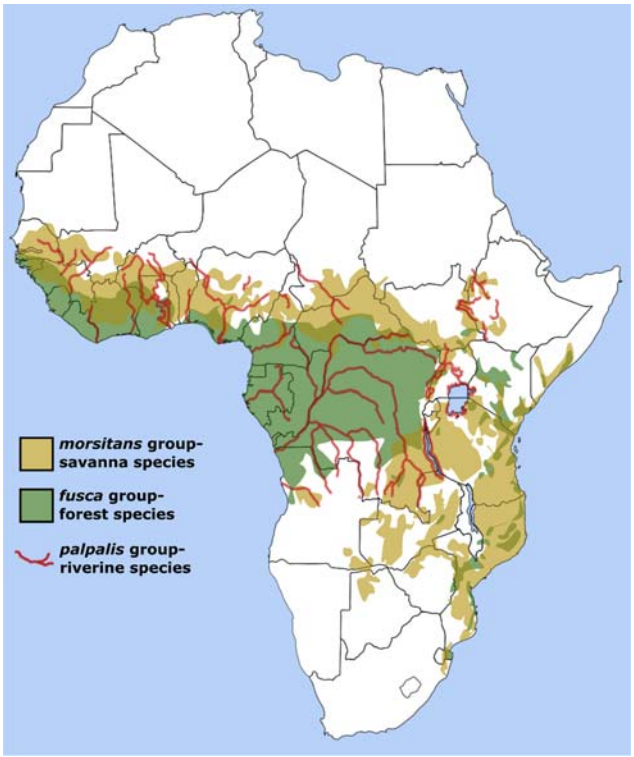


FIGURE 18.2 Distribution of *morsitans* group, *fusca* group, and *palpalis* group tsetse flies in Africa. *Original image prepared by Jonas G. King.*

MORPHOLOGY

Glossina species are tan or brown flies that range in length from 6 to 14 mm, excluding the proboscis. Members of the *fusca* group, which is considered phylogenetically primitive, are the largest, at 9.5−14 mm long. The *palpalis* and *morsitans* group species are small to medium in size, about 6.5−11 mm long. Species in the *palpalis* group generally have a uniformly dark brown abdominal tergum and the dorsal aspect of each hind tarsal segment is dark brown or black. Species in the *morsitans* group usually have dark segmental bands on the abdomen and only the distal segments of the hind tarsi are darkened dorsally.

Tsetse adults are characterized by several distinctive morphological features. These include the shape of the proboscis, the position and branching of the fringe on the arista of their antenna, and the wing venation and folding pattern. The swollen, bulbous base of the proboscis that lies under the head is different from the angled and thinner bases of the proboscises of the Stomoxyini. When the fly is not feeding, the proboscis extends directly forward between the palps in front of the head (Fig. 18.1). The proboscis (Fig. 18.3) is composed of two elongate, stylet-like mouthparts: the labrum and hypopharynx. The stylets are protected ventrally by the labium. The labellum at the tip of the labium is armed with teeth for cutting into host skin. The labrum, bounded by the hypopharynx and the labium, forms the food canal through which blood is drawn as the fly feeds (Fig. 18.3). The hypopharynx has a hollow central portion that forms the salivary canal through which saliva is secreted into the feeding site.

The three-segmented antennae arise on the frons just below the ptilinal suture, as in muscoid flies. The first segment is small; the second is at least two to four times larger than the first and generally about as long as wide; the third is elongate and oval to peapod-shaped and bears the distinctive arista. The arista has a conspicuous fringe of hairs along its dorsal surface; these hairs have small branch hairs that are not found on any other aristate flies (Fig. 18.4). The large brown or reddish eyes are separated in both sexes and comprise most of the posterior portion of the head.

The base of the thorax is only slightly wider than the width of the head across the eyes. The thorax tapers to a waist-like constriction at the level of the scutellum. The

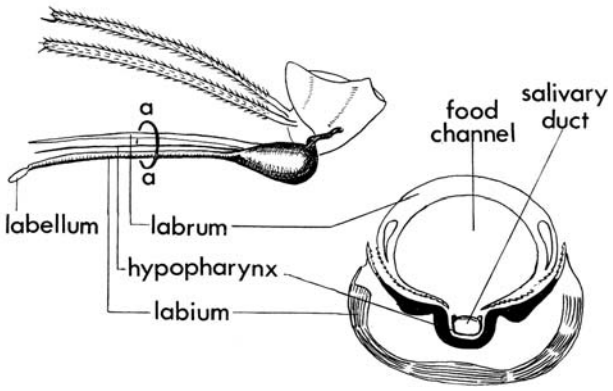


FIGURE 18.3 Details of proboscis and palps of *Glossina* species, with palps separated from the proboscis (*left*) and cross-section about midway along length (at a) of proboscis (*right*). *From Potts (1973); after Newstead et al. (1924).*

wings vary from hyaline to dusky, depending on the species. They are folded scissors-like over the back, with the tips extending slightly beyond the end of the abdomen. The tsetse wing has a distinctive, hatchet-shaped discal cell. This is formed by the fourth (medial) vein that curves anteriorly to produce a wing cell (discal cell) resembling the elongate handle of a hatchet attached to a thickened blade (Fig. 18.5).

The base of the abdomen is about equal in width to the head and thorax. Male tsetse can be readily distinguished from females by the presence of a prominent button-like hypopygium on the ventral surface of the posterior of the abdomen (Fig. 18.6). The morphological details of both male and female genitalia provide taxonomic characters that are used to distinguish tsetse species.

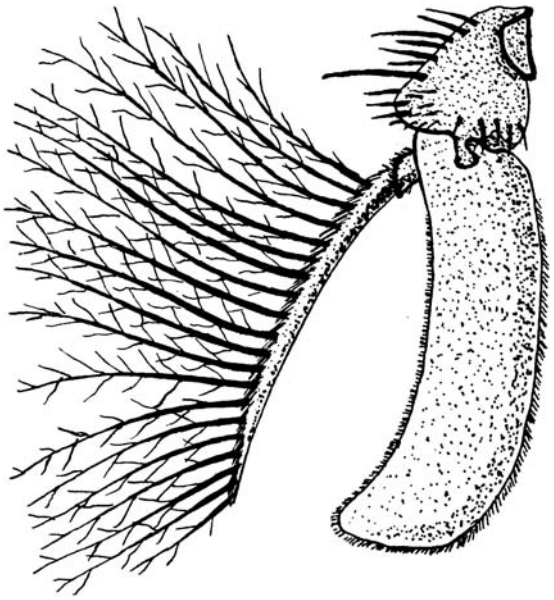


FIGURE 18.4 Antenna of *Glossina fuscipleuris*, showing plumose setae on arista characteristic of tsetse fly adults. *After Zumpt (1936).*

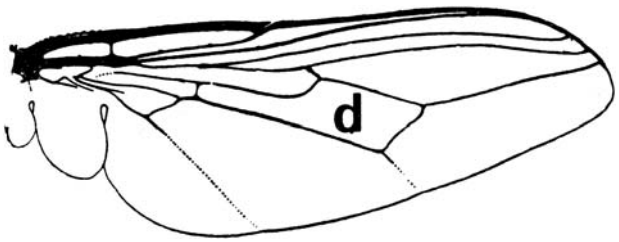


FIGURE 18.5 Wing structure and venation of adult tsetse fly (*Glossina* species), showing characteristic hatchet-shaped discal cell (d). *Modified from Potts (1973).*

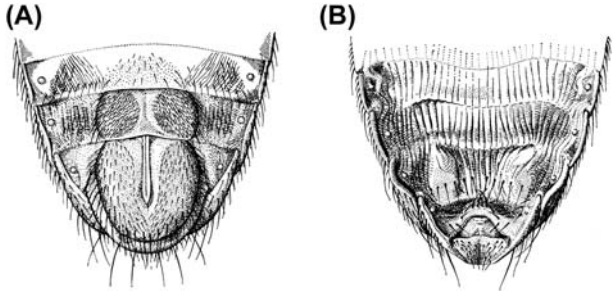


FIGURE 18.6 Posterior ends of the abdomens of *Glossina* adults, ventral view, showing sexual differences. (A) Male, with knob-like appearance of hypopygium drawn up into the abdomen; (B) Female, lacking knob-like hypopygium. *From Potts (1973).*

The alimentary tract is adapted for hematophagy. The strong musculature of the pharynx forms a cibarial pump for imbibing blood. The proventriculus secretes a peritrophic membrane that lines and protects the midgut. The midgut contains symbionts (Enterobacteriaceae) that provide compounds associated with vitamin B metabolism. Females devoid of these symbionts are unable to reproduce. The reproductive tract of the female fly is unusual compared with that of most oviparous dipterans; it is similar, however, to the reproductive system seen in the other hippoboscoid families (Hippoboscidae, Streblidae, and Nycteribiidae). Each of the two ovaries has only two ovarioles. The ovarian ducts form a common duct that expands to form a uterus in which one embryo at a time is retained during development. Associated with the uterus is a pair of specialized branched glands that produce nutrients for the developing tsetse larva. Because of this function, they are commonly called **milk glands**.

LIFE HISTORY

Tsetse adults of both sexes bite vertebrates and imbibe blood, the fly's only food. Unfed females are sexually receptive about 1 day after emergence from the puparium, whereas male tsetse require several blood meals before they

are fully fertile. At close range, the male visually locates a female; once contact is made, a pheromone in the cuticle of the female stimulates mating. The female endocrine system will induce ovulation only if mating lasts longer than an hour. Sperm are transferred in a spermatophore and are stored in the female's spermathecae. Once inseminated, the female remains fertile for life, but females will mate more than once.

About 9 days after copulation, the first ovulation of a single egg occurs and sperm are released through the spermathecal duct by dilation of a sphincter. The egg is positioned with the micropyle against the spermathecal duct opening, allowing for fertilization. The fertilized egg moves posteriorly into the uterus, where hatching occurs about 4 days later. The first-instar larva uses an "egg tooth" on its anterior end to rupture the chorion of the egg. The larva is retained in the uterus, where it is held against the uterine wall by a supporting structure called the **choriothete**. Secretions from the milk glands pool around the larval mouth and are easily ingested. The developing larva molts twice within the uterus, becoming a second-instar larva 1 day after hatching, and a third instar about 1.5 days later. The third-instar larva is fully developed about 2.5 days after the second molt, at which time it occupies most of the female's abdomen and is about equal in weight to the rest of the female's body. The female continues to ingest blood, albeit in progressively smaller amounts, as the larva grows.

About 9 days after ovulation, the fully developed, third-instar larva is deposited on the ground by the female. Shortly thereafter, the female ovulates again (within as little as 1 h after **larviposition**). After this first larviposition, a well-nourished female will deposit a third-instar larva about every 7–11 days, depending on the ambient temperature. The average interval for all tsetse species is 9–12 days. The ovaries and the ovarioles in each alternate in releasing a single egg at each ovulation, starting with the right ovary. Follicular relicts seen in dissected flies reflect the ovulation history of individual females and can help in estimating the longevities of wild-caught female flies.

Tsetse females generally live for about 20–40 days but may have a maximum life span of 3–4 months. The males typically mate only once or twice during their lives and apparently survive in the wild for 2–3 weeks (Glasgow, 1963; Potts, 1973). More accurate estimates of longevity of some species have become possible with newly developed fluorescence techniques that measure accumulated pteridines in tsetse tissues (Leak, 1999).

The full-size third-instar larva is cream colored and oval-shaped. It measures 3–8.5 mm in length, depending on the species, and has two prominent black knobs at the posterior end (Fig. 18.7). These conspicuous knobs are respiratory lobes that function only during and for a short time after intrauterine life. The active larva is deposited on the ground, usually in loose soil shaded by trees or other vegetation. The larva, which is negatively phototactic and positively thigmotactic, quickly burrows to 1.5–2.5 cm below the soil surface. Within a few hours of deposition, the larval integument hardens and darkens and the third-instar larva becomes an immobile brown to black puparium. About 2–4 days later, molting occurs within the puparial case and a true pupa is formed. A key for identifying puparia to species is given by Jordan (1993).

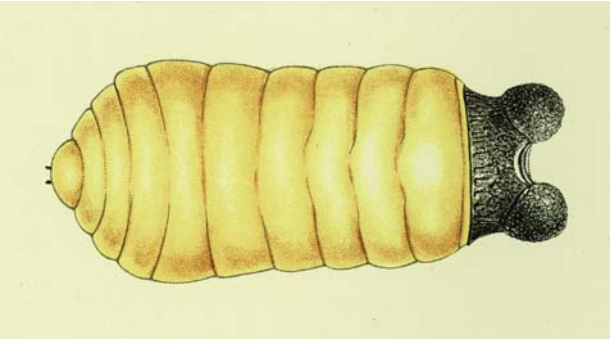


FIGURE 18.7 Larva of tsetse fly (*Glossina morsitans),* with distinctive knob-like respiratory lobes at posterior end. *From Newstead et al. (1924).*

Adult flies emerge about 30 days after formation of the puparium. As in all other cyclorrhaphan flies, eclosion involves the breaking of the circular puparial cap by a ptilinum. The teneral adult pushes its way to the surface of the substrate, where it rests for a short time, usually less than an hour, before it can fly. The teneral fly does not fully harden and the thoracic flight muscles do not completely develop until about 9 days later, after the fly has had at least a few blood meals (Glasgow, 1963; Potts, 1973; Lehane, 2005).

The low reproductive rate in tsetse is compensated by the extreme protection given to each larva by the female, by virtue of the viviparous mode of development. However, the low reproductive rate makes the impact of any loss of female flies greater than in species that mass-produce eggs.

BEHAVIOR AND ECOLOGY

Although tsetse are found over an area estimated to be at least 10 million square kilometers, the distribution of the flies is discontinuous. The areas they inhabit may extend to several hundred kilometers and form what have been traditionally called **fly belts**. Within these belts are patches of forest and bush where environmental conditions such as shade and high humidity are suitable for tsetse survival and reproduction. Local residents living in their vicinity are often aware of these areas of high tsetse concentrations. One or more species of tsetse usually are found where woody vegetation is at least 4.5 m high. In many cases, the presence of particular species of tsetse can be predicted by observing the types of shrubs and trees that occur in a given habitat. Rather than representing direct associations of

tsetse species with specific plants, the plant communities observed probably reflect differences in a variety of microhabitat factors that directly affect the survival of tsetse, such as the water content of the soil, the availability of mammalian hosts, and the occurrence of natural predators. Remotely sensed satellite data that provide identification of different types of vegetation over large geographic areas have been used to estimate distributions of different species of tsetse (Rogers et al., 1994; Robinson et al., 1997; Bouyer et al., 2006).

Tsetse flies are restricted in northern Africa by desert conditions and in southern Africa by the deserts of Namibia and Botswana and their lower ambient temperatures. Tsetse live in areas where the annual rainfall is at least 0.4 m/year. They require temperatures between 16 and 40°C, with optimal development occurring at 22−24°C; for this reason, the flies are not found at elevations above approximately 1,500 m.

The potential difficulty of males and females finding each other in low-density populations is apparently overcome in some species by the attraction of both males and females to large moving animals. Mating usually occurs on or in the vicinity of a host. Once they have mated, however, females and males tend to be more attracted to stationary animals. Tsetse feed on an array of hosts including reptiles and mammals, but rarely birds. Individual species and species groups have definite host preferences. These preferences are of considerable epidemiological significance in relation to the reservoir hosts of the pathogenic trypanosomes transmitted by the flies to humans and domestic animals.

Host preferences vary among tsetse species. Members of the *palpalis* group feed mostly on reptiles (e.g., crocodiles and monitor lizards) in their riverine and lacustrine habitats and on bushbuck, oxen, and occasionally smaller mammals and humans that visit these watering spots. Species of the *morsitans* group, living in scattered patches of vegetation in open country, feed mostly on the mammals of the savanna. In addition to showing a strong preference for warthogs, the savanna-dwelling tsetse feed on a diversity of mammalian species including bushbuck, buffalo, giraffe, kudu, rhinoceros, duiker, bush pig, and oxen. The one forest-dwelling species in the savanna group, *G. austeni*, feeds almost exclusively on suids such as bush pigs and forest hogs. The *fusca* group feeds on a variety of host species including bushbuck, buffalo and other cattle, giraffe, rhinoceros, elephant, hippopotamus, bush pig, river hog, porcupine, aardvark, and even the ostrich (Lehane, 2005). Humans are not the preferred hosts of any of these fly species. In some cases, people in the vicinity of other mammals will actually repel tsetse, whereas hungry flies will suck blood from humans who enter tsetse habitat.

Host attraction and host recognition are mediated by visual and olfactory cues. Their vision enables tsetse to react to a herd of moving cattle as far away as 180 m. The attraction of both male and female flies to large moving objects accounts for the common occurrence of tsetse attacking occupants of trucks and tourists in jeeps on safaris. Tsetse species in the *morsitans* group, living in open spaces, have shown the greatest attraction to host odors. Certain tsetse species are attracted to components of ox breath, such as carbon dioxide, acetone and octenol, and phenols found in mammalian urine (Willemse and Takken, 1994). Host odors have been shown to be attractive to tsetse from distances up to 100 m.

Although tsetse feed mostly in the daylight, adult feeding does occur at night, as in the case of *G. medicorum* that feeds on the nocturnal aardvark. Tsetse rarely fly for more than 30 min/day and are known to disperse up to about 1 km/day. They spend most of their time resting on vegetation. Most species are found below 3 m, where they rest on wood surfaces of trees during the day and on leaves at night. Recently engorged flies mostly rest with their heads directed upward, allowing excess water to be excreted away from their bodies. Hungry flies often rest horizontally, with the dorsal side down. When seeking a host, *Glossina* species can fly rapidly, reaching speeds above 6.5 m/s (approximately 25 km/h).

Host behavioral differences may account in part for the feeding preferences shown by tsetse species. Mammals that are heavily fed upon and irritated by other kinds of biting flies sometimes react with strong defensive behaviors, such as muscle twitching and rapid tail movements, that repel tsetse. Tsetse are more prone to start feeding on calm animals and often seem to prefer to feed on a host that is in the shade. The latter may be an adaptation to avoid reaching lethal body temperatures and may serve as a means of avoiding predation during feeding or just after, when the fly takes off and alights a short distance from its host (Glasgow, 1963).

Upon landing on a host, a tsetse fly grips the skin with its claws and applies pressure to the skin surface with its proboscis. The teeth and rasps on the labellum aid the labium in penetrating the skin. Strong back-and-forth movements of the fly's head cause the labium to rupture one or more capillaries in the skin, resulting in a hemorrhage within the bite site. The blood is rapidly sucked into the food canal of the labrum by the negative pressure produced by the cibarial pump in the fly's head. Saliva is pumped intermittently through the salivary canal of the hypopharynx into the wound. The saliva contains anticoagulant substances, including an antithrombin and an apyrase that inhibits platelet aggregation. As in other hematophagous insects that have anticoagulins in their saliva, tsetse flies presumably benefit from these substances by their role in increasing blood flow at the feeding site, thereby reducing feeding time and the vulnerability of the fly to host defenses. If a tsetse fly is disturbed while

penetrating the skin, it will rapidly withdraw the proboscis and fly away. However, once feeding begins, a tsetse is less likely to react to movement and physical stimuli that would normally cause it to escape (Glasgow, 1963; Lehane, 2005).

Tsetse engorge fully within about 1−10 min; the length of time depends in large part on how quickly the labium is able to rupture a capillary. The actual penetration of host skin occurs rapidly, whether it involves the thick hide of a rhino or a thin artificial feeding membrane. During feeding, a clear fluid is excreted from the anus. A tsetse fly imbibes about 0.03 mL of blood, and when fully engorged it weighs about two to three times its unfed body weight (Fig. 18.8). The ungainly fully fed insect slowly flies from the host (about 1.6 m/s) and lands on a nearby tree or another substrate. There, the fly continues to excrete anal fluid as a means of ridding itself of excess water, while concentrating its blood meal. About 40% of the blood meal weight is lost in the first 30 min after feeding. The rapid loss of excess fluid that begins during blood feeding helps the fed fly regain flight agility as quickly as possible. This helps it evade the defensive movements of the host and destruction by predatory flies, other arthropods, and vertebrate predators. A larva being carried by a female is especially vulnerable to loss just after the female has taken on the extra burden of a blood meal and has lost much of her maneuverability. Complete digestion of the blood meal occurs by about 48 h. The interval between blood meals varies, with a mean of 3−5 days.

Because tsetse feed exclusively on blood, their main source of energy is derived from protein. They depend on the amino acid proline as the major energy source for flight. The energy is produced by the partial oxidation of proline to alanine in flight muscle (Glasgow, 1963; Lehane, 2005). The unique metabolism of tsetse flies enables them to live in dry habitats in which blood is their sole source of nutrition and water, and to develop massive thoracic flight muscles that allow them to fly with heavy loads of blood and/or an internally developing larva.

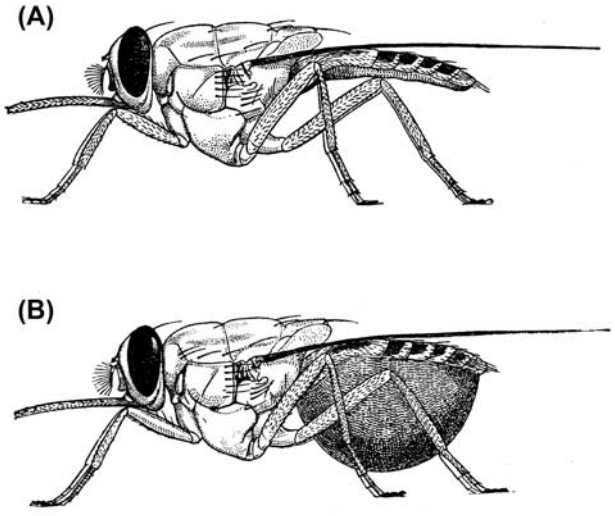


FIGURE 18.8 Tsetse fly (*Glossina morsitans*), female. (A) Before feeding; (B) after feeding. *From Austen (1903).*

PUBLIC HEALTH IMPORTANCE

For centuries, tsetse flies have had a great impact on human health in Africa, both as efficient vectors of trypanosomes that cause extreme human suffering in the form of African sleeping sickness and as vectors of trypanosomes that kill nonnative animals, preventing the development of animal domestication. The exclusion of cattle from most of the African continent has prevented their use as draught animals, as sources of human dietary protein from meat and milk, and as sources of manure and transport. The number of human disease cases (African sleeping sickness) has fluctuated with changes in the frequency of surveillance and control measures. In the mid-1960s, fewer than 5,000 cases were reported from all of Africa, but the disease increased since 1970; more than 38,000 cases were reported in 1998. Since that peak, the number of cases has fallen as the result of increased international programs, such as those supported by the World Health Organization (WHO) as well as those instituted by various governmental and nongovernmental organizations. Fewer than 3,000 new cases of West African sleeping sickness were reported in 2015, the lowest ever recorded by WHO, and 117 new cases of East African sleeping sickness were recorded in 2014. Although those numbers are thought to represent underestimates, there has been a dramatic decline in human African trypanosomiasis. Nevertheless, African animal trypanosomiasis continues to inhibit agricultural productivity and economic development.

African Sleeping Sickness

Trypanosomes were first associated with African sleeping sickness in 1903, after the British Tsetse Fly Commission, composed of David Bruce, David Nabarro, Aldo Castellani, and others, was sent to Africa to investigate outbreaks among British colonists. The clinical presentation of the disease was different in West African and East African countries; the East African form was much more severe. The trypanosomes isolated from the two forms of African sleeping sickness were named to reflect their geographic distributions (Fig. 18.9). The West African form was named *Trypanosoma gambiense* for Gambia. The parasite still causes sleeping sickness in Guinea, the Ivory Coast, southern Nigeria, Cameroon, Equatorial Guinea, Sao Tome, Congo, the Democratic Republic of the Congo, and Angola. In central Africa, the northern-most cases are in the

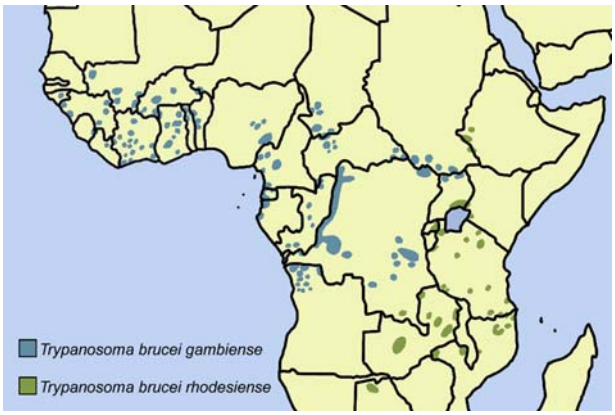


FIGURE 18.9 Distribution of *Trypanosoma brucei gambiense* and *Trypanosoma brucei rhodesiense. Source: WHO Report on Global Surveillance of Epidemic-prone Infectious Diseases—African trypanosomiasis.*

Central African Republic, northern Uganda, and Sudan. The East African form was named *Trypanosoma rhodesiense* for Rhodesia, now Zimbabwe, but this form now causes human disease north of Zimbabwe in southern Uganda, Rwanda, Burundi, Tanzania, Malawi, and Zambia. Most persons recently diagnosed with sleeping sickness (98%) have the West African form; of those, about 90% are from the Democratic Republic of the Congo, where the disease is considered epidemic, as it is in Chad, the Central African Republic, Sudan, Congo, Angola, and Uganda. The East African form is also epidemic in Uganda.

The two species of trypanosomes pathogenic in humans are morphologically identical and microscopically indistinguishable from *Trypanosoma brucei*, the species that causes some of the trypanosomal disease seen in domestic animals in Africa. For taxonomic purposes, *T. gambiense* and *T. rhodesiense* are considered subspecies of *T. brucei*, namely *Trypanosoma brucei gambiense* and *Trypanosoma brucei rhodesiense*, respectively (Hoare, 1972).

The term **African sleeping sickness** refers to the drowsy to comatose condition of acutely ill patients. This disease occurs in two clinical forms that result from differences in the pathogenicity of the trypanosomes transmitted by tsetse flies in West and East Africa. **West African trypanosomiasis** is a chronic illness involving mental deterioration and progressive weakening, and **East African trypanosomiasis** is an acute, rapidly fatal, febrile illness characterized by myocarditis and meningoencephalitis. The West African disease has been known since the 14th century when it was described by the Arab writer al-Qalqashandi. The first medical description of the disease was written in 1734 by John Atkins, who had served as a British Navy surgeon on slave ships traveling from West Africa to the West Indies. His description of the clinical signs of the disease were accurate but his ideas concerning its cause created an extremely prejudiced view of those afflicted with the "Negro lethargy." Atkins attributed the disease to the "natural weakness of the brain … brought about by lack of use" (McKelvey, 1973).

The first recorded large-scale epidemics of sleeping sickness occurred in the middle to late 19th century during the period of active European exploration and colonization of the African continent. Controversy exists regarding the role of British and other imperialistic activities in either stimulating these outbreaks or just recognizing and alleviating them. If nothing else, expanded navigation of waterways such as the Congo River, which led to increased trade and development by late−19th century colonists, facilitated the spread of tsetse and sleeping sickness from western and central Africa to eastern regions of the continent. Although there is strong historical evidence for this pattern of dispersal, extreme differences in the clinical presentations and pathogeneses of West and East African trypanosomiases led to the more probable explanation that the geographical forms of the parasite evolved independently as humans were exposed under different ecological conditions to parasites harbored by wild ungulate mammals.

More than three-quarters of a million people died from sleeping sickness in Africa between 1896 and 1906. With the development of modern drug treatments, mortality of patients who are diagnosed early in the disease has been greatly reduced but untreated cases are invariably fatal. An estimated 13 million Africans in 36 countries (Fig. 18.9) are at risk for infection by exposure to tsetse flies, but less than 10% of the at-risk population is monitored. Imported cases in the United States, mostly involving tourists to East Africa, average about one per year, but the risk for serious illness or death is great because of the American medical community's unfamiliarity with the disease.

In both West and East African trypanosomiasis, trypanosomes injected into the skin by a tsetse fly reproduce locally in connective tissue to form a nodule or chancre, called a **trypanoma**.

West African Trypanosomiasis

In the West African form, Stage 1 of the disease (the early or hemolymphatic stage), the skin lesion and associated erythematous swelling occur within a week or so after the bite. The trypanosomes then spread to the lymphatics and the resulting lymphadenopathy in the back of the patient's neck is called **Winterbottom sign**. This distinctive sign is diagnostic for the disease in a person who has been exposed to tsetse. Urticaria and rashes are common in this chronic form of the disease. The illness progresses as the trypanosomes continue to multiply in the lymphatic and circulatory systems. After months or years, Stage 2 (the late or neurologic stage) develops as the parasites enter the central

nervous system and produce symptoms such as behavioral and personality changes, hallucinations and delusions, drowsiness by day (i.e., sleeping sickness), tremors, and stupor. In untreated patients, stupor is often followed by convulsions and inevitably by death.

East African Trypanosomiasis

In the East African form, the disease progresses more rapidly than in the West African form. Stage 1 begins with an acute onset of fever, headache, and dizziness within a few days after the fly bite. There is usually little or no lymph node involvement. Instead, early circulatory system disease that includes myocardial and pericardial inflammation becomes clinically apparent as tachycardia and arrhythmias. Immune complexes composed of trypanosomal variant antigens and complement-fixing host antibodies stimulate release of proteolytic enzymes. Either the latter or toxin production combined with host autoantibodies causes damage to red blood cells, brain and heart tissue, and other organs. Anemia, thrombocytopenia, and disseminated intravascular coagulation followed by renal disease may precede Stage 2, during which there is localization of the trypanosomes in the blood vessels of the central nervous system. Hemorrhages, edema, and thrombosis leading to neuronal degeneration are common after inflammation of the cerebral arteries. Damage to brain tissue results in convulsions and other signs seen in the West African form, but much sooner, with death often occurring in weeks to months. Definite diagnosis of either form of the disease requires observation or isolation of trypanosomes from blood, cerebrospinal fluid, scrapings from a trypanoma (chancre), lymph node aspirates, or bone marrow.

Life Cycle of Trypanosomes

The developmental cycles of *T. b. gambiense* and *T. b. rhodesiense* in the flies appear to be identical, even though the tsetse vector species often are different. Trypanosomes ingested by a tsetse fly in a blood meal pass into the midgut, where their life cycle continues. The relatively short trypanosomes that enter the fly transform into thin procyclic forms, which then multiply and become **trypomastigotes** in about 3–4 days. These forms then multiply for about 10 days before they move to the posterior part of the midgut. There, they either pass through the peritrophic membrane or around its posterior open end into the ectoperitrophic space. The trypomastigotes move anteriorly in the ectoperitrophic space of the midgut and reach the junction with the proventriculus about 5 days later. The parasites elongate and penetrate the soft basal ring of the peritrophic membrane to return to the endoperitrophic space. Having returned to the endoperitrophic space of the proventriculus, the trypomastigotes migrate anteriorly through the esophagus and pharynx and enter the proboscis. They then enter the open end of the hypopharynx and migrate posteriorly through the salivary ducts into the salivary glands. In the lumen of the glands and attached to the epithelium, the trypomastigotes transform into **epimastigotes**, which multiply and form short **metatrypanosomes** that are infective to vertebrates. The metatrypanosomes are injected by **salivarian transmission** when the infected tsetse feeds on another vertebrate host.

An alternative trypanosome life cycle has been observed in some flies. In these flies, migration of trypomastigotes from the midgut to the salivary glands involves a different anatomical route. The trypomastigotes that have moved anteriorly in the ectoperitrophic space of the midgut enter the midgut cells, penetrate the wall of the midgut, and enter the hemocoel. From there, they migrate anteriorly and penetrate the hemocoel side of the salivary glands to reach the lumen. Whether the parasites move anteriorly via the gut lumen or the hemocoel, the complete trypanosome cycle in the fly usually takes 11–38 days but it may be as long as 80 days (Hoare, 1972; Aksoy et al., 2003).

In nature, small numbers of tsetse may be infective (e.g., with *T. brucei*, less than 0.4%) and still maintain high rates of trypanosome transmission. The physiological and ecological factors that attract tsetse flies to hosts enable continued cycling of the trypanosomes between insect and vertebrate hosts. In epidemic situations, tsetse and other bloodsucking flies, such as tabanids and stomoxyines, may transmit trypanosomes from person to person by mechanical transmission.

Epidemiologically, the West African and East African forms of the disease are different. The species of tsetse flies that act as vectors are different. The vertebrate host species and the degree to which humans are part of the natural life cycles of the trypanosomes are different.

In West Africa, the major vectors of sleeping sickness are tsetse species in the *palpalis* group. The medically most important species in this group include *G. palpalis*, *G. tachinoides*, and *G. fuscipes*. These species are found in shaded forested areas close to rivers, streams, and lakes. In West African trypanosomiasis, transmission to humans usually involves a solely human–tsetse cycle with no nonhuman reservoir hosts, although some domestic and wild animals are known to be infected with *T. b. gambiense*. Therefore, West African trypanosomiasis is considered an anthroponosis. Humans are most often infected by exposure within a peridomestic environment that encompasses forested waterways near their dwellings. Activities such as washing, bathing, gathering water for cooking, and wood-gathering for fires and construction

purposes place humans in the riverine habitats of the *palpalis* group of tsetse flies.

In East Africa, the major vectors of sleeping sickness are tsetse species in the *morsitans* group. The medically most important species in this group include *G. morsitans*, *G. pallidipes*, and *G. swynnertoni*. These species are found in vegetation such as tall grasses, thickets, and small groups of trees that occur in open savanna. An exception to the vector distinction between East and West Africa is the occurrence of *G. fuscipes* in Uganda and Kenya along the northern shore of Lake Victoria, where outbreaks of East African trypanosomiasis are associated with this *palpalis* group species. In East African trypanosomiasis, transmission involves a wild animal—tsetse—human cycle. Antelopes, the bushbuck (*Tragelaphus scriptus*), and the hartebeest (*Alcelaphus buselaphus*) are natural reservoirs for the trypanosome. Humans are incidental hosts. Therefore, East African trypanosomiasis is a zoonotic disease that can be designated an anthropozoonosis (Baker, 1974). In East Africa, African men and tourists generally have the highest risk of infection because it is the men who are the hunters and honey gatherers, and the tourists on safaris are the ones who most often enter the savanna areas where game animals and the *morsitans* group species thrive. Identification of a gene difference between *T. brucei brucei* and *T. brucei rhodesiense* has allowed rapid differentiation between these parasites found in nonhuman hosts. Recognition that cattle are often infected with *T. brucei rhodesiense* has led epidemiologists also to consider domestic livestock as important reservoirs of human infection (Maudlin, 2004).

The distribution of human trypanosomiasis in Africa is not as widespread as the distribution of tsetse species. The reasons are that many tsetse species do not readily feed on humans, and many potential vector species inhabit areas where there is little or no contact between the flies and humans.

Besides transmitting trypanosomes, tsetse flies have other direct but minor effects on public health. Some people who are particularly sensitive to arthropod antigens develop large skin rashes when bitten, and anaphylactic reactions to tsetse bites are known. However, as with most hematophagous species that are efficient vectors of human pathogens, people who are bitten usually feel little pain or only slight irritation.

VETERINARY IMPORTANCE

Tsetse-borne trypanosomiasis in both domestic and wild, nonhuman animals is caused by a number of trypanosome species. Most wild hosts in Africa are immune to these parasites or have inapparent or mild infections. Infections that cause disease in wildlife are rare because wild animals that develop pathological changes after infection are species that are rarely fed upon by tsetse (Ford, 1971). However, more than 30 species of wild animals native to Africa harbor trypanosomes that are pathogenic when transmitted to domestic animals. The disease associated with any of these infections is called **nagana**.

Nagana

Nagana, which killed camels and horses used by 19th-century missionaries, is now considered to have been a major factor in halting the spread of Islam through sub-Saharan Africa. The disease was known to 19th-century European explorers in Africa, who similarly lost large numbers of pack animals such as horses, mules, and oxen. In 1895, David Bruce recognized an association between the disease and tsetse flies. He named the new disease *nagana*, which is a Zulu word for "low or depressed in spirits" (McKelvey, 1973). Bruce also identified the etiologic agent that was later named *T. brucei* in his honor. In 1909, Friedrich Kleine demonstrated the biological transmission of trypanosomes by tsetse, which led to the elucidation of the life cycle of the trypanosome in the fly by Muriel Robertson in 1913. Bruce's earlier observations had involved only short-term mechanical transmission of the parasite.

Nagana continues to have a major impact on preventing the development of commercial domestic animal production over about one-third of the African continent. The scarcity of domestic animals results in a severe lack of animal protein for use as human food, a lack of draught animals for use in crop production, and the absence of manure suitable for use as fertilizer. Between 40 and 60 million cattle and millions of sheep, goats, horses, mules, pigs, and camels are at risk for infection in Africa. Unlike African sleeping sickness, in which human disease does not occur over the entire distribution of tsetse vectors, nagana occurs wherever tsetse are found, in addition to other areas where infection can be maintained by mechanical transmission by biting flies other than tsetse.

Chronic disease involving anemia and weakness is common (Fig. 18.10). Affected animals have reduced muscle mass, pendulous fluid-filled abdomens, scurfy skin, rough coats, and enlarged lymph nodes. Chronic fever and watery diarrhea are common. Most organ systems are infected and enlargement of the heart, lungs, liver, and spleen is often seen at necropsy. Chronically infected animals are unsuitable for use as food or as suppliers of manure for agricultural use. Early death may result from secondary infections. For further information on the clinical pathology of nagana, see Losos and Ikede (1972) and Jubb et al. (1985).

The six species of trypanosomes that cause nagana are listed with some of their wild and domestic animal hosts in Table 18.1. The trypanosomes are identifiable by

FIGURE 18.10 A naturally infected yearling cow sick with nagana (bovine trypanosomiasis), showing stunted growth and characteristic "nagana" pose. *From Murray et al. (1979), with permission of the Food and Agriculture Organization of the United Nations.*

differences in morphology, antigenicity, and DNA analysis. The trypanosome species differ to some extent in the anatomical sites where they develop in the tsetse vector. Hoare (1972) reviewed the morphology and life cycles of the tsetse-borne trypanosomes of Africa. *T. brucei*, a parasite of diverse domestic and wild mammals, has a developmental cycle in its tsetse vector identical to that of its human forms, *T. b. gambiense* and *T. b. rhodesiense*. It undergoes development during migration from the proboscis to the midgut and subsequently to the salivary glands. The vector cycle of *Trypanosoma suis*, a parasite of pigs, is almost identical to the latter forms and occupies the same anatomical sites. The vector cycles of *Trypanosoma congolense*, a parasite of all domestic animals, antelopes, and other wildlife, and *Trypanosoma simiae*, found in pigs, cattle, horses, camels, and warthogs, are almost identical to each other. These trypanosomes are never found in the salivary glands of the fly. Development is restricted to the midgut, proventriculus, esophagus, and the food canal of the labrum from which infective forms pass into a vertebrate host. In tsetse infections with *Trypanosoma vivax* and *Trypanosoma uniforme*, parasites of diverse vertebrate hosts (Table 18.1), development of the parasite is even more restricted; it is limited to the proboscis where the trypanosomes are found in the labrum and hypopharynx. The only tsetse-borne trypanosome found outside of Africa is *T. vivax*, which also occurs in Central and South

TABLE 18.1 Tsetse Vectors and Vertebrate Hosts of Human and Animal Trypanosomiases

Disease	Disease Agent	Vectors	Hosts
West African sleeping sickness	*Trypanosoma brucei gambiense*	*Glossina fuscipes, Glossina palpalis, Glossina tachinoides*	Human
East African sleeping sickness	*Trypanosoma brucei rhodesiense*	*Glossina morsitans, Glossina pallidipes, Glossina swynnertoni*	Human, antelope (bushbuck, hartebeest), cattle
Nagana	*Trypanosoma brucei*	*Glossina fuscipes, Glossina longipalpis, Glossina morsitans, G. palpalis, G. pallidipes, G. tachinoides*	All domestic mammals; antelope (e.g., impala, hartebeest, wildebeest); warthog, hyena, lion
	Trypanosoma suis	*Glossina brevipalpis, Glossina vanhoofi*	Suids (domestic pigs, warthogs)
	Trypanosoma congolense	*G. morsitans* group; *G. brevipalpis, G. fuscipes, G. palpalis, G. tachinoides, G. vanhoofi*	All domestic mammals, elephant, zebra, antelope (e.g., impala, hartebeest, duiker, gnu); giraffe, bushpig, hyena, lion
	Trypanosoma simiae	*Glossina austeni, G. brevipalpis, Glossina fusca, Glossina fuscipleuris, G. longipalpis, G. morsitans, G. pallidipes, G. palpalis, Glossina tabaniformis, G. tachinoides, G. vanhoofi*	Domestic pig, warthog, camel, horse, cattle
	Trypanosoma uniforme	*Glossina fuscipes, G. palpalis*	Cattle, goat, sheep, antelope (e.g., bushbuck, sitatunga, waterbuck); buffalo, giraffe
Nagana or Souma	*Trypanosoma vivax*	*G. morsitans* group; *G. fuscipes, G. palpalis, G. tachinoides, G. vanhoofi*	Domestic mammals (especially cattle, horse, mule); wild bovids, zebra, antelope (e.g., impala, hartebeest, gnu); giraffe, warthog, lion

Information compiled from Hoare (1972).

America. As in Africa, this trypanosome can be transmitted mechanically among cattle by tabanids and other hematophagous flies in Latin America.

The only nonmammalian trypanosome transmitted by tsetse is *Trypanosoma grayi*, a parasite of crocodiles. Its development in the fly is restricted to the posterior midgut, hindgut, and rectum. Infective metatrypanosomes are transmitted to crocodiles when they ingest infected flies.

The tsetse vectors of the trypanosomes causing nagana include species of the *palpalis*, *morsitans*, and *fusca* groups (Table 18.1). *Glossina* species of all three groups transmit *T. congolense*, *T. simiae*, and *T. vivax*. Species in both *palpalis* and *morsitans* groups transmit *T. brucei*. Only tsetse species of the *palpalis* group transmit *T. uniforme*, and only *Glossina* species of the *fusca* group transmit *T. suis*. Transmission of the latter two trypanosomes by single-vector group species restricts the distribution of *T. uniforme* to areas near waterways, and of *T. suis* to dense forested habitats, where their respective vectors live.

The wild animal hosts listed in Table 18.1 are reservoir hosts for the trypanosomes that cause disease in the domestic animals listed. Recognition of the geographic and ecological distribution of reservoir hosts and specific tsetse vector species can help determine where introduction of domestic animals is most likely to succeed; however, after the wide areas inhabited by the diverse reservoir and vector species are excluded, little habitat remains for the maintenance of healthy domestic animals.

PREVENTION AND CONTROL

Several approaches have been taken to prevent African sleeping sickness and nagana and to control tsetse vectors. These include intensive treatment and isolation of infected human and domestic animal hosts to try to break transmission cycles, use of trypanotolerant animals for agricultural purposes, laboratory research on the development of vaccines for human and nonhuman hosts, as well as chemical and ecological attacks on the tsetse flies themselves.

Drugs containing arsenic have been used for decades to treat human sleeping sickness, but they cause severe side effects. Recent recommendations provide some alternative drug treatments for some stages of the disease. Pentamidine, a synthetic aromatic diamidine, is the first-choice drug for treatment of first-stage infection with *T. b. gambiense.* Suramin, a polysulfonated naphthyl urea, is now the drug of choice for the hemolymphatic stage of *T. b. rhodesiense* human trypanosomiasis. Both pentamidine and suramin are administered by injection and neither drug is effective as a treatment for Stage 2 disease.

Diagnosis of the second stage of both forms of the disease requires lumbar puncture examination, which is used to demonstrate the presence of trypanosomes in the cerebral spinal fluid. Because of the nondescript signs and symptoms in patients with Stage 1 disease of both the western and eastern forms, the disease is often not recognized or is misdiagnosed at that stage. Stage 2 Gambian sleeping sickness is now treated with a combination of nifurtimox and eflornithine. The treatment regimen requires hospitalization because the drugs are administered by both intravenous and oral routes. Unfortunately, the only drug available to treat second-stage Rhodesian sleeping sickness is the highly toxic arsenical derivative, melarsoprol. It causes pain and fatal encephalopathy in up to 5% of treated patients. Doctors Without Borders (*Médecins sans Frontières*) is responsible for the supply and distribution of all human African trypanosomiasis drugs used in the world.

Samorin (isometamidium chloride) is given to cattle as a preventative, and Berenil (diminazene aceturate) is effective in treating infected domestic animals. Although marketed exclusively for veterinary purposes, the latter compound has been administered to large numbers of patients in Africa and has been effective in treating early stages of both forms of human trypanosomiasis.

Maintenance of noninfected human or domestic animal populations requires regular **surveillance** for trypanosomes. Examining blood for trypanosomes is still common. However, use of the card agglutination trypanosomiasis test for antibodies is proving more efficient for surveillance under field conditions.

The use of **trypanotolerant breeds** of domestic animals is being studied and new agricultural practices are being developed that enable Africans to breed native, normally wild, trypanotolerant animals for food and other domestic purposes. Indigenous cattle breeds such as N'dama, which tolerate nagana trypanosomes without developing disease, are the focus of investigations to determine mechanisms of tolerance, with the hope of selectively breeding or genetically engineering new tolerant breeds. Trypanotolerant game animals such as antelopes are being assessed for use in meat production. Trypanotolerant animals require careful handling because stress can trigger disease or death from their infections.

The development of an effective **vaccine** for domestic animals or humans has been thwarted by the presence of variant surface glycoproteins on the trypanosomes that enable the parasites to change their antigenicity in response to each wave of antibodies during the course of infection.

Tsetse populations have been directly attacked with insecticides and, historically, indirectly attacked by destroying tsetse resting and oviposition sites and wild hosts. The direct approach, used in attempts to eradicate the fly, involves aerial spraying and ground spraying from backpacks and trucks. The indirect approach, which generally is not tolerated today, involved cutting or burning vast areas of vegetation to destroy adult resting sites and puparia, and game hunting to rid large areas of sources of blood for tsetse. Development involving

building and paving, with its concomitant destruction of vegetation, has had effects similar to that from purposeful clearing of tsetse habitats. To be effective, most direct attacks on tsetse flies require frequent repetition of treatments, which are either costly or harmful to the environment. One direct approach that has been considered for general use is to treat cattle with an insecticide when they are run through an acaricide dip to kill ticks. In that way, cattle that are attacked by tsetse can also remove flies from the environment.

Another approach to tsetse control is the mass rearing and release of sterile male flies. In experimental tests, eradication has been achieved in fenced grazing areas as large as several hundred square miles, surrounded by tsetse-free buffer zones. The buffer zones are continually monitored for tsetse, and tsetse eradication is maintained with aerial or ground insecticides or insecticide-impregnated attractant baits to keep fertile flies from migrating into the eradication area. The African initiative (Pan African Tsetse and Trypanosomiasis Eradication Campaign) has begun employing the sterile male technique for early control in isolated areas. The wider use of such a program has been questioned for various practical and economic reasons. The extreme effort and expense required to maintain tsetse-free zones across the continent of Africa make the program impractical and the risks associated with releasing laboratory-bred flies that are capable vectors are too high for general use in Africa. Rational, cost-effective control measures that have succeeded in the past would appear preferable to any attempt at continent-wide eradication (Rogers and Randolph, 2002).

Any effective means of controlling tsetse flies requires constant surveillance followed by the use of control measures aimed at specific populations of flies. Historically, surveillance for tsetse involved African youths ("fly boys") walking around a designated route of about 1.6 km called a **fly round** and catching flies with hand nets. This method evolved into walking with an ox as bait (ox round), bicycling over longer fly rounds, or riding on the back of trucks and catching tsetse attracted to the vehicles. Because of the low density of tsetse, fly-round surveillance over small areas, sometimes combining hand-netting with sprayer backpacks, became a useful control technique.

Surveillance by adult trapping has a long history involving different trap designs and identification of tsetse attractants. As in many other hematophagous flies, the attraction of tsetse to dark objects and carbon dioxide, as well as other products of mammalian metabolism, has been put to practical use in designing tsetse traps. Tsetse behavior in relation to trapping methods and host odors has been reviewed by Colvin and Gibson (1992), Willemse and Takken (1994), and Green (1994).

Studies of traps and baits for surveillance have led to improvements that make control with these devices the method of choice. Much of the work on attractants and trapping was done by Glyn Vale and colleagues in Zimbabwe. His studies and those of others led to the development of traps of various kinds, such as those resembling hosts (e.g., Morris trap), biconical designs (e.g., Challier and Laveissiére trap), and those with square or rectangular cloth targets. One of the most effective targets is black and blue and baited with attractant components of ox breath or urine. Attractants include acetone, 1-octen-3-ol, and phenols (4-methyl- and 3-*n*-propyl). The target is designed so that it can be used with either an electrocution device or an insecticide. An unattended trap charged with a residual insecticide can be used to remove flies from the environment for 12−18 months, long enough to eradicate local populations of tsetse. Other methods being studied involve targets impregnated with insect growth regulators or chemosterilants, and combinations of these substances with tsetse sex pheromone. Former large-scale use of insecticides and animal baits has given way to less costly, nonpolluting, and more efficient selective control with traps or targets. The possibility that traps can be maintained at little cost by local landowners, such as pastoral farmers, removes the need for extensive interference by foreign agencies and places the responsibility for maintenance on those whose economic well-being is directly related to the success of the trapping program.

In surveys for trypanosomes, the accuracy of identifying infected flies and individual trypanosome species has been enhanced with the use of polymerase chain reaction (PCR) analysis. In Gabon, for example, tsetse flies were identified, dissected, and studied microscopically for the presence of trypanosomes. The microscopic examinations were followed by preserving the tsetse organs and PCR analysis was used to identify the trypanosome species present. Both human and animal pathogenic forms were identified. The trypanosome infection rate of the flies was found to be 31.9% using PCR versus 6.3% using microscopic examination.

Modern surveillance includes the use of detailed satellite images combined with global positioning satellite−guided ground surveys to produce mathematical models for recognizing different riverine forest community types. Landsat 7−detailed images have been used in this way in West Africa to classify types of vegetation and water cover, i.e., water, swamp forests, woodland savanna, shrub savanna, early crops, late crops, and bare ground. Ground surveys for tsetse using Varoua biconical traps (in which a cone of mosquito netting is placed over three panels of black and blue fabric with 120-degree angles between them) have been successfully used to provide collection data that can be correlated with the satellite-derived classification of riverine habitats. The identification of types of vegetative communities and the degree of fragmentation are indicators of the suitability of the habitats for tsetse and can be used to predict the distribution and abundance of flies

that enable the institution of selective control measures. Area-wide integrated pest management to control a tsetse population in a circumscribed area may include the use of trapping, insecticide-impregnated targets, live baits, and sterile insect technique.

These modern approaches have been used to assess the distribution of tsetse in Burkina-Faso and Senegal (Guerrini et al., 2008; Bouyer et al., 2010). The use of maps derived from satellite remote sensing combined with mathematical modeling has enabled recognition of natural and fragmented tsetse habitat. In West Africa, where forested areas have been cleared, riverine species of tsetse have adapted to survive in perennial crop areas, such as where citrus is grown, and in swamps with reeds and riparian thickets. Although transpiration from trees in forested riverine habitats maintains humidity levels critical for the survival of the flies, during the dry season, humidity is often provided by underground river networks, springs, or human irrigation activity. In periurban, densely populated areas, tsetse may live in artificial habitats such as parks and tree plantations.

In addition to the use of modern surveillance techniques combined with improved fly control methods to reduce fly populations, there continue to be genetic studies of ways to change trypanosome transmission capabilities and to identify aspects of fly reproduction that may be vulnerable to manipulation. As in experimental studies of triatomine symbionts, tsetse symbionts are being studied with the goal of engineering their genes to produce antitrypanosomal substances or to eliminate the vector capability of the host flies (Aksoy, 2003; Benoit et al., 2015).

Natural enemies of tsetse include puparial parasites such as ants and beetles, over 20 species of wasps, and at least 10 species of bombyliid flies (*Thyridanthrax* spp.). Predators of adults include spiders, odonates, asilids, and sphecid and vespid wasps. Field studies of predation of *G. pallidipes* puparia in Kenya suggest that more than 20% of all puparia may be killed by predators during their buried, 30-day developmental period. The parasites and predators of tsetse were reviewed by Mulligan (1970), Laird (1977), and Leak (1999).

The vast land mass over which tsetse flies occur is governed by the jurisdictions of 36 different countries, which with their own agendas relating to conservation and development makes it difficult to create a unified policy for the control of the flies. The Pan-African Tsetse and Trypanosomiasis Eradication Campaign was developed to address the tsetse problem as a serious impediment to sustainable agricultural development in most of the African countries south of the Sahara. Given the immense challenge of identifying the tsetse populations that pose threats to domestic animals and the adaptations of the flies to diverse nonuniform habitats, it would appear that controlling the flies within well-delineated geographical areas is a more realistic goal than eradicating the various tsetse species.

REFERENCES AND FURTHER READING

Aksoy, S. (2003). Control of tsetse flies and trypanosomes using molecular genetics. *Veterinary Parasitology, 115*, 125–145.

Aksoy, S., & Rio, R. V. M. (2005). Interactions among multiple genomes: Tsetse, its symbionts and trypanosomes. *Insect Biochemistry and Molecular Biology, 35*, 691–698.

Aksoy, S., Gibson, W. C., & Lehane, M. J. (2003). Perspectives on the interactions between tsetse and trypanosomes with implications for the control of trypanosomiasis. *Advances in Parasitology, 53*, 1–84.

Austen, E. E. (1903). *A monograph of the tsetse-flies [Genus Glossina, Westwood]*. London: Longmans & Co. (reprinted 1966 by Johnson Reprint Corp.). 319 pp.

Baker, J. R. (1974). Epidemiology of African sleeping sickness. In *"Trypanosomiasis and Leishmaniasis with special reference to Chagas' disease." Ciba foundation symposium, new series 20*. Amsterdam: Elsevier, 353 p.

Beard, C. B., O'Neill, S. L., Mason, P., Mandelco, L., Woese, C. R., Tesh, R. B., et al. (1993). Genetic transformation and phylogeny of bacterial symbionts from tsetse. *Insect Molecular Biology, 1*, 123–131.

Benoit, J. B., Attardo, G. M., Baumann, A. A., Michalkova, V., & Aksoy, S. (2015). Adenotrophic viviparity in tsetse flies: Potential for population control and as an insect model for lactation. *Annual Review of Entomology, 60*, 351–371.

Bouyer, J., Guerrini, L., Desquesnes, M., de la Rocque, S., & Cuissance, D. (2006). Mapping African animal trypanosomiasis risk from the sky. *Veterinary Research, 37*, 633–645.

Bouyer, J., Seck, M. T., Sall, B., Ndiaye, E. Y., Guerrini, L., & Vreysen, M. J. B. (2010). Stratified entomological sampling in preparation for an area-wide integrated pest management program: The example of *Glossina palpalis gambiensis* (Diptera: Glossinidae) in the Niayes of Senegal. *Journal of Medical Entomology, 47*, 543–552.

Buxton, P. A. (1955). *The natural history of tsetse flies*. London: H.K. Lewis & Co., Ltd., 816 pp.

Colvin, J., & Gibson, G. (1992). Host-seeking behavior and management of tsetse. *Annual Review of Entomology, 37*, 21–40.

de la Rocque, S., Michel, J. F., Bouyer, J., De Wispelaere, G., & Cuissance, D. (2005). Geographical information systems in parasitology: A review of potential applications using the example of animal trypanosomiasis in West Africa. *Parassitologia, 47*, 97–104.

Drugs for Neglected Diseases Initiative (DNDi) – Human African trypanosomiasis (2015). dndi.org (Click on "Sleeping Sickness" for a Human African Trypanosomiasis Fact Sheet).

Elsen, P., Amoudi, M. A., & Leclercq, M. (1990). First record of *Glossina fuscipes fuscipes* Newstead, 1910 and *Glossina morsitans submorsitans* Newstead, 1910 in southwestern Saudi Arabia. *Annales de Societé de Belge Médecin Tropicale, 70*, 281–287.

Ford, J. (1971). *The role of the trypanosomiases in African ecology–a study of the tsetse fly problem*. Oxford: Clarendon Press, 568 pp.

Glasgow, J. P. (1963). *The distribution and abundance of tsetse*. New York: Macmillan Co., 241 pp.

Green, C. H. (1994). Bait methods for tsetse fly control. *Advances in Parasitology, 34*, 229–291.

Guerrini, L., Bord, J. P., Ducheyne, E., & Bouyer, J. (2008). Fragmentation analysis for prediction of suitable habitat for vectors: Example of riverine tsetse flies in Burkina Faso. *Journal of Medical Entomology, 45*, 1180–1186.

Hoare, C. A. (1972). *The trypanosomes of mammals*. Oxford: Blackwell Sci. Publ., 749 pp.

Jordan, A. M. (1986). *Trypanosomiasis control and African rural development*. London: Longman, 357 pp.

Jordan, A. M. (1993). Tsetse-flies (Glossinidae). In R. P. Lane, & R. W. Crosskey (Eds.), *Medical insects and arachnids* (pp. 333–338). London: Chapman & Hall.

Jordan, A. M. (1995). Control of tsetse flies (Diptera: Glossinidae) with the aid of attractants. *Journal of the American Mosquito Control Association, 11*, 249–255.

Jubb, K. V. F., Kennedy, P. C., & Palmer, N. (1985). *Pathology of domestic animals* (Vol. 3). Academic Press, 527 pp.

Laird, M. (Ed.). (1977). *Tsetse: The future for biological methods in integrated control*. Ottawa: International Development Research Centre, 220 pp.

Leak, S. G. A. (1999). *Tsetse biology and ecology: Their role in the epidemiology and control of trypanosomiasis*. CABI Publishing, 568 pp.

Lehane, M. J. (2005). *Biology of blood-sucking insects* (2nd ed.). Cambridge University Press, 321 pp.

Losos, G. J., & Ikede, B. O. (1972). Review of pathology of diseases in domestic and laboratory animals caused by *Trypanosoma congolense, T. vivax, T. brucei, T. rhodesiense* and *T. gambiense*. *Veterinary Pathology (Suppl.), 9*, 71 pp.

Maudlin, I., Holmes, P. H., & Miles, M. A. (2004). *The trypanosomiases*. CABI Publ, 632 pp.

Mbang, N. O. A., Mawili-Mboumba, D. P., Chouaibou, M., Mavoungou, J., M'Batchi, B., & Bouyou Akotet, M. K. (2016). High frequency of (Kinetoplastida: Trypanosomatidae) type among (Diptera: Glossinidae) in a historic *Trypanosoma* foci in north-eastern Gabon: Preliminary study. *Journal of Medical Entomology, 53*, 945–948.

McAlpine, J. F. (1989). Phylogeny and classification of the Muscomorpha. In J. F. McAlpine, & D. M. Wood (Eds.), *Monograph No. 32, agriculture, Ottawa: Vol. 3. Manual of Nearctic Diptera* (pp. 1397–1518).

McKelvey, J. J., Jr. (1973). *Man against tsetse: Struggle for Africa*. Ithaca: Cornell University Press, 306 pp.

Mulligan, H. W. (1970). *The African trypanosomiases*. New York: Wiley-Interscience, 950 pp.

Murray, M., Morrison, W. I., Murray, P. K., Clifford, D. J., & Trail, J. C. M. (1979). *Trypanotolerance – a review* (Vol. 31, pp. 2–12). World Animal Review (FAO).

Newstead, R., Evans, A. M., & Potts, W. H. (1924). Guide to the study of tsetse-flies. In *Liverpool School of tropical medicine Memoir (new series) No. 1*.

Pépin, J., & Milord, F. (1994). .The treatment of human African trypanosomiasis. *Advances in Parasitology, 33*, 1–47.

Peters, W., & Pasvol, G. (2007). *Atlas of tropical medicine and parasitology*. p. 69, Fig. 196 (2nd ed.). New York: Year Book Publ.

Potts, W. H. (1973). Glossinidae (tsetse-flies). In K. G. V. Smith (Ed.), *Insects and other arthropods of medical importance* (pp. 209–249). London: British Museum (Natural History).

Quinn, T. C. (1996). African trypanosomiasis (sleeping sickness). In J. C. Bennett, & F. Plum (Eds.), *Cecil textbook of medicine* (20th ed., pp. 1896–1899). Philadelphia: W.B. Saunders Co.

Robinson, T., Rogers, D., & Brian, W. (1997). Mapping tsetse habitat suitability in the common fly belt of southern Africa using multivariate analysis of climate and remotely sensed vegetation data. *Medical and Veterinary Entomology, 11*, 235–245.

Rogers, D. J., Hendricks, G., & Slingenbergh, J. H. W. (1994). Tsetse flies and their control. *Revue scientifique et technique (International Office of Epizootics), 13*, 1075–1124.

Rogers, D. J., & Randolph, S. E. (1985). Population ecology of tsetse. *Annual Review of Entomology, 30*, 197–216.

Rogers, D. J., & Randolph, S. E. (2002). A response to the aim of eradicating tsetse from Africa. *Trends in Parasitology, 18*, 534–536.

Simarro, P. P., Cecchi, G., Paone, M., Franco, J. R., Diarra, A., Ruiz, J. A., et al. (2010). The atlas of human african trypanosomiasis: A contribution to global mapping of neglected tropical diseases. *International Journal of Health Geographic Research, 9*, 57. http://www.ij-healthgeographics.com/content/9/1/57.

Vale, G. A. (1993). Development of baits for tsetse flies (Diptera: Glossinidae) in Zimbabwe. *Journal of Medical Entomology, 30*, 831–842.

Willemse, L. P. M., & Takken, W. (1994). Odor-induced host location in tsetse flies (Diptera: Glossinidae). *Journal of Medical Entomology, 31*, 775–794.

World Health Organization. (1996). Geographical distribution of foci of gambiense and rhodesiense sleeping sickness. In *Vector biology and control division*. Geneva: WHO 96.140.

Zumpt, F. (1936). *Die Tsetsefliegen*. Jena: Fischer.

Zumpt, F. (1973). *The stomoxyine biting flies of the world*. Stuttgart: G. Fischer Verlag, 175 pp.

Chapter 19

Myiasis (Muscoidea, Oestroidea)

Philip J. Scholl[1], Douglas D. Colwell[2] and Ramón Cepeda-Palacios[3]

[1]United States Department of Agriculture, Agricultural Research Service (retired), Oxford, FL, United States; [2]Livestock Parasitology, Agriculture and Agri-Food Canada, Lethbridge Research and Development Centre, Lethbridge, AB, Canada; [3]Animal Science and Environmental Conservation Department, Autonomous University of Baja California Sur, La Paz, Mexico

Myiasis is the invasion of a living vertebrate animal by fly larvae. This invasion may or may not be associated with feeding on the tissues of the "host." Myiasis-causing flies are represented by a diversity of species. Some are rarely involved in myiasis, whereas for others it is the only way of life. Many of these same fly species also feed on carrion. Among flies, dietary proteins are required for growth, egg production, and development. Proteins may be obtained by adult flies, by their larvae, or by both. In the case of larval diets, proteins are assimilated, stored, and carried through the pupal stage for subsequent use by the reproducing adult fly. A larval diet rich in proteins dictates that there is less need for adults to seek proteins. Thus myiasis is a means of exploiting a rich protein source by the larva for its own growth and, in some cases, for reproduction by the adult.

Myiasis is classified based on the degree to which a fly species is dependent on a host. Three types of myiasis generally are recognized: accidental, facultative, and obligatory myiasis.

In **accidental myiasis**, also called **pseudomyiasis**, the fly larvae involved normally are not parasitic but under certain rare conditions can become so. This type of myiasis can occur, for example, when fly eggs or larvae contaminate foods that are subsequently ingested by an animal, as in the case of pomace flies and fruit flies (*Drosophila* spp.). The 50 or so fly species involved include those that typically are free-living in all stages and rarely are parasitic. In most cases these flies pass unharmed through the host's alimentary tract, but they can cause discomfort, nausea, diarrhea, and a plethora of related problems on their way through. In some cases, symptoms can be severe. Invasion of the alimentary tract can occur in two ways: either through ingestion of contaminated food or via retroinvasion through the host's anus. There is some doubt as to whether these cases are true myiasis because there is scant evidence that any fly development takes place after the ingested eggs hatch.

Facultative myiasis involves larvae that can be either free-living saprophages or parasites. These flies are opportunistic, having the ability to exploit living tissue. An example of facultative myiasis is the invasion of open sores on livestock by maggots of blow flies, which normally frequent carrion.

Kettle (1995) recognizes three types of facultative myiasis: **primary myiasis**, involving those species that can initiate myiasis; **secondary myiasis**, involving species that continue myiasis but only after it is started by primary species; and **tertiary myiasis**, involving species that join the primary and secondary species just before host death. Facultative myiasis species are the evolutionary bridge linking saprophagous feeders to those restricted to feeding on living tissues.

Many or even most of these facultative myiasis flies feed on dead, decaying tissues rather than invading healthy tissue (e.g., surgical maggots) but are able to shift from dead to living tissue and back again with alacrity (e.g., *Cochliomyia macellaria*, *Wohlfahrtia nuba*). In a sense, these are borderline parasites that can invade a sick or injured host and continuing their larval development after the death of that host. The adult flies are attracted to open wounds or chronic surface sores with purulent exudates.

In **obligatory myiasis,** the maggots of the fly species involved are always parasitic; they require a living host for their development. Examples are primary screwworms and bot flies. Included here are those species that cause **temporary myiasis**, which involves the intermittent contact between a fly larva and its host, such as nestling maggots and floor maggots. In this type of myiasis, the maggots do not keep continual contact with their host. Occasional

Medical and Veterinary Entomology. https://doi.org/10.1016/B978-0-12-814043-7.00019-4

parasitism of atypical hosts by obligate myiasis-producing flies is called **incidental myiasis**.

Myiasis also can be categorized in relation to the site of larval invasion, or subsequent development in the host. Thus, the descriptives *gastrointestinal*, *urogenital*, *ophthalmic*, *nasopharyngeal*, *auricular*, and *cutaneous* are antecedent to the word *myiasis*, indicating the general site of maggot infestation.

Gastrointestinal myiasis refers to fly larvae in the alimentary tract of a host. This can be accidental myiasis, such as the ingestion of false stable fly eggs or larvae in uncooked fruits, or obligatory myiasis,such as the development of horse stomach bots. **Enteric myiasis** refers specifically to the intestinal tract. **Urogenital myiasis** is the invasion of the urethra and/or genitalia by fly larvae. This occurs when a host is debilitated and the urogenital openings exposed, or when the host has a urogenital infection producing exudates that attract flies. Cases of urogenital myiasis usually involve blow flies and flesh flies. **Ophthalmic myiasis** is the invasion of eye tissues by fly larvae; most cases are caused by the sheep nose bot fly (*Oestrus ovis*); occasionally, other species of Gasterophilinae, Hypodermatinae, Cuterebrinae, Calliphoridae, and Sarcophagidae are involved (Panadero and Otranto, 2015). **Nasopharyngeal myiasis** is the invasion of nasal and deep oral cavities and recesses by fly larvae. As with urogenital myiasis, this often is associated with a microbial infection, but it also can be caused by nose bot flies in healthy hosts. **Auricular myiasis** is the invasion of ears by fly larvae, usually caused by blow flies or flesh flies. **Cutaneous myiasis** involves invasion of the skin, usually by blow flies, flesh flies, screwworms, or certain bot flies. When cutaneous myiasis is associated with a break, laceration, or open sore in the host's skin, it is called **traumatic myiasis**.

Myiasis apparently has evolved along different lines in different groups of flies. Gastrointestinal myiasis and urogenital myiasis, for example, appear to represent a transition from species contaminating host foods to those associated with host excretions. They usually involve free-living species. Obligatory cutaneous myiasis appears to have evolved from carrion-breeding flies and from predaceous flies that prey on them. The origin of obligatory nasopharyngeal myiasis is less clear but probably is linked with host secretions associated with upper respiratory infections. Ophthalmic myiasis and auricular myiasis are characteristically either accidental or incidental in nature, resulting in damage to tissues at those respective sites. Temporary myiasis appears to have evolved from nest associates or lair-frequenting scavenger species that fed on organic morsels.

TAXONOMY

Myiasis-producing species are all flies belonging to the Order Diptera. These flies are found in either the suborder Nematocera (adults with long, segmented antennae) or Brachycera (adults with short, single-segmented antennae called arista) (Fig. 19.1A). Nematocera include less important

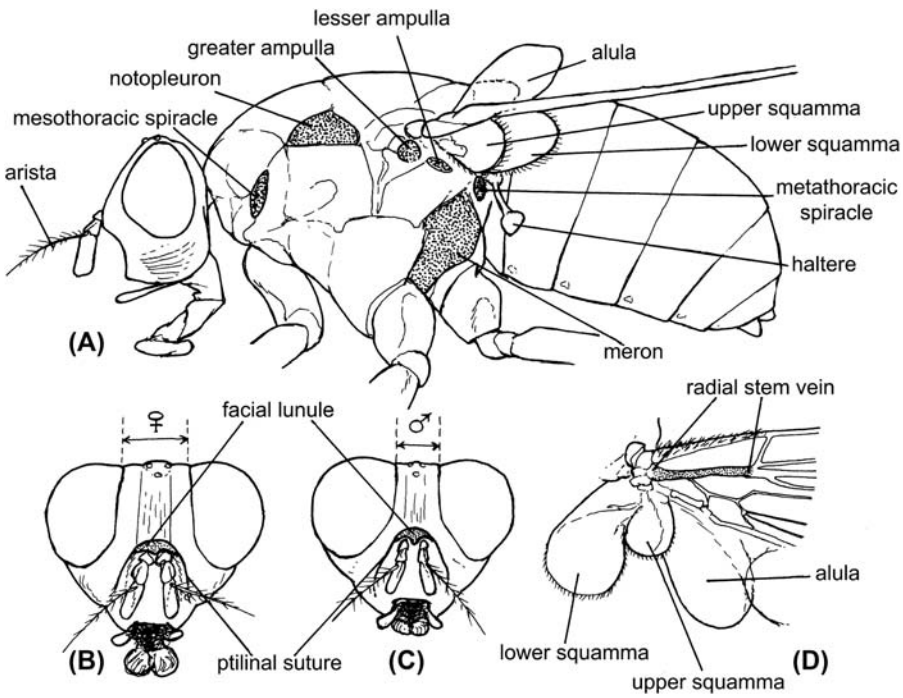


FIGURE 19.1 External morphology of representative adult muscoid fly. (A) Lateral view. (B) Frontal view, female. (C) Frontal view, male. (D) Dorsal view of wing base. *Original by E. Paul Catts.*

superfamilies such as Tipuloidea and Psychodoidea. The Brachycera is subdivided into the Cyclorrapha infraorder (species forming a barrel-like puparium with a circular opening used by the imago to exit, Fig. 19.2); the Schizophora subtaxon (true flies with a ptilinal sac, a special structure in the face used to escape the puparium, Fig. 19.1C); and the Calyptratae (adults that possess calypters or squamae, two lobes located at the posterior wing base, Fig. 19.1D).

The clear majority of species involved in myiasis are members of two superfamilies and six families of calyptrate flies: Muscoidea (Anthomyiidae, Fanniidae, and Muscidae) and Oestroidea (Calliphoridae, Sarcophagidae, and Oestridae). A dozen other families in eight superfamilies include species reported to cause myiasis; however, with the exception of the nest skipper fly (Neottiphilidae) and 10 species of Australian frog flies (Chloropidae), these cause accidental myiasis only. In contrast, all species of bot flies (Oestridae) cause obligatory myiasis. Myiasis-causing species among the muscids, calliphorids, and sarcophagids are typically facultative or obligatory myiasis producers. Table 19.1 shows the taxonomic relationships and associated types of myiasis for those flies known to invade living hosts.

The Anthomyiidae are a large family with more than 100 genera worldwide. Although members are called root maggot flies, the larvae occur in a wide range of habitats other than roots. Cladistically, the fanniids are a sister group to the muscids and often are included as a subfamily of the Muscidae. Most muscids are house fly-like in appearance, having a rather drab coloration with dorsal, longitudinal stripes on the thorax. Muscidae is a very large family with worldwide distribution that includes species typically associated with excrement and decaying plant matter.

The superfamily Oestroidea is composed of six families: Calliphoridae, Sarcophagidae, Oestridae, Tachinidae, Rhinophoridae, and Mystacinobiidae. The first five of these include parasitic species. Tachinids and rhinophorids parasitize invertebrate hosts, mostly other insects, and are not involved in myiasis. Adult mystacinobiids are phoretic, not parasitic, on bats.

Members of the Calliphoridae are called **blow flies**. This large family includes more than 1,000 species worldwide. Most have a polished or metallic blue, green, or bronze appearance and are also sometimes called **bottle flies**. There is confusion and continuing speculation concerning the phylogenetic relationships within this family. The major subfamilies include Calliphorinae, Chrysomyinae, Mesembrinellinae, Polleniinae, and Rhiniinae. Other subfamilies contain specialized genera or genera with limited distributions. The largest subfamily, Rhiniinae, includes about 40 genera of Old World blow flies. The Mesembrinellinae are large, showy tropical blow flies, whereas the Calliphorinae are the more temperate-climate bottle flies. The subfamily Chrysomyinae includes the economically important **screwworms** and the bird-nest blow flies. Members of the subfamily Rhiniinae are of no significance regarding myiasis.

The Sarcophagidae are the **flesh flies,** with some 2,000 species distributed worldwide. Many of these are parasitic on hymenopterous hosts or are predaceous on other insects, but a few cause myiases. Members of two of the four

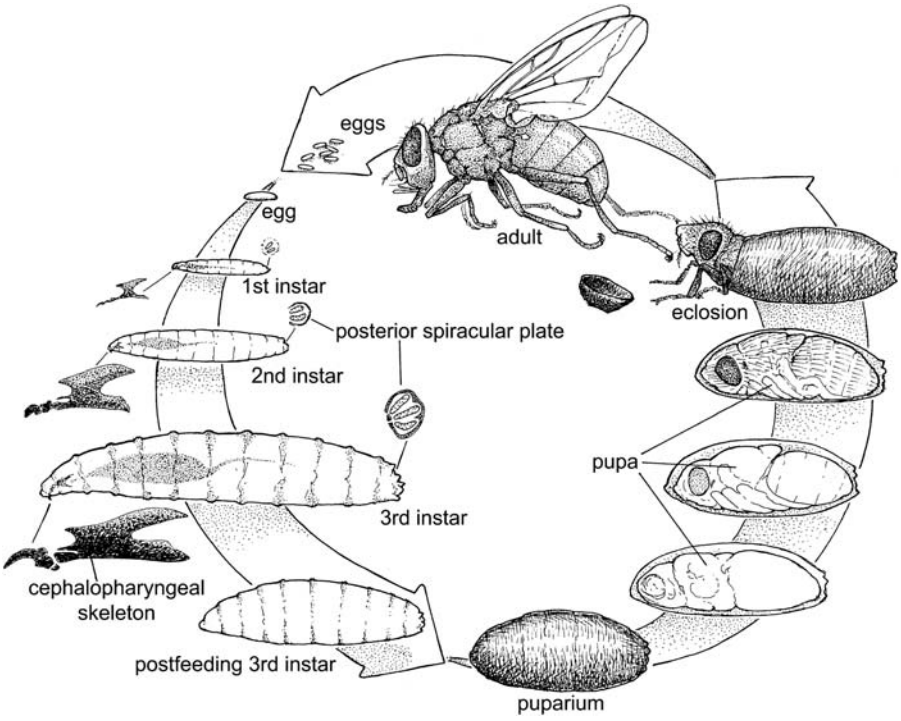


FIGURE 19.2 Typical life cycle of muscoid and oestroid flies. *Original by E. Paul Catts.*

TABLE 19.1 Taxonomic Relationships of Flies Known to Cause Myiasis; Accidental (A), Facultative (F), or Obligatory (O)

Superfamilies and Families	Common Names	Genera and Species	Type of Myiasis
Tipuloidea			
Tipulidae	Crane flies	(unspecified)	Gastrointestinal (A)
Psychodoidea			
Psychodidae	Moth flies	*Psychoda* (3 spp.)	Gastrointestinal, urogenital (A)
		Psychoda albipennis	
		Clogmia sp.	
Stratiomyoidea			
Stratiomyidae	Black soldier fly	*Hermetia illucens*	Gastrointestinal (A)
Asiloidea			
Therevidae	Stiletto flies	*Thereva* sp.	Gastrointestinal (A)
Platypezoidea			
Phoridae	Humpback flies, scuttle flies	*Megaselia* (3 spp.)	Gastrointestinal (A), traumatic (F)
		Megaselia scalaris	
		Megaselia spiracularis	
Syrphoidea			
Syrphidae	Flower flies	*Eristalis* (2 spp.)	Gastrointestinal (A)
Tephritoidea			
Piophilidae	Cheese skipper fly	*Piophila casei*	Gastrointestinal (A)
Neottiophilidae	Nest skipper fly	*Neottiophilum praeustrum*	Cutaneous (O)
Ephydroidea			
Drosophilidae	Pomace flies, fruit flies	*Drosophila melanogaster*	Gastrointestinal (A)
Carnoidea			
Chloropidae	Australian frog flies	*Batrachomyia* (10 spp.)	Cutaneous (O)
Muscoidea			
Anthomyiidae	Anthomyiid flies	*Hylemya* (2 spp.)	Gastrointestinal(A)
Fanniidae	Lesser house fly	*Fannia canicularis*	Gastrointestinal, urogenital, traumatic (A)
	Latrine fly	*Fannia scalaris*	
Muscidae	House fly	*Musca domestica*	Gastrointestinal, urogenital, traumatic (A)
	False stable fly	*Muscina stabulans*	Gastrointestinal, traumatic (A)
	—	*Muscina* (2 spp.)	Gastrointestinal, traumatic (A)
	—	*Hydrotaea rostrata*	Gastrointestinal, traumatic (A)
	Tropical nest flies	*Passeromyia* (3 spp.)	Cutaneous (O)
	Tropical nest flies	*Mydaea* (25 spp.)	Cutaneous (O)
	Neotropical nest flies	*Neomusca* (*Philornis*) (35 spp.)	Cutaneous (O)

TABLE 19.1 Taxonomic Relationships of Flies Known to Cause Myiasis; Accidental (A), Facultative (F), or Obligatory (O)—cont'd

Superfamilies and Families	Common Names	Genera and Species	Type of Myiasis
Oestroidea			
Calliphoridae	Green bottle flies	*Bufolucilia* spp.	Traumatic, cutaneous, gastrointestinal, nasopharyngeal, auricular (O)
		Lucilia (=Phaenicia) spp.	Traumatic, cutaneous, gastrointestinal, nasopharyngeal, urogenital, auricular (F)
	Blue bottle flies	*Calliphora* spp.	Traumatic, cutaneous, gastrointestinal, nasopharyngeal, urogenital, auricular (F)
		Eucalliphora spp.	
		Paralucilia sp.	
		Protophormia sp.	
		Cynomya sp.	
	Black blow fly	*Phormia regina*	
	Primary screwworm	*Cochliomyia hominivorax*	Traumatic (O)
	Secondary screwworm	*Cochliomyia macellaria*	Traumatic (F)
	Old World screwworm	*Chrysomya bezziana*	Traumatic (O)
	—	*Chrysomya* (9 spp.)	Traumatic (F)
	Bird blow flies	*Protocalliphora* (90 spp.)	Traumatic (O)
	Tumbu fly	*Cordylobia anthropophaga*	Traumatic (O)
		Cordylobia (5 spp.)	Cutaneous (O)
	African mouse/Lund's fly	*Auchmeromyia senegalensis*	Traumatic (O)
	Congo floor maggot	*Auchmeromyia* (4 spp.)	Traumatic (O)
	African suid maggots	*Pachychoeromyia praegrandis*	Traumatic (O)
	—	*Trypocalliphora linderi*	Traumatic (O)
	Asian deer/water buffalo skin maggots	*Booponus* (4 spp.)	Cutaneous (O)
		Neocordylobia (2 spp.)	Cutaneous (O)
		Pachychocaromyia praegrandis	Traumatic (O)
		Trypocalliphora linderi	Traumatic (O)
	Indian elephant skin maggot	*Elephantoloemus indicus*	Cutaneous (O)
Sarcophagidae	Flesh flies	*Sarcophaga* spp.	Traumatic (F), gastrointestinal (A)
		Wohlfahrtia (4 spp.)	Traumatic (O)
	Ground squirrel fly	*Neobellieria citellivora*	Cutaneous (O)
	Lizard flesh fly	*Anolisomyia* sp.	Cutaneous (O)
	Turtle flesh fly	*Cistudinomia cistudinis*	Cutaneous (O)
	Toad flesh fly	*Notochaeta bufonovoria*	Cutaneous (O)
	Lizard egg fly	*Eumacronychia nigricornis*	Cutaneous (O)

Continued

TABLE 19.1 Taxonomic Relationships of Flies Known to Cause Myiasis; Accidental (A), Facultative (F), or Obligatory (O)—cont'd

Superfamilies and Families	Common Names	Genera and Species	Type of Myiasis
	Sea turtle egg fly	*Eumacronychia sternalis*	Cutaneous (O)
	Terrapin egg fly	*Metoposarcophaga importuna*	Cutaneous (O)
Oestridae	Bot and grub flies	(29 genera, 166 spp.)	Cutaneous, gastrointestinal, nasopharyngeal (O)
Cuterebrinae	New World skin bot flies	(2 genera, 58 spp.)	Cutaneous (O)
	Rodent and rabbit bot flies	*Cuterebra* (57 spp.)	Cutaneous (O)
	Howler monkey bot	*C.* (=*Allouattamyia*) *baeri*	Cutaneous (O)
	—	*C.* (=*Pseudogametes*) (2 spp.)	Cutaneous (O)
	—	*C.* (=*Metacuterebra*) (13 spp.).	Cutaneous (O)
	—	*C.* (=*Rogenhofera*) (6 spp.)	Cutaneous (O)
	Tórsalo, human bot fly	*Dermatobia hominis*	Cutaneous (O)
Hypodermatinae	Old World skin bot flies	(10 genera, 32 spp.)	Cutaneous (O)
	Common cattle grub	*Hypoderma lineatum*	Cutaneous (O)
	Northern cattle grub	*Hypoderma bovis*	Cutaneous (O)
	Reindeer grub	*Hypoderma tarandi*	Cutaneous (O)
	Deer and yak warble flies	*Hypoderma* (3 spp.)	Cutaneous (O)
	—	*Ochotonia lindneri*	Cutaneous (O)
	Old World rodent grubs	*Oestromyia* (5 spp.)	Cutaneous (O)
	—	*Oestroderma potanini*	Cutaneous (O)
	Saiga warble fly	*Pallisiomyiaantilopum*	Cutaneous (O)
	Pavlovsky's gazelle warble fly	*Pavlovskiata subgutturosae*	Cutaneous (O)
	—	*Portschinskia* (7 spp.)	Cutaneous (O)
	—	*Przhevalskiana* (6 spp.)	Cutaneous (O)
	Antelope warble flies	*Strobiloestrus* (3 spp.)	Cutaneous (O)
	Unknown host	*Gruninomyia mira*	Cutaneous (O)
Oestrinae	Nose bot flies	(9 genera, 34 spp.)	Nasopharyngeal (O)
	Deer nose bots	*Cephenemyia* (8 spp.)	Nasopharyngeal (O)
	Camel nose bot	*Cephalopina titillator*	Nasopharyngeal (O)
	Tuberculous nasal bots	*Gedoelstia* (2 spp.)	Nasopharyngeal (O)
	Hairy nasal bots	*Kirkioestrus* (2 spp.)	Nasopharyngeal (O)
	Sheep nose bot	*Oestrus ovis*	Nasopharyngeal, ocular (O)
	———	*Oestrus* 5 spp.	Nasopharyngeal (O)
	African elephant throat bot	*Pharyngobolus africanus*	Nasopharyngeal (O)
	Deer and gazelle throat bots	*Pharyngomyia* (2 spp.)	Nasopharyngeal (O)

TABLE 19.1 Taxonomic Relationships of Flies Known to Cause Myiasis; Accidental (A), Facultative (F), or Obligatory (O)—cont'd

Superfamilies and Families	Common Names	Genera and Species	Type of Myiasis
	Horse nasal bot fly	*Rhinoestrus purpureus*	Nasopharyngeal (O)
	Nasal bot flies	*Rhinoestrus* (10 spp.)	Nasopharyngeal (O)
	Kangaroo throat bot fly	*Tracheomyia macropi*	Tracheal (O)
Gasterophilinae	Stomach bot flies	(5 genera, 17 spp.)	
	Horse stomach bots	*Gasterophilus* (9 spp.)	Gastrointestinal (O)
	Common horse bot	*G. intestinalis*	Gastrointestinal (O)
	Horse throat bot	*G. nasalis*	Gastrointestinal (O)
	Horse nose bot	*G. haemorrhoidalis*	Gastrointestinal (O)
	Broad-bellied horse bot	*G. nigricomus*	Gastrointestinal(O)
	Dark-winged horse bot	*G. pecorum*	Gastrointestinal (O)
	Black elephant stomach bot	*Cobboldia elephantis*	Gastrointestinal (O)
	Blue elephant stomach bot	*C.* (=*Platycobboldia*) *loxodontis*	Gastrointestinal (O)
	Green elephant stomach bot	*C.* (=*Rhodhainomyia*) *roveri*	Gastrointestinal (O)
	Rhinoceros stomach bots	*Gyrostigma* (3 spp.)	Gastrointestinal (O)
	African elephant skin bot	*Neocuterebra squamosa*	Cutaneous (O)
	African elephant foot bot	*Ruttenia loxodontis*	Cutaneous (O)

subfamilies include myiasis-causing species, the Sarcophaginae and Miltogramminae.

The Oestridae are the **bot flies**, with about 180 species worldwide. Adult flies are robust with appearances varying from bee-like (very effective mimics) to nearly hairless with numerous wartlike structures (e.g., *Oestrus ovis*). Larvae of all of these cause obligatory myiasis. They are placed in four subfamilies: Cuterebrinae, Hypodermatinae, Oestrinae, and Gasterophilinae (Wood, 1987; Pape, 2001). Phylogenetic relationships among these subfamilies have been recently described by Pape (2006). The rationale for combining the previously grouped three (e.g., Zumpt, 1965) or four (e.g., Guimarães and Papavero, 1999) families into a single family was discussed by Colwell et al. (2006).

For general taxonomic information and keys for identifying adults and larvae of muscoid and oestroid flies, see James (1947), Curran (1965), Zumpt (1965), Greenberg (1971), McAlpine et al. (1981, 1987, 1989), Furman and Catts (1982), Smith (1986), Lane and Crosskey (1993), Wall and Shearer (1997), and Guimarães and Papavero (1999). Additional sources for identifying the immature stages are Liu and Greenberg (1989) and Stehr (1991). Calliphorid eggs can be identified with the report by Greenberg and Singh (1995). The Calliphoridae are discussed by Hall (1948), Dear (1985), Ribeiro and Carvalho (1998), Carvalho and Ribeiro (2000), and Whitworth (2006). The Oestridae are treated by Papavero (1977), Wood (1987), Pape (2001), and Colwell et al. (2006).

MORPHOLOGY

Members of the superfamilies Muscoidea and Oestroidea are distinguished from other calyptrate flies by possessing both a **ptilinal suture** and **facial lunule** (Fig. 19.1B and C): the antennal pedicel with a complete dorsal seam and flagellum composed of a single compound segment bearing an arista or stylus; and a bulbous, greater ampulla below the wing base (Fig. 19.1A). All oestroid flies have a vertical row of bristles on the thoracic meron (Fig. 19.1A). The

adult Muscoidea are distinguished from the Oestroidea by lacking bristles on the thoracic meron even though the rest of the body is bristled.

Larvae of the muscoid and oestroid families are commonly called **maggots**. The larvae of muscoid flies usually lack armature, or body spines, whereas oestroid larvae possess armature ranging from sparse segmental belts of spines to a rather complete spiny vestiture. The third larval instars of both superfamilies can be distinguished by the form and number of slits in the **posterior spiracular plates** and by the size and shape of the internal **cephalopharyngeal skeleton** (Fig. 19.2). The body form of muscoid maggots is peglike and gradually tapers from a blunt posterior end to a pointed anterior end. Larvae of *Chrysomya rufifacies* and species in the genus *Fannia* are atypical, bearing segmental protuberances. Larvae of blow flies and flesh flies are more cylindrical than those of muscoid flies; they are tapered more abruptly at each end. Larvae of these 2 families can be distinguished by the slant of the inner posterior spiracular slits. The slits of blow flies slant toward the midline, whereas those of flesh flies slant away from the midline.

Mature bot fly maggots, some of which are called **grubs**, are much larger and thicker in cylindrical form than those of other oestroids. Except for the larvae of Gasterophilinae and Cuterebrinae, which possess elbowed or highly sinuous posterior spiracular slits, respectively, oestrid larvae have porous posterior spiracular plates that mask the more typical arrangement of slits.

The **puparium** is a strong protective case that contains the developing pupa and is formed from the integument of the last larval instar. All puparia except those of oestroids can be separated at the family level by examining remnant larval features (mouthparts and posterior spiracles). In contrast, oestrid puparia exhibit overall morphological forms that characterize each subfamily.

Among the adult muscoids, the wing venation of anthomyiids differs from muscids by the length of the anal vein. Some muscid genera (e.g., *Morellia*, *Neomyia,* and *Hydrotaea*) have a metallic or polished abdomen, superficially resembling blow flies. Most adult muscoid flies have sucking, rasping, or sponging mouthparts. A few (e.g., *Stomoxys*, *Haematobia*) possess a hardened labium as a piercing structure.

Among the oestroid flies, calliphorid adults have well-developed sponging-sucking mouthparts, the thoracic meron has a vertical row of bristles, and the postscutellum is small. Typically, these flies are metallic blue, green, or bronze in body coloration. Members of the major subfamilies Chrysomyinae and Calliphorinae differ in the presence or absence of hairs on the **radial stem vein** of the wing (Fig. 19.1D).

Adult sarcophagids also have sponging mouthparts, the thoracic meron is bristled, the postscutellum is small or absent, and three or four notopleural setae are usually present. Body coloration is typically gray and black, giving them the general appearance of large houseflies. Their color patterns, however, are more distinct than those of muscoid flies. The abdomen typically displays a tessellated, or checkerboard, pattern of gray and black.

Unlike other oestroid flies, adult oestrids lack head and body bristles, except for the bristles on the meron. They are medium-sized to large, hairy flies that either resemble bees or are hairless, often said to resemble bird droppings. They possess small antennae and relatively small sponging-sucking mouthparts, which in most instances are atrophied and nonfunctional. All except members of the Gasterophilinae have large wing *squammae* (Fig. 19.1D).

LIFE HISTORY

The life history of muscoid and oestroid flies follows the typical holometabolous and cyclorrhaphous pattern of four stages: egg, larva (three instars), pupa (in a puparium), and adult (Fig. 19.2). Male flies are usually smaller and emerge earlier than females. All of these flies form the puparium free from the larval substrate. Generally, given an adequate larval diet, more time in the developmental life cycle of an individual is spent in the pupal stage than in either that of the egg or larva. Among the oestrid flies, the cuterebrines have a long pupal stage that allows overwintering free from the host. Other bots have a prolonged larval period as an adaptation for overwintering within the protection of their host. Pupariation (puparium formation) is typically preceded by a wandering, postfeeding period by the last larval instar.

Adult longevity among these flies varies depending on environmental conditions. Longevity ranges from as little as one to several days among stomach bot and cattle grub flies, to 2 weeks for other bot flies, to 1 month or more among muscoids and other oestroids. In general, bot fly adults have a shorter life because they do not feed or imbibe water.

All myiasis-causing female flies have meroistic polytrophic ovaries, and they either oviposit or larviposit, but the degree of embryo development within the egg prior to deposition differs. Usually eggs are fertilized at the time of oviposition. Consequently some, such as muscids and most oestroids, lay eggs that require an additional period of embryonic development before hatching (i.e., oviposit). In contrast, the nose bot flies and flesh flies retain fertilized eggs in an expanded segment of the common oviduct named the uterine pouch, or uterus. They deposit packets of active larvae within sticky membranous “shells” that hatch immediately (i.e., larviposit).

In most cases, eggs or larvae are laid in contact with the larval nutrient substrate. However, in a few cases (e.g.,cuterebrines and nest maggots), the first-instar larvae must locate and invade their nutrient resources.

After hatching, the larvae begin to feed by scratching the nutrient substrate with their hooklike mandibles. This loosens fragments of substrate, which are bathed in the maggot's powerful salivary enzymes and are sucked up as a nutrient soup. Only the cephalopharyngeal skeleton and spiracular plates are fixed in size at the time of molting relative to the total length and girth of the feeding larva. The ratio between skeleton and body sizes indicates the larval age within each instar. The body integument remains pliable, facilitating considerable growth and expansion during each stadium. While inside a living host, larvae are relatively protected from exploitation by predators or parasites, but they must cope with their host's immune defenses. This is an advantage over free-living related species or siblings developing in carrion where competition and exploitation are major risks.

The next period in development is that of the wandering, mature, postfeeding larva. Larvae may continue to feed on available nutrients even after reaching and passing a critical size necessary for wandering and subsequent pupation. This critical size for some species is reached early in the third stadium so that, if the nutrient source becomes exhausted, the result can be very small adult flies. The duration of wandering is an innate character for each species and is influenced by environmental factors such as temperature, moisture, soil condition, and light. It may be shortened considerably if a suitable pupation site is obtained early on. Once a site is found, the larva becomes immobile and contracts in size. The resulting puparium is formed by the synchronized, concerted action of muscular and cuticular structures resulting in a rigid, hardened protective shell formed from the cuticle of the last larval instar. All these puparium-formation processes are driven by hormonal control. As soon as the puparium has hardened, the metamorphic histolysis begins, giving origin to the cryptocephalic pupa. Later on, eversion of the imaginal head coincides with transformation of the pupa into the pharate (hidden) adult. A series of changes such as eye and integument pigmentation characterize the remaining adult intrapuparial development (Cepeda-Palacios and Scholl, 2000).

Adult flies typically emerge from the puparium in the morning hours. The teneral adult exits the puparium through a predetermined portal formed by an anterior opening called the **operculum** or by detachment of the anterior end of the puparium. After a short period needed to harden their wings, adults are ready to take to flight in search of a mate and, in the case of females, a suitable adult and larval nutrient source. Mating usually occurs after adult flies have matured for a period of a few days to about 1 week. Many adult females (e.g., *Cochliomyia hominivorax* and the nonfeeding bot flies) do not require protein for the first ovarian cycle and are referred to as being **autogenous**, whereas other species such as *C. macellaria* do require protein to develop eggs and are referred to as **anautogenous**. Adult females in this group require additional nutrients and a period of ovarian development of several more days.

Flies that lay eggs that require time to complete their embryonic development outside of the female are **oviparous**. In bot flies, gonotrophic development is often initiated during the larval stage and continues through pupation, so that eggs can be deposited with less delay. The more primitive cuterebrine bot flies require about 5 days between eclosion and oviposition. In contrast, cattle grub and stomach bot flies have fully developed eggs at eclosion, and their females are ready to oviposit as soon as mating is completed. As already noted, nose bot flies and flesh flies retain eggs in utero so that their eggs hatch immediately after fertilization. This delayed oviposition is correctly termed **ovoviviparity** but usually is referred to as **larviposition**.

Unlike other oestroids, the bot flies have only one egg-producing (gonotrophic) cycle per ovariole and later the whole ovary is atrophied (Cepeda-Palacios and Scholl, 1999). Although subsequent cycles do not occur, among the cattle grubs each ovarian follicle may develop two eggs simultaneously, thus doubling the egg production of the one gonotrophic cycle (Scholl and Weintraub, 1988). This limitation to a single cycle among bot flies reflects their shorter, nonfeeding adult life span. In contrast, muscoid and other oestroid flies (calliphorids and sarcophagids) can hold their unfertilized eggs for several weeks until a suitable larval breeding medium is found. Following oviposition or larviposition, they can initiate additional gonotrophic cycles.

The primary activities of male muscoid and oestroid flies are to locate and copulate with receptive females. Males are capable of multiple matings during their life but play no further role in reproduction or dispersal of progeny. Typically, females mate only once, storing spermatozoa in the spermathecae. Dispersal of progeny is accomplished by the female fly and by the mobile host. This is another peculiar aspect of obligatory myiasis because the female fly usually attacks more than one host, thus distributing her progeny. The larvae, when mature, drop from their ambulatory hosts and thus can be scattered over an extensive area.

A number of vernacular terms are associated with myiasis-causing flies. Some refer to the adult flies, or more often their larvae, and others refer to the pathology that they cause. The term **maggot** is of Scandinavian origin and used for the larval stage of muscoid, calliphorid, and sarcophagid flies. The term **bot** is applied only to oestrid larvae and

is derived either from a Gaelic word *botus* meaning belly worm or from the Italian word *botta*, which refers to the cutaneous ulcer or furuncle caused by these flies. This ulcer, or open cyst, is termed a **warble,** which is derived from the Scandinavian word *varbulde*, meaning "boil." This term is misused at times to refer to the larva instead of its furuncle. In the southeastern United States, warbles have been called **wolves** since colonial times, in reference to the boil-like cuterebrine infections in rodents and rabbits. **Grub** is a term applied to cattle bots (as well as to scarabid beetle larvae). This term probably is derived from the Indo-European word *ghrebl* meaning "to scratch, scrape, or bore into." The common name of **screwworm** probably describes the threaded appearance of the maggot stage of these blow flies and the belief that they twist and bore their way into host flesh.

When flies oviposit on a host, or on carrion, the action is referred to as **striking** or **fly strike**. This is an English term originating from the Latin *stringere* meaning "to touch lightly or brush against." Its use probably was more in reference to the ovipositing of hovering nose bot flies or stomach bot flies.

A host or other substrates on which fly eggs have been laid is said to be **fly blown**. The use of "blow" in this context comes from the Old English term *blawan* and probably refers to the production of gas from bloated carrion containing maggots. This is also the source of the name **blow fly** for those flies that are most conspicuous at carrion, the Calliphoridae.

ECOLOGY AND BEHAVIOR

Studies on ecology of myiasis-producing species are scarce, particularly at the population dynamics level. There exists, however, information for some calliphorid species and environmental relationships; most of these data are focused on strike prevention/monitoring and for forensic purposes. Even more critical is the case of bot flies with reduced and scattered adult distribution and the lack of diverse trapping and attraction methods.

The environmental constraints on myiasis-causing flies during the free-living periods of their life cycle are the same as for other Diptera, primarily feed, moisture, and temperature. These constraints are of little importance during the parasitic period of their life cycle because they are provided by the host at levels well within the tolerance limits of eggs and larvae. Some myiasis-causing species lay their eggs free from the host and thus are adapted to a wider range of environmental conditions (e.g., cuterebrines and nest maggots). Muscoid and oestroid larvae are adapted in form and behavior to life in a moist, organic substrate ranging from wet feces to living tissue. Constraints on larval development, unlike on adults, stem more from host resistance and interspecific or intraspecific competition.

Facultative myiasis-causing flies generally develop in carrion and feces as massed aggregations of maggots. In such situations, the increased temperature that allows for rapid larval development mostly comes from metabolic heat produced by the clustered maggots themselves. For these species, a living, protein-rich, moist, warm host substrate merely substitutes for the nonliving substrate. Facultative myiasis often results in the death of the host either by direct effects of maggots or by the indirect effect of stress, which predisposes the host to predation or disease. Sterile, laboratory-reared maggots, however, can have therapeutic benefits and have been used in treating deep wounds and sores because they feed only on dead tissue.

For flies that cause facultative myiasis, finding a receptive mate generally is not difficult. The synchronous emergence of sibling adults developed from the same mass of maggots puts newly emerged males and females in close proximity. These species also find their oviposition sites by responding to the same stimuli that attract them to any nonliving resource (e.g., fetid or putrid odors, purulent discharges and/or accumulations of animal excretions). They show very little discrimination regarding host species. Host size and habitat are the principal limiting factors. Even though their resources have a patchy distribution, these flies can respond quickly to chemical stimuli at very low concentrations in order to locate widely scattered resources.

All fly species causing obligate myiasis in live vertebrates can be considered as carnivorous in the food chain. The greater longevity of muscoid flies, blow flies, and flesh flies also requires that they imbibe fluids to maintain an internal water balance and that they obtain an energy source beyond that acquired during larval development. The energy source is often honeydew and plant juices, especially nectar. This is why adults of these flies also occur on flowering plants and are important pollinators for many plants, including certain crops. Most female flies also require a protein supplement to complete oogenesis, which is supplied by a wide array of feeding sources (e.g., blood secretions, excretions, wounds, and carrion).

The obligatory myiasis-causing screwworms (e.g., primary New World screwworm, Old World screwworm, and wound flesh fly) are closely related to the facultative myiasis-causing species. The obligatory **primary species** invade healthy tissue and enlarge wounds and are often found deep in these wounds. In contrast, the **secondary species** do not feed on healthy tissue, almost always invade necrotic wounds, and feed on dead tissue. These secondary invaders are usually found on the surface or outer part of the wound. Because their progeny are often widely scattered, the adults aggregate at certain flowering shrubs where they mate and feed. Their egg development, mating, and indiscriminate oviposition on any suitable host are similar to that of facultative myiasis-causing flies normally occurring in carrion.

For the obligatory myiasis-causing oestrid flies, mating, host seeking, and host–parasite interactions are more complicated. Because adults usually emerge from scattered sites, contact between males and females requires **aggregation behavior**. Species in each of the oestrid subfamilies aggregate at specific topographic sites where mating takes place. This probably is the case for all oestrids. Some bot flies also mate near potential hosts. Where hosts are plentiful, aggregation behavior can cause crowding of adult bot flies, thus exposing the flies to predation and to less-than-ideal environmental conditions for survival. At these sites bot flies exhibit a male spacing behavior that counteracts crowding, interferes with intraspecific pairing, minimizes predation, and ensures that all available aggregation sites are occupied.

Adult oestrid flies do not feed and have relatively short life spans, living on water and fat reserves accumulated as larvae. For example, *Oestrus ovis* adult females at eclosion have a complement of only 77 mg water and 12 mg fat. These reserves are sufficient to support mate seeking, egg incubation, host location, and larviposition activity for 12–15 days (Cepeda-Palacios et al., 2011).

Most bot flies display a high degree of **host specificity**, with each species parasitizing only one, or rarely a few, host species. *Dermatobia hominis* is an important exception, with a very large host range. Host species other than the native hosts are either intolerant or refractory to parasitism by a given species of bot fly. Although other host species are susceptible to bot fly invasion, this does not mean that they are suitable hosts or that mature larvae and adults are produced. Susceptible, but unsuitable, hosts tend to be species that are unrelated phylogenetically and ecologically to the native host species. Thus, dogs and cats are susceptible, unsuitable hosts for cuterebrine bot infections, whereas a given mouse species can be completely refractory to infection by the bot fly of a related mouse species. This high degree of host specificity reflects a long coevolutionary relationship between bot flies and their respective hosts, which helps to explain why no larvae in any of the four subfamilies of Oestridae have ever been reared in vitro from newly hatched first instars to pupae.

Each bot fly species typically develops only at a specific site in its native hosts (e.g., nose bots, stomach bots, and foot bots). Even among the cutaneous bot flies (excluding *Dermatobia*), the location of warbles on the host occurs at specific anatomical sites. In non-native and atypical hosts, however, this site occurrence can be erratic, often with grave consequences for the host and its parasites.

Host–parasite interaction involving bot flies is a delicate balance of tolerance by the host and limited defense response by the parasite. It also involves immunosuppression induced by some larvae. In bot fly infestations, excessive parasite burden usually is avoided by limiting the number of larvae to which a host is exposed. Bot fly females rarely dump their total reproductive complement on a single host. If this occurs, the host is likely to succumb to predators or other parasites due to the stress imposed by the developing bot larvae.

The number of bot fly larvae infesting an individual host has been shown to vary considerably. There are instances in which female oestrid flies have been shown to prevent host overloading by spreading groups of larvae among hosts. This is far less than the thousands of maggots that can make up the maggot mass of other myiasis-causing flies. Although much larger than other myiasis-causing flies, oestrid larvae appear to cause much less stress to their host. Although the reasons are unclear, this has the advantage of reducing the likelihood that the host will die during their development. Also, intrahost regulatory mechanisms from older larvae may prevent new larval infections.

First-instar bot larvae move from their point of host invasion to the site of development in their host. Studies have shown that this movement within host tissues is typically along predetermined pathways within connective tissue or along the fibrous membranes covering and separating muscles (i.e., fascial planes). This keeps the larvae from prolonged contact with the host's hemopoietic defenses and helps to explain the seeming lack of immune resistance. Immune resistance by hosts to bot fly-caused myiasis apparently lasts only a short time after an infestation.

The wall of the warble is formed by the host's response to foreign body invasion. The warble wall confines the developing larvae in a multilayered pocket of fibrous connective tissue. The wall is characterized by the presence of giant cells typical of a chronic inflammatory response. In addition, the developing larva secretes a bacteriostatic substance that prevents secondary microbial infection of the warble. In a native host, once the mature larva has dropped free, the empty warble collapses and heals rapidly.

Some hosts show exaggerated, usually futile, behavioral responses to certain bot flies in the act of ovipositing. For example, cattle panic when under attack by adults of the northern cattle grub (*Hypoderma bovis*). They respond by headlong flight with tail erect, called **flagging**, apparently attempting to outdistance the hovering fly. This response is more commonly called **gadding**, a term derived from the Old English *gad,* meaning "a sharply pointed stick used to goad or prod livestock." Reindeer and caribou show a similar response to attacks by *H. tarandi*. In contrast, cattle display far less concern for the attacks by adults of the common cattle grub (*H. lineatum*), which often oviposit while animals are ruminating in a recumbent position. Gadding behavior also is shown by horses when under attack by the horse throat bot fly (*Gasterophilus nasalis*), whereas horses usually pay little heed to oviposition by the common horse bot fly (*G. intestinalis*).

Another behavioral avoidance response is shown by deer, sheep, and goats when under attack by nose bot flies. A threatened host will lower its head and press its muzzle into a clump of grass or against the ground or herd mates to deter strikes by the hovering female flies (e.g., *Oestrus ovis*, *Cephenemyia* spp.).

Host−parasite interaction involving species that cause facultative myiasis show little adaptation to neutralizing host defenses. When present in large numbers, however, rapidly developing larvae overcome the nonspecific host response to tissue invasion characterized by self-grooming and inflammation. Unlike oestrid larvae, most other myiasis-causing larvae can complete their development in the dead host.

Myths

Several misconceptions have developed concerning bot flies and myiasis. The horror of harboring or viewing maggots by humans can cause extreme stress in some individuals. The reaction can be severe enough to result in mental disorders and self-mutilation (Clarke, 2013). There also is the notion that adults of deer nose bots (*Cephenemyia* spp.) are capable of flying at speeds in excess of the speed of sound. The belief had its origin from a single questionable field observation of the deer nose bot fly coursing by a man standing on a hilltop. This tale gained some ill-founded credibility from entomologists and was published as fact for some time. The reasoning was that the bot fly needed to fly fast in order to overtake a swift running host. This reasoning ignored the fact that deer are incapable of running speeds in excess of 80 km/h (=50 mph) and that the fly usually oviposits while the host is standing still. Although there have been no definitive studies of this subject, field observers conclude that *Cephenemyia* spp. probably cannot exceed a flight speed of 48 km/h (=30 mph).

Another myth is that the larva of the rodent bot fly *Cuterebra emasculator* emasculates its squirrel and chipmunk hosts by consuming the host's testes as it develops. This tale arose because the specific site for larval development by this species is the inguinal area. Studies have shown, however, that the enlarging bot larva and warble in the scrotal sack of male hosts merely prevent the seasonal descent of gonads into the scrotum from the host's body cavity and do not cause sterility.

Because of the conspicuous and sometimes self-destructive gadding response of cattle and horses under certain bot fly attacks, the belief arose that the bot flies involved were stinging their hosts. The extensible terminal segments of the abdomen of *Gasterophilus* females suggest a formidable sting. However, these flies contact individual host's hairs only during oviposition. They do not injure or even touch the skin surface. Hosts are frightened by the buzzing sound made by these flies as they hover, and when one individual in a herd bolts, the herd instinct prompts the same reaction from others.

There is a common belief in the southeastern United States that the flesh of squirrels and rabbits infected with "wolves" (*Cuterebra* spp.) is unfit to eat. Because of this, the entire squirrel carcass is often discarded. This, too, is a myth. In fact, skin-bot larvae are eaten as a delicacy in sub-Arctic human cultures that traditionally herd reindeer in which bots commonly occur. Although the host flesh surrounding the warble may be discolored, there is no health hazard in eating either it or the bot larva. Squirrels and rabbits have long been a staple in the fall and winter diet of rural people in the United States. In some states the legal hunting season even has been postponed to begin after the bot season has ended to avoid the wasteful discarding of bot-infested game.

FLIES INVOLVED IN MYIASIS

Flies that typically develop in dung or decaying plant matter generally are involved in gastrointestinal myiasis. A few muscids also are adapted for blood-feeding on nestling birds. The carrion-breeding flies and their more fastidious relatives, screwworms and flesh flies, are adapted for both facultative and obligatory myiasis. The bot flies (Oestridae) are all obligatory myiasis producers.

The families and groups of flies that follow include species that cause myiasis in humans and other animals. A more extensive listing of families, species, and the types of myiasis that they cause, is presented in Table 19.1. For reviews of this subject, see James (1947), Zumpt (1965), Leclercq (1969), Hall and Wall (1995), Wall and Shearer (1997), Guimarães and Papavero (1999), Colwell et al. (2006), and Hall et al. (2016).

Psychodidae (Moth Flies, Drain Flies)

This family is very large and includes a few species involved in facultative myiasis in humans. Adults are minute blackish or greyish flies covered with hairs, even on the wings. Their wings possess longitudinal venation only. They show cosmopolitan distribution, being commonly located in bathrooms and toilets. Larvae feed on decaying organic matter and hair detritus in open drains or sewers. *Psychoda albipennis* has been reported to cause urogenital myiasis, and *Clogmia (Telmatoscopus) albipunctata* causes urogenital and intestinal myiasis (El-Badry et al., 2014).

Stratiomyidae (Soldier Flies)

The only species in this family that reportedly causes myiasis is the black soldier fly (*Hermetia illucens)*. Although originally a New World species, it is widely distributed in warmer temperate and tropical areas of the

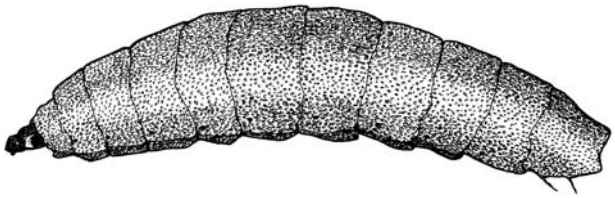


FIGURE 19.3 Black soldier fly, *Hermetia illucens*, larva (Stratiomyidae). *Original by E. Paul Catts.*

world. The larvae (Fig. 19.3) develop in decaying fruits, vegetables, and other plant material, decomposing animal carcasses, and excrement. Only a few human cases of intestinal myiasis involving *H. illucens* have been documented. At least one case resulted in rather severe enteric disturbances, apparently due to the large size and vigorous activity of the larvae (James, 1947).

Phoridae (Humpback Flies)

Adults are also known as "coffin flies" or "scuttle flies." They are blackish or brownish-yellowish flies and small (0.5−6 mm in length), with a humped structure on the thorax and flattened head. Three myiasis-causing species are included in this family, from which *Megaselia scalaris* and *Megaselia spiracularis* are so far best described. They usually live in temperate or cold areas associated with stored food, fecal matter, and fermenting organic matter. Larvae cause urogenital and wound secondary myiases in humans and other farm and pet hosts, including reptiles (Vanin et al., 2013). *Megaselia scalaris* is the species commonly involved in phorid myiasis cases in North America.

Syrphidae (Flower Flies, Hover Flies, Rat-Tailed Maggots)

This is a large family (180 genera, 6,000 species) that includes only a few taxa that cause gastrointestinal myiasis. Adults also are called drone flies because of their bee-like appearance and resemblance to honeybee drones. The terms *flower flies* and *hover flies* refer to their common habit of visiting flowers for nectar and pollen and their ability to hover motionless in flight. The larvae of *Eristalis* (Fig. 19.4) and other aquatic genera are called rat-tailed maggots, referring to the long, telescopic, three-segmented respiratory tube at their posterior end via which they breathe at the water surface.

The syrphid species most frequently involved in myiasis is *Eristalis tenax*. Its larvae develop in sewage, liquefied excrement, decaying animal carcasses, and other decomposing plant and animal material of a liquid consistency. Several human cases of gastrointestinal myiasis have been reported, with live larvae being passed in stools. Two other

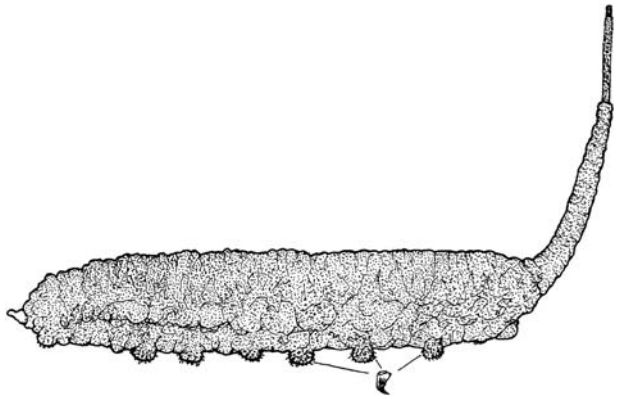


FIGURE 19.4 Rat-tailed maggot, *Eristalis tenax* (Syrphidae). *Original by E. Paul Catts.*

Eristalis spp. have been identified in human myiasis: *E. arbustorum* in Europe and *E. dimidiata* in the United States (James, 1947). Leclercq (1969) described several human cases involving rat-tailed maggots, including a man in Germany who passed more than 40 *Eristalis* larvae in 1 day and another 10−30 larvae on each of the six following days. *Eristalis* larvae also have been the cause of vaginal myiasis in cattle.

Piophilidae (Skipper Flies)

This is a small family of about 70 species in 35 genera worldwide. Females of the common cheese skipper (*Piophila casei*), oviposit on putrid, dried, cured or smoked meats and cheeses, typically depositing 400−500 eggs per female. Adult cheese skippers are small (3−5 mm), slender, glossy black flies with yellow on the lower face and part of the legs. Larvae are slender, cylindrical, white, and truncated caudally with three pairs of short caudal protuberances, the ventral pair being the largest (Fig. 19.5). Larvae require about 5 days to develop under warm conditions. In temperate regions the mature larvae overwinter. The name *skipper* originates from the ability of the larva to flex head-to-tail in a circle and, following total-body muscular contraction and release, the larva propels itself off the substrate for a considerable distance (up to 24 cm). This behavior is used as a means of escape when disturbed or when dispersing to suitable pupation sites. The pupal

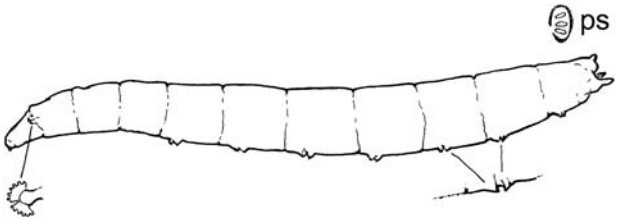


FIGURE 19.5 Cheese skipper, *Piophila* sp., larva (Piophilidae). *Original by E. Paul Catts.*

stage lasts about 5 days. The life cycle, egg-to-egg, can be completed in as little as 2 weeks.

Piophila casei is a widely distributed species that commonly infests cured meats, cheeses, and dried fish. It probably is the species most commonly involved in gastrointestinal myiasis of humans. The tendency of people to leave cured meats and cheeses unrefrigerated makes these foods available to gravid females for oviposition. These flies can survive the rigors of alimentary-tract passage and can even pupate and emerge as adults before leaving the host. The related *P. vulgaris* and *Stearibia* spp. are common inhabitants of dried carrion.

Neottiophilidae (Nest Skipper Flies)

This small Palearctic family includes two genera: *Neottiophilum* and *Actinoptera*. The larvae cause cutaneous myiasis by sucking the blood of nesting birds. Larvae attack with their mandibles and can penetrate deeply enough into their host to cause septicemia and death. Larvae pupate in the host's nest in the fall and emerge as adults in the spring. The adult is a yellow-brown fly, about 7−8 mm in length, with wings "pictured" with a few brown spots. Hosts typically are passeriform birds. Local strains of *N. praeustum* show narrow host specificity to different avian species.

Drosophilidae (Pomace Flies, Vinegar Flies, Fruit Flies, and Wine Flies)

This is a large family (3,000 spp. in 60 genera) of small red-eyed flies (1.6 mm) whose adults favor the odors of overripe or fermenting plant products, usually fruits. Larvae feed on microorganisms found in such substrates. The genus *Drosophila* is the largest and includes more than half of the species in this family. Larvae have posterior spiracles on paired caudal protuberances (Fig. 19.6), which also are evident in the puparia. The best known species is the highly domesticated kitchen gnat *D. melanogaster*. An additional seven species also are locally common domestic pests (e.g., *D. busckii*, *D. funebris*, *D. hydei*, *D. immigrans*, *D. repleta*, *D. simulans*, and *D. virilis*). The life cycle for these species is typically 12−14 days, making these small flies useful as biological models in studies of genetics, physiology, cytology, and population dynamics. Because of their attraction to fruits and vegetables, these species can cause accidental gastrointestinal myiasis.

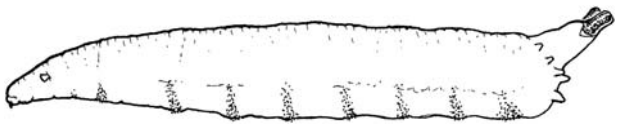


FIGURE 19.6 Pomace fly or fruit fly, *Drosophila* sp., larva (Drosophilidae). *Original by E. Paul Catts.*

Chloropidae (Grass Flies and Australian Frog Flies)

The genus *Batrachomyia* includes 10 species whose larvae occur individually in swollen, subcutaneous pockets on the body (not the legs) of Australian frogs. The adult flies are yellow-brown in color and possess hairy eyes. Adults feed on plant juices. Their eggs require high humidity and are laid near, but not on, the host. After moving to a frog host, the larvae attack and appear to feed on blood, reaching a length of 10 mm when fully mature. The mature larvae are peculiar in appearance, having paired anterior and posterior "tentacles" with each bearing a spiracle (Fig. 19.7). Seasonal prevalence in frog populations can be as high as 25% with a parasite load of 1−4 maggots per host. Death results in about 10% of the frogs at the time of larval drop.

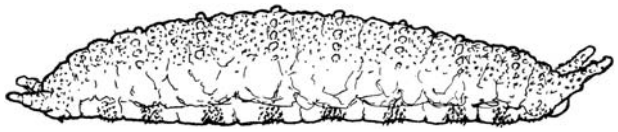


FIGURE 19.7 Australian frog fly, *Bratrachomyia* sp., larva (Chloropidae). *Original by E. Paul Catts.*

Anthomyiidae (Root Maggots)

The Anthomyiidae and the following two closely related families, Fanniidae and Muscidae, are quite similar in their development, behavior, and occasional association with cases of gastrointestinal myiasis. Both the immatures and adults are similar in appearance to muscids. As the name "root maggot" implies, most anthomyiid larvae feed on plants. The anthomyiid genus *Hylemya*, however, is very large with 180 species occurring in a diversity of habitats in the Nearctic region. The gastrointestinal myiasis that they can cause generally results from the ingestion of larvae infesting vegetables or fruits.

Fanniidae (Faniid Flies)

Four species of *Fannia* have been reported to cause myiasis: **little housefly** (*F. canicularis*), **latrine fly** (*F. scalaris*), *F. incisurata,* and *F. manicata*. Adults of these flies look like small, slender house flies. They are drab gray in color and lack black stripes on the thorax. The larvae commonly occur in feces, in rotting fruits or bulbs, and in bird nests. Larvae occasionally occur in older, somewhat dried carrion. The larvae have a characteristic, fringed appearance (Fig. 19.8) that easily distinguishes them from other muscoid maggots. The fringes apparently allow these maggots to float in a near-liquid medium. The life cycle requires about 1 month. Although cases of enteric

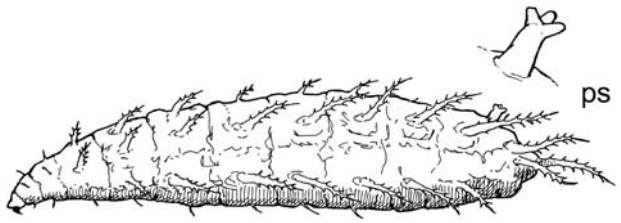


FIGURE 19.8 *Fannia* sp., larva (Fanniidae); *ps*, posterior spiracle. *Original by E. Paul Catts.*

and urethral myiasis in humans have been documented (James, 1947; Leclercq, 1969), the involvement of *Fannia* spp. in myiasis is rare, except as important mechanical vectors of *Dermatobia hominis* in its southern range.

Muscidae (Dung Flies)

The large family Muscidae includes at least seven genera in which species cause myiasis (Table 19.1). Muscid larvae (Fig. 19.9) develop in a wide diversity of decaying organic matter, usually of plant or fecal origin. Occasionally they develop in old or buried carrion that is unsuitable for blow-fly exploitation. All stages of myiasis-causing muscids are typically house fly-like in appearance. Exceptions are *Neomyia* and *Hydrotaea* spp., in which some adults have metallic coloration similar to blow flies, and the nest flies, which are larger and yellow to yellow-brown in color. Gastrointestinal myiasis caused by muscids usually results from oviposition on wet foods. It also may result from retro-infection through the host's anus following fly attraction to foul odors or soiling by feces. Urogenital myiasis may occur in association with purulent discharges, urine-soaked clothing, and secondary microbial infections.

The genus *Musca* includes about 60 species, which are confined mostly to the Old World. The two most important species that have invaded the temperate regions of the New World are the **house fly** or **typhoid fly** (*Musca domestica*) (Fig. 19.9) and the **face fly** (*Musca autumnalis*). In the Old World tropics, the prevalent species is the **bazaar fly** (*Musca sorbens*). Both the house fly and bazaar fly oviposit

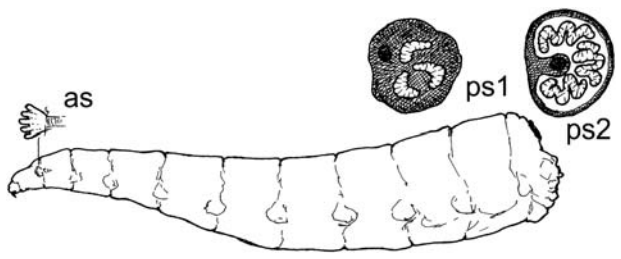


FIGURE 19.9 Typical muscid larva (Muscidae), third instar. *as*, anterior spiracle; *ps1*, posterior spiracular plate, house fly (*Musca domestica*); *ps2*, posterior spiracular plate, false stable fly (*Muscina stabulans*). *Original by E. Paul Catts.*

in a wide range of wet, decaying organic matter. Greenberg (1971) suggests that the house fly and bazaar fly were originally adapted, as larvae, to develop in wet ungulate feces. They do show preference for accumulations of animal excrement, especially from horse, cow, human, pig, and poultry. Their egg complement ranges from 1,000 to 3,000 per female and is laid over the adult life span in clusters of 120–150 eggs. The quality of larval and adult diets largely determines the number of eggs produced by any single female. Maggots develop rapidly in 3–5 days under wet, warm conditions but are intolerant of desiccation. In the tropics, their life cycle can be as brief as 10–12 days, whereas 3 weeks is more typical in other regions. All stages can overwinter, but in colder areas there is a dramatic winter die-off. Larvae of *Musca* spp. that invade wounds feed primarily on necrotic tissues. *Musca* species with rasping-sucking mouthparts that feed on blood or other secretions (e.g., *M. autumnalis*) are not known to cause myiasis.

The genus *Muscina* includes eight species, three of which are implicated in accidental myiasis (*M. stabulans*, *M. assimilis,* and *M. pabulorum*). The **false stable fly** (*M. stabulans*) (Fig. 19.9) is the most important and is involved primarily in gastrointestinal myiasis. Occasionally their maggots occur in fetid sores or wounds. Adults of this species look much like house flies, except that they are usually larger and more robust. They are attracted to, and feed on, plant juices, rotting fruits, and insect-excreted honeydew. Females oviposit by scattering their 140–200 eggs on the surface of overripe, decaying fruit. They also oviposit on accumulations of dead insects or feces, usually from human sources, and on buried carrion. Early-instar larvae are saprophagous but become predaceous as they mature. Third instars prey on smaller maggots. This transition from a saprophagous to a predaceous habit has two advantages over species whose larvae remain saprophagous: first, the maturing larvae can store protein resources obtained from their prey to be used by the adult in reproduction, and, second, this habit enables this species to exploit a wider range of protein-poor resources as a larval substrate. Larval development varies from 2 to 3 weeks. They usually overwinter as pupae.

Hydrotaea spp. are metallic-colored muscids usually found in association with feces and older carrion. Like *M. stabulans*, their dark, cream-colored larvae (up to 15 mm long) become predaceous on other maggots. In Australia, *H. rostrata* has been implicated as a tertiary invader in myiasis of sheep, but it appears to feed on necrotic tissues only.

Tropical Nest Flies

These muscid flies include the genera *Passeromyia*, *Mydaea*, and *Philornis*. Species in these three genera

usually parasitize the young of cavity-nesting birds. Although few details are known about these flies, their biology appears to be similar and are discussed collectively here. Species in the first two genera are widely distributed in the Ethiopian, Oriental, and Australian Regions. The latter genus is found in the New World tropics. Currently *Philornis* is the valid generic name for those flies formerly listed in the genus *Neomusca*.

Adults of these tropical nest flies feed on plant juices and wet feces. Gravid females oviposit in bird-nest debris near nesting birds. After the eggs hatch, the maggots crawl to the nestlings, scratch the skin with their mouth hooks, and imbibe blood. As they feed they continue to scratch and penetrate host tissues. In heavy infestations they can penetrate the host body cavity with fatal results for the bird. The larvae develop rapidly, in less than 1 week, before leaving the host to pupate in the nest debris. Postfeeding larvae of *Philornis* spp. exude a frothy, sticky, salivary spittle that coats the puparium and to which camouflaging debris adheres. Despite this defensive measure, pupae are subject to attack by parasitic wasps.

Calliphoridae (Blow Flies, Carrion Flies, Floor Maggots, Nest Maggots, and Screwworms)

The most generalized of the six families of Oestroidea is the Calliphoridae with more than 1,000 species. Among the members of this large family, there is a transition from the facultative myiasis habit by a large number of normally saprophagous species to obligatory myiasis by a relatively small number of species (ca. 100). The larvae typically feed on wet, living or dead flesh. Desiccation is detrimental to both egg and larval survival. The following discussion treats the more important, widely distributed and common genera of myiasis-causing calliphorids.

Carrion-Associated Blow Flies

These are the showy metallic **blue-bottle flies**, **green-bottle flies**, and **black blow flies** (Fig. 19.10). They include members of the genera *Calliphora*, *Chrysomya*, *Cynomya*, *Eucalliphora*, *Lucilia* (=*Phaenicia*), *Paralucilia*, *Phormia*, and *Protophormia*, which are commonly associated with dead animal tissues, or carrion. The Old World genus *Chrysomya* also includes one species (*C. bezziana*) that causes obligatory myiasis. The importance of this group as primary agents of cutaneous myiasis and impact on animal production cannot be overstated, especially *Lucilia sericata*, *L. cuprina*, and *Phormia regina*. These three species are discussed in the section on Veterinary Importance. The duration of their life cycle in carrion differs among species but, in general, roughly one-third of the time is spent as eggs and larvae, one-third as pupae, and one-third as adults from emergence to mating and oviposition. Life cycles typically take 3−4 weeks but are prolonged by cold temperatures. These flies are attracted to fetid, purulent open sores and chronic nasopharyngeal or urogenital infections. Heavy larval infestations often result in death of the host, after which the maggots continue to feed on the resulting carrion.

The body form of most of these calliphorid larvae is typical of members of this family (Fig. 19.11) and is distinguished by the arrangement and shapes of their spiracles and cephalopharyngeal skeleton. The larvae of a few genera, however, are atypical in possessing numerous girdling bands of fleshy processes (e.g., *Chrysomya* spp.) similar to those of the genus *Fannia*. Like *Muscina stabulans*, the maggots of the **hairy maggot blow fly** (*Chrysomya rufifacies*) can switch from a myiasis to a predatory role. Postfeeding maggots drop free from the host or leave the carcass of a dead host to wander in search of pupation sites.

Toad Blow Flies (Bufolucilia spp.)

These flies include several species that cause primary obligatory myiasis in amphibians. Although they are common in the Old World, *B. silvarum* is the only North American species. The female lays her eggs on the back and flanks of a living toad, a risky activity for any fly. The resultant larvae invade the host's nasal passages or eye sockets, where they develop rather quickly, in less than 1 week. The host dies at about the time of larval maturation, after which some larvae may remain to feed on the dead host. Maggots thus also occur on dead amphibians. The metallic green adults are attracted to other carrion as well.

Screwworms (Cochliomyia, Chrysomya spp.)

The **New World screwworm** (=**American screwworm**) (*Cochliomyia hominivorax*) and the **Old World screwworm** (*Chrysomya bezziana*) cause obligatory myiasis and can be of major economic importance. Both are **primary screwworms** with similar biology. Females, when gravid, are attracted to fresh open wounds on any warm-blooded animal. The female quickly oviposits 100−200 eggs on the dry perimeter of the chosen site, with each female producing up to 1,000 eggs in her lifetime. Female flies commonly feed at the wound, thus obtaining protein for producing their next egg mass. After a brief incubation period of 10−20 h, the eggs hatch and the larvae begin feeding on the open wound. The maggots develop rapidly to maturity in 4−12 days and then drop to the ground to pupate. After about 1 week as pupae, adults emerge, mate at aggregation sites, and after several days seek a protein meal and new living host. The entire cycle (egg-to-egg) takes 2−3 weeks.

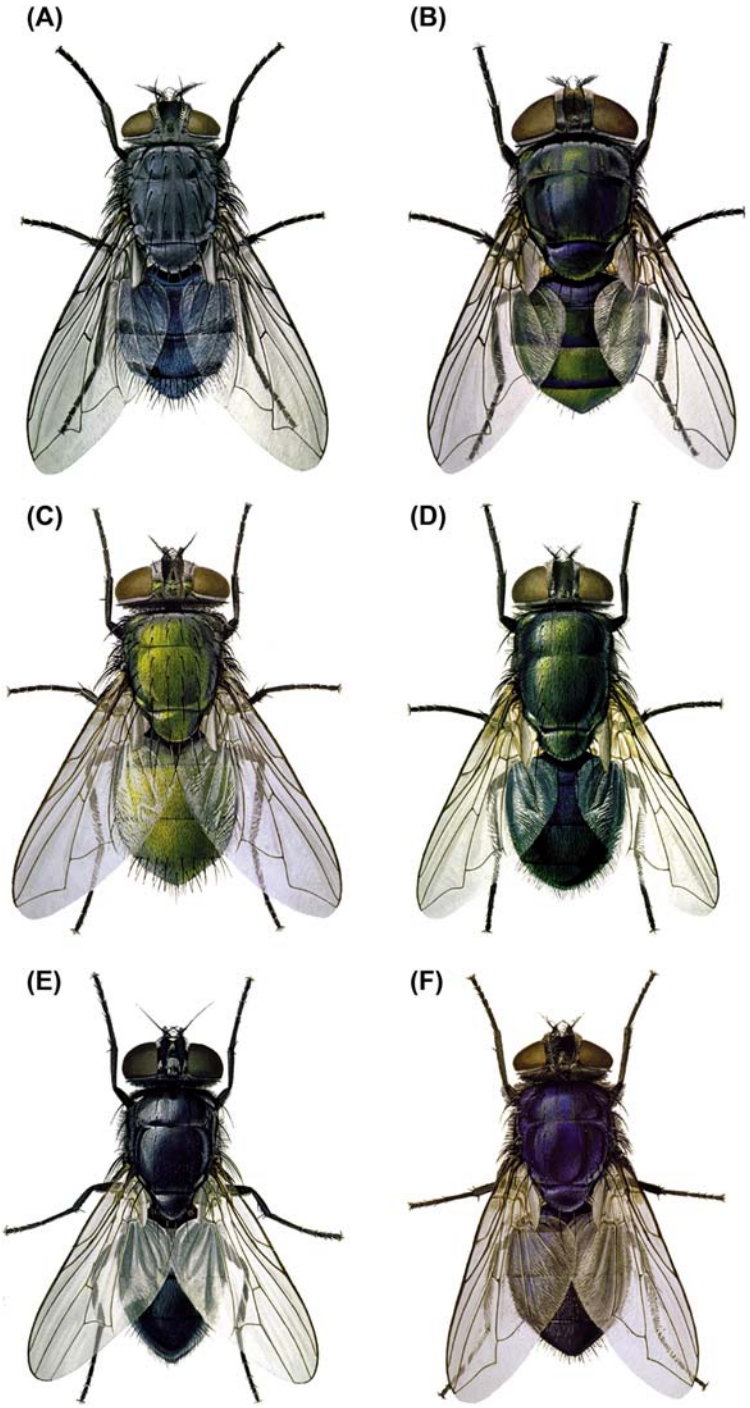


FIGURE 19.10 Blow flies that cause myiasis, adults (Calliphoridae). (A) *Calliphora vicina*. (B) *Chrysoma megacephala*. (C) *Lucilia* (=*Phaenicia*) *sericata*. (D) *Phormia regina*. (E) *Ophyra leucostoma*. (F) *Protophormia terranovae*. *From Greenberg (1971).*

Infested wounds range from mere thorn scratches or insect and tick bite marks to gaping lacerations. Infestations of the umbilicus of newborn calves by *C. hominivorax* and *C. bezziana* are common. Livestock husbandry operations such as castrating, dehorning, branding, and shearing also cause wounds subject to invasion. Untreated screwworm cases can be fatal, due to the invasion of host's vital organs, septicemia caused by feeding maggots, or secondary infections. The maggots literally eat the host alive. In areas of mild winters, adults can be active during warm spells. In temperate regions screwworm attacks are restricted to the warm seasons, but in the tropics they are more or less continuous.

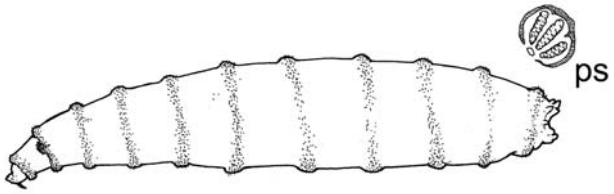


FIGURE 19.11 Typical blow-fly larva (Calliphoridae), third instar. *ps*, posterior spiracular plate. *Original by E. Paul Catts.*

Cochliomyia hominivorax (Fig. 19.12A) is a major livestock pest, especially to cattle in the Neotropics. Although formerly it ranged throughout tropical and temperate regions of the New World, innovative control measures using male sterilization and baiting of females have eliminated it from the Nearctic. *Chrysomya bezziana* is distributed widely in Southeast Asia, New Guinea, and Africa. It is the Afro-Asian counterpart of *C. hominivorax* and is primarily important as a parasite of livestock. Like the New World screwworm, this species is attracted to open wounds as well as to moist body openings. Generally, eggs are deposited in the late afternoon such that their development is completed by the next morning, thereby avoiding lethal exposure to direct sunlight and drying. The rapidly developing maggots consume flesh in localized sites, often penetrating *en masse* deep into the host tissues with fatal consequences.

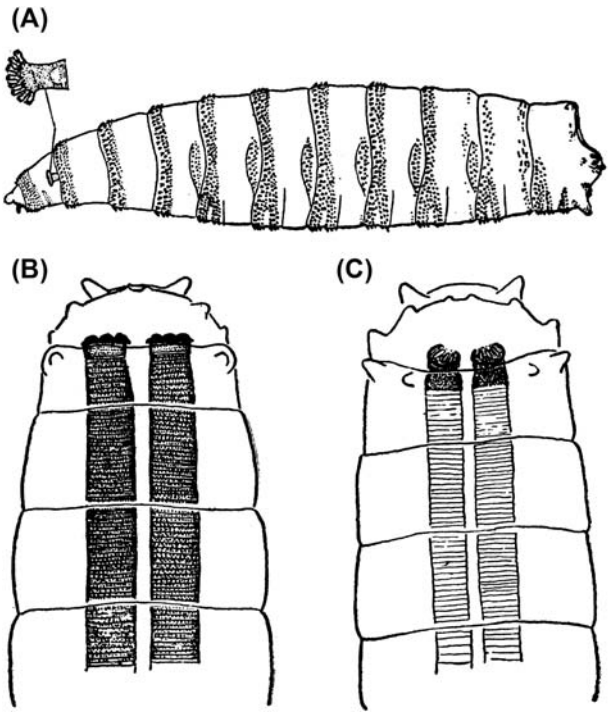


FIGURE 19.12 New World screwworm larvae (Calliphoridae), third instars. (A and B) Primary screwworm (*Cochliomyia hominivorax*), with enlargement of anterior spiracle and darkened portion of large tracheal trunks from posterior spiracles extending into three or four abdominal segments. (C) Secondary screwworm (*C. macellaria*), with darkened portion of the tracheal trunks restricted to part of one abdominal segment. *Modified from James (1947).*

Certain advantages are gained by these flies attacking only living hosts. There is less interspecies competition than among carrion-exploiting species, and the constant body heat of the host enhances maggot development. Additionally, the host tissues are less acidic and more digestible to the maggots than that of fresh carrion. Finally, and possibly more importantly, they avoid predation by ants and other predators since carcasses do not tend to persist for long in the tropics.

Species of **secondary screwworms** are close relatives of primary screwworms but cause facultative, rather than obligatory myiasis. They are termed "secondary" because they often infest wounds after invasion by primary myiasis-causing flies but have also been observed in the absence of primary screwworm invasion. The most important species are *Cochliomyia macellaria* (Fig. 19.12B) and *Chrysomya megacephala*. The latter species has become established in the Neotropics and appears to outcompete the former species. Another related blow fly is *Chrysomya rufifacies*. It is a widely distributed carrion-breeding fly that commonly shifts from being a scavenger in carrion to a predator of other maggots. However, on the island of Maui in Hawaii, maggots of *C. rufifacies* have been known to cause primary myiasis in the umbilicus of newborn calves, with prevalence of infestations as high as 30%.

Nest Blow Flies (Protocalliphora spp.)

These flies are members of the large genus *Protocalliphora* (90 species), Holarctic flies whose maggots (Fig. 19.13) are obligatory parasites of nestling birds. The intermittent, temporary bloodsucking habits of these maggots are similar to those of the tropical muscid nest-maggots. The maggots attach to the host by means of their mouth hooks to feed and then drop free to hide in nest debris while they digest their bloodmeals. Direct adverse effects on the nestlings are apparent only when maggot numbers are high. There is evidence, however, that their frequent bleeding of the host prolongs nestling development. The longer the time that birds stay in the nest, the more they are at risk of predation. Maggots thus can indirectly influence nestling success. Many species of *Protocalliphora* show high host specificity,

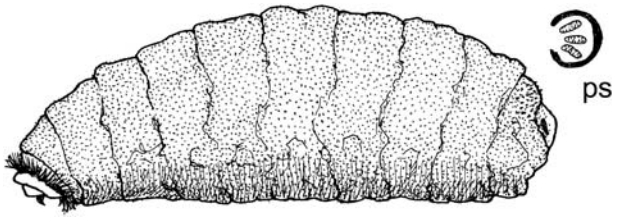


FIGURE 19.13 Nest blow fly, *Protocalliphora* sp., third-instar larva (Calliphoridae). *ps*, posterior spiracular plate. *Original by E. Paul Catts.*

indicating a long relationship with their respective hosts. These flies seem to favor cavity-nesting birds. Pupation and oviposition take place in the host nest. The manner in which the adult fly locates a specific host's nest is unknown.

Tumbu Fly (Cordylobia anthropophaga)

The tumbu fly is the most important of the six species of *Cordylobia*, a small group of African blow flies whose maggots develop in warble-like cysts in hosts ranging from rodents to dogs and humans. Rodents are assumed to be their primitive hosts, the flies having become secondarily adapted to other species. Except for their smaller size, these flies have biologies similar to the New World bot flies (Cuterebrinae), in many ways suggesting convergent evolution.

The adult tumbu fly is yellow-brown in color with two rather broad but variable dorsal thoracic stripes. Mature maggots are up to 15 mm in length and densely, but incompletely, covered with small, backward-directed, single-toothed spines. The posterior spiracles have a weakly sclerotized peritreme and three sinuous slits (Fig. 19.14). Adults feed on decaying fruits, carrion, and feces. Females are shade-loving and deposit eggs singly in dry sand or dirt contaminated with host urine or feces. Females also are attracted to dry, urine-soiled diapers or clothing. They lay up to 500 eggs over their lifespan of 2–3 weeks. The eggs hatch after several days, after which the first instars wait in the dry substrate for a host. Contact with a host stimulates the maggots to attach and penetrate the skin. Maggots develop in shallow warbles within or just beneath the skin in about 7–10 days and drop free to the ground to pupate in surface debris. Adults emerge after another several weeks. Although the tumbu fly invades a wide range of hosts, its successful development varies significantly among different host species. The domestic dog is an important reservoir, but maggots develop best in native rodents.

Congo Floor Maggot (Auchmeromyia senegalensis)

The Congo floor maggot (formerly *A. luteola*) is one of four or five species in this genus causing obligatory, temporary myiasis similar to that caused by *Protocalliphora* spp. They all occur in Africa south of the Sahara, and most are associated with the burrows of larger mammals such as warthogs. *Auchmeromyia senegalensis*, however, appears to prefer humans as hosts. Adults are yellow-brown in color with markings similar to the tumbu fly. They feed on rotting fruits and feces (e.g., human, monkey, pig). Females can lay up to 300 eggs. The maggots (Fig. 19.15) spend most of their time hidden in loose dirt and floor debris of native huts. At night they crawl to sleeping hosts, scrape or otherwise break the skin, and suck the oozing blood. After feeding for about 20 min, the maggots return to hide in debris until the next night. They require two bloodmeals for each of the three instars and pupate in debris. The life cycle takes about 10 weeks.

Deer and Water Buffalo Skin Maggots (Boopona spp.)

These myiasis-causing flies include four species of yellow-brown blow flies whose maggots parasitize the skin of the back and feet of cervids and bovids in Eastern Europe, Asia, and the Orient. In the case of cervids, they also attack the soft, developing antler buds. Their eggs are attached singly to hairs of the host and require 3–5 days to develop prior to hatching. These maggots invade the host skin, where they develop individually in warble-like boils in about a 1-week period. Mature maggots drop from the host to the ground to pupate, and adults emerge in 2–3 weeks.

Elephant Skin Maggot (Elephantolomeus indicus)

This species is a small, orange-brown blow fly whose maggots develop only in warble-like boils in the skin of the Asian elephant. Little is known about its biology.

Sarcophagidae (Flesh Flies)

Species of this large, widely distributed family are classified into two subfamilies: the Miltogramminae, which, with few exceptions, are obligatory parasitoids of insects and other arthropods, and the Sarcophaginae,with necrophagous species that include facultative and obligatory parasites causing myiasis. Adults (Fig. 19.16) are typically

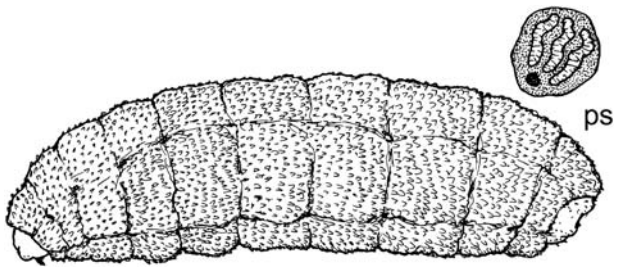


FIGURE 19.14 Tumbu fly, *Cordylobia anthropophaga*, third-instar larva (Calliphoridae). *ps*, posterior spiracular plate. *Original by E. Paul Catts.*

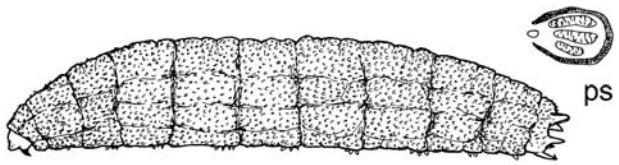


FIGURE 19.15 Congo floor maggot, *Auchmeromyia senegalensis* (Calliphoridae), third instar. *ps*, posterior spiracular plate. *Original by E. Paul Catts.*

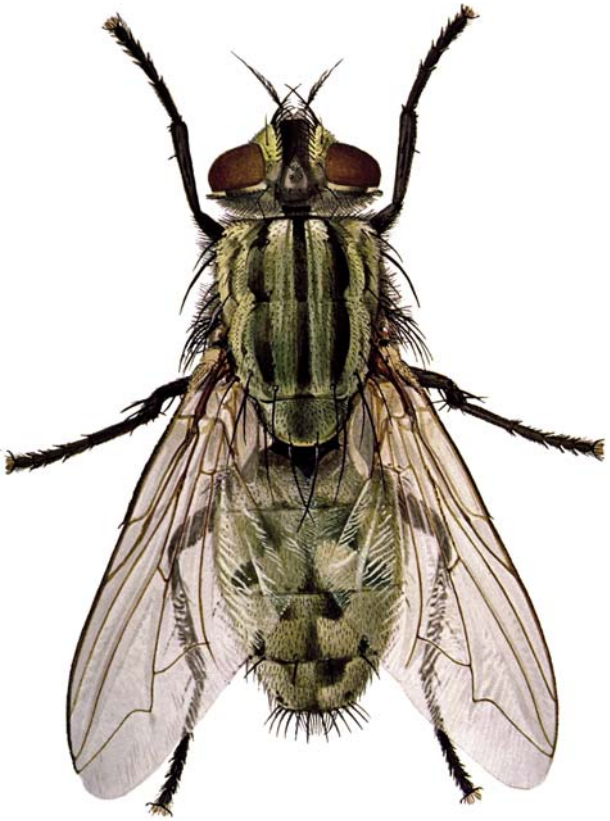


FIGURE 19.16 Red-tailed flesh fly, *Sarcophaga haemorrhoidalis* (Sarcophagidae), adult. *From Greenberg (1971).*

medium-sized to large, black and gray flies with longitudinal thoracic stripes and a checkered, or tessellated, abdominal pattern. All sarcophagid species are larviparous; the gravid female retains the eggs in an expanded, bilobed, uterine pouch until they are ready to hatch. Females produce 30–200 larvae, depending on the species involved. Flesh fly larvae (Fig. 19.17) are more robust than blow fly larvae and possess paired mandibles in all instars. Their posterior spiracles are recessed in a deep cavity, and the inner slit of each spiracle is parallel to, or slants away from, the ventral mid-body line.

Sarcophaga spp. usually are associated with carrion or feces but can cause facultative wounds and accidental gastrointestinal myiasis. About 20 *Sarcophaga* spp. have been incriminated in cases of gastrointestinal myiasis. One widely distributed species is the **red-tailed flesh fly** (*S. haemorrhoidalis*), which frequents feces and is attracted indoors by fecal odors. Only a few species in other sarcophagine genera have been recorded as causing myiasis of the gastrointestinal or wound type.

Few flesh flies cause obligatory myiasis. The most widespread and important species are in the miltogrammine genus *Wohlfahrtia*. They include the Old World species *W. magnifica* and the New World species *W. opaca* and *W. vigil*. These three species have evolved as primary

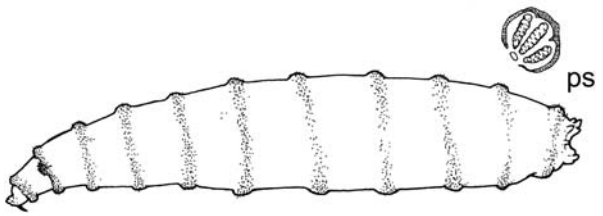


FIGURE 19.17 Flesh fly, *Sarcophaga* sp. (Sarcophagidae), third-instar larva. *ps*, posterior spiracular plate. *Original by E. Paul Catts.*

invaders. Females larviposit at moist body openings and at fresh wounds or scratches. Larvae can even penetrate thin, unbroken skin. The maggots burrow into the subcutaneous tissue to feed, inducing the formation of a boil-like cyst around groups of larvae with a small, common breathing pore opening to the outside.

Wohlfahrtia adults resemble large house flies with very distinct, longitudinal, thoracic stripes. Unlike other flesh flies, these have the abdomen clearly marked with black spots. A gravid *Wohlfahrtia* female produces 120–170 larvae. In a host, the maggots grow rapidly and can cause considerable tissue destruction. After about 1 week, maggots drop to the ground to pupate and can overwinter in this stage.

Wohlfahrtia magnifica has become a major cause of myiasis in Europe, where it causes damage to livestock raised in both extensive and intensive systems. The closely related *W. nuba* is not parasitic but feeds only on necrotic flesh. It has been used successfully in treating ragged, infected wounds similar to the use of *Lucilia sericata*. Most other *Wohlfahrtia* spp are scavengers, but all are probably capable of at least facultative myiasis. An uncommon species that has been reported from ground squirrels (*Spermophilus* spp.) is *Neobellieria citellivora* (Shewell, 1950). In ground squirrels, infection is lethal and can overwhelm them in approximately 7 days (Michener, 1993).

A group of four unrelated genera of flesh flies cause facultative, and apparently obligatory, myiasis of certain amphibians and reptiles. In some cases, they also attack amphibian and reptilian eggs, killing the developing embryos. These genera are *Anolisimyia*, *Cistudinomyia*, *Eumacronychia*, and *Metoposarcophaga*. Little is known about their biology.

Oestridae (Bot Flies)

Bot flies are the most highly evolved group of obligate myiasis–causing parasites of mammals. They are treated as four distinct subfamilies in the Oestridae. The most primitive are the Cuterebrinae, the **New World skin bot flies**. Their counterparts are the Hypodermatinae, the **Old World skin bot flies**. The **nose bot flies** are in the Oestrinae with their probable center of origin being in Africa. The remaining

subfamily is Gasterophilinae, the **stomach bot flies,** which also appears to have evolved in Africa. All four of these subfamilies were recognized previously as families and are treated as such in earlier literature. The subfamilies of Oestridae can be separated as third-instars by their general appearance and by the form of their caudal spiracular openings. Bot fly maggots are thick, robust, grublike larvae with moderate to heavy spiny armature. As with most other flies discussed in this chapter, they pass through three larval instars and drop to the ground to pupate. Except for members of the Cuterebrinae, all bot flies overwinter as larvae in the host. The cuterebrines typically overwinter as diapausing pupae free from the host, although there are exceptions in subtropical and tropical regions.

Bot flies differ from other obligatory myiasis producers in several ways. First, the adults do not feed or take in nutrients. Most of them have only rudimentary, nonfunctional mouthparts and are unable to feed. Those few with functional mouthparts and an associated alimentary tract probably only imbibe water to maintain an internal fluid balance. Second, bot flies (with the notable exception of *Dermatobia hominis*) either show a high degree of host specificity or parasitize only a small group of related hosts. Although some bot fly maggots occasionally occur on atypical hosts, the susceptibility of a host does not necessarily imply the suitability of that host for normal, or successful, bot fly development. Third, bot fly maggots show a marked level of site specificity in a normal host. In abnormal hosts, site specificity can be erratic and can lead to dire results for both host and parasite. Fourth, the site of invasion by the first-instar bot maggot generally is not the site of maggot development. With the exception of *D. hominis*, first-instar oestrid flies move from the point of invasion to a different site for further development. Interaction between the host and its developing bot fly maggots is generally benign, with the associated pathology and parasite burden being tolerated well by native, coevolved hosts. Bot fly maggots generally cause little injury to their hosts at low to moderate population levels. However, some recent research has shown that infestation of hosts can either result in juvenile death (howler monkeys) or result in the loss of body fat, which causes female reindeer to become reproductively sterile.

Humans are not among the normal hosts for any bot fly species, including the so-called human bot fly (*D. hominis*). However, people may become incidentally infested by bot flies under certain circumstances. In such cases, the associated pathology tends to be more severe than that of their normal hosts.

Burrowing first-instar bot flies occasionally cause paralysis or death of the host. Developing larvae located in warbles at critical sites such as around the eyes and on the feet can increase the risk of predation by interfering with the host's ability to see or escape. Small mammalian hosts encumbered by an ever-enlarging cluster of warbles also may have difficulty in foraging.

Another characteristic of bot flies is that the bee-like adults usually aggregate at specific topographic sites for pairing and copulating. Favored sites are hilltops, cliff faces, steep slopes, prominent rocks or trees, and streambeds. Male flies remain at these sites throughout their brief life, but females leave the sites soon after mating to search for suitable hosts or oviposition sites.

The major importance of bot flies is the economic losses that they cause in livestock operations (e.g., cattle, sheep, goats, reindeer, and horses). Secondary microbial infection of the bot warble is rare because bot fly maggots produce bacteriostatic secretions as they develop. Even after the larva exits the warble, other myiasis-causing flies rarely exploit the empty wound which normally heals very rapidly. Bot fly maggots cannot complete their development in a dead host. If the host dies, so do its bots.

The evolutionary history of bot flies is not known, but warrants comment. Zumpt (1965) proposed two possible routes for bot fly evolution. One route is through blood-sucking larvae such as nest maggots or floor maggots. The other is through carrion-breeding species and screwworms. Both alternatives seem plausible about skin bots, but they do not explain how the more internally adapted groups, such as nose bots and stomach bots, originated. The nose bots may have evolved from myiasis-causing flies that were attracted to mucopurulent nasal secretions in hosts suffering from respiratory infections. Stomach bots, on the other hand, may have originated from fly species infesting decaying, fermenting forage, a diet favored by many large herbivores. Interesting discussions of evolution of the oestrid flies and their hosts can be found in treatments of this subject by Papavero (1977) and Pape (2006).

Many of the oestrid flies can now be identified by molecular sequencing techniques and in particular high throughput sequencing, which makes it possible to clearly identify those species that are involved in myiasis. While the coverage is not nearly complete, there is an increasing number of species that have been sequenced at least for their cytochrome oxidase 1 (*COX1*) gene (which is generally accepted for the barcoding of species). Recently there have been several examples in which traditional methods of identification (morphology) have been overturned by the molecular data. In some of these cases, it will require a thorough examination of the concepts of host specificity and host range.

New World Skin Bot Flies (Cuterebrinae)

There are two genera and 58 species in this subfamily of bot flies, all restricted to the Western Hemisphere. The

largest genus is *Cuterebra* (57 species), which includes some of the largest bot flies. Some of these robust, thick-bodied bot flies (Fig. 19.18) are up to 30 mm in length. Their normal hosts are rodents (e.g., *Microtus*, *Neotoma*, and *Peromyscus* spp.) and lagomorphs (e.g., *Lepus* and *Sylvilagus* spp.). At temperate latitudes, cuterebrine maggots show seasonal peaks in prevalence. For example, 40% prevalence in *Peromyscus* populations is not unusual during late summer. Non-native rodents and rabbits (e.g., *Mus*, *Rattus*, *Cricetus*, and *Oryctolagus* spp.) also are parasitized, but in these hosts the pathology is more severe and can lead to death of both the host and parasite.

Cuterebra spp. oviposit in areas close to the center of host activity (e.g., near nests and lairs or along runs). After about 1 week, the eggs hatch in response to a sudden increase in temperature, normally indicating a nearby, warm host. First-instar maggots adhere to the host pelage, crawl to natural body orifices of the head, and penetrate the mucosal tissue at such sites as the mouth and nose. After about 1 week in the pharyngeal areas of the host, the maggots actively burrow through sheets of host connective tissue to a species-specific, cutaneous site for maggot development (Fig. 19.19). Once there, the maggot cuts an opening through the skin and molts within the newly formed warble. Depending on the bot species involved, maggot development at this site requires 3–8 weeks, during which the much-enlarged maggot can increase some 100,000-fold in weight. When mature, the third instars (Fig. 19.20) back out through the warble pore and drop to the ground to pupate. After the bot exits, the collapsed warble heals quickly, usually without secondary infection. In cool, temperate regions, it is the pupa that diapauses and overwinters, and there is only one adult flight season per year. Warmer areas probably have two flight seasons where adults are on the wing during late spring and summer. The adult life span is about 2 weeks.

FIGURE 19.18 Rodent bot, *Cuterebra fontinella*, adult female (Oestridae, Cuterebrinae); reared from white-footed mouse, *Peromyscus leucopus*. *Photograph by Sturgis McKeever.*

FIGURE 19.19 Cotton mouse, *Peromyscus gossypinus*, with mature rodent bot, *Cuterebra* sp., or wolf; posterior end of bot, with posterior spiracles exposed, projecting from location at base of host tail. *Photograph by Gary R. Mullen.*

Generally, there is little economic importance associated with *Cuterebra* spp., although they can be a seasonal problem in commercial rabbit operations. Sport hunters often discard bot-infested squirrels (Fig. 19.21) and rabbits in the erroneous belief that the carcasses are spoiled by the presence of these maggots. A few *Cuterebra* spp., especially *C. fontinella*, have been colonized in the laboratory and used as natural bot-host models for the study of bots affecting livestock.

Tórsalo (Dermatobia hominis)

The **tórsalo** is a Neotropical species that occurs widely from southern Mexico to Argentina. In Brazil this very important parasite is known locally as **berne**. Although primarily a pest of cattle, it also infests humans, dogs, monkeys, sheep, horses (rarely), and other domestic and wild mammals. This is the only bot fly that frequently parasitizes humans, hence its alternative common name, the **human bot fly**.

It is a woodland species encountered along forest margins of river valleys and lowlands. It is unusual among cuterebrine flies because of its unique oviposition behavior and means of egg dispersal. Rather than depositing eggs directly onto a host, the adult female (Fig. 19.22) captures various zoophilic or anthropophilic arthropods, usually

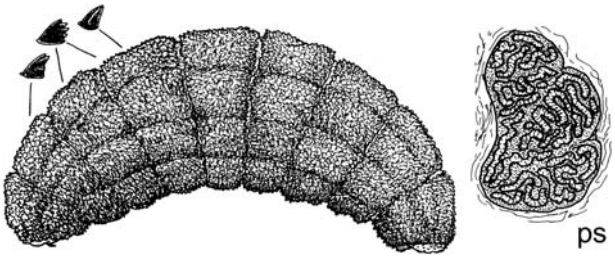


FIGURE 19.20 Rodent bot, *Cuterebra* sp. (Oestridae, Cuterebrinae), third-instar larva. *ps*, posterior spiracular plate. *Original by E. Paul Catts.*

FIGURE 19.21 Multiple wolves of squirrel bot (*Cuterebra* sp.) in shoulder area of gray squirrel, *Sciurus carolinensis*. *Photograph by Martin Bennett; courtesy of Wikimedia Commons.*

dipterans, and glues her eggs in clusters (6–45 eggs) to their abdomen (Fig. 19.23). Embryo development requires 5–15 days. These egg carriers, or **porters**, subsequently transport the eggs to a vertebrate host, where they hatch while the arthropod feeds. Among the more common porters are day-flying mosquitoes, such as *Psorophora ferox* (Fig. 19.24) and muscid flies (e.g., *Sarcopromusca pruna*, *Stomoxys calcitrans*, *Fannia* spp., and *Synthesiomyia* spp.). The newly emerged larvae enter the skin either through the feeding puncture or via hair follicles, soft folds of skin, or areas of moist skin in contact with clothing or bedding. Development occurs at the point of entry, forming a boil-like

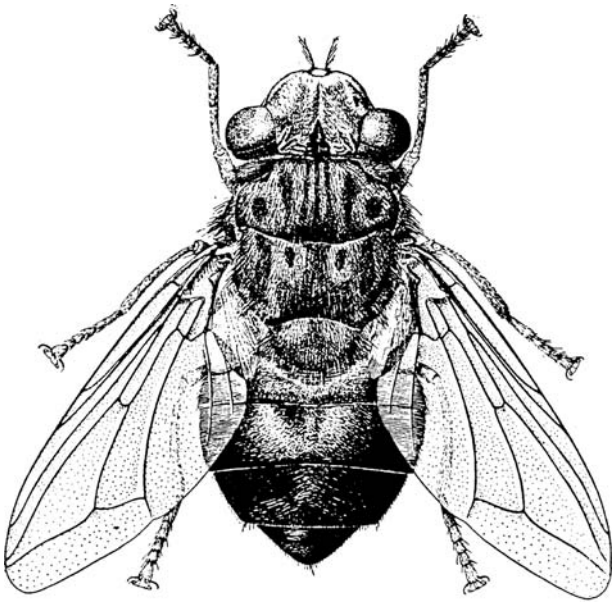


FIGURE 19.22 Tórsalo, or human bot fly, *Dermatobia hominis* (Oestridae, Cuterebrinae), adult female. *From James (1947).*

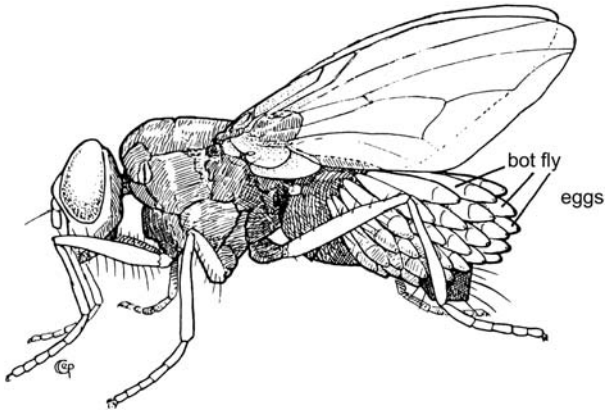


FIGURE 19.23 A muscid fly porter (*Sarcopromusca arcuata*) to which a tórsalo bot fly (*Dermatobia hominis*) has attached her eggs. *Original by E. Paul Catts.*

pocket, or furuncular lesion, where the larva (Fig. 19.25) undergoes three instars. This development usually takes 5–10 weeks but sometimes as long as 3 months or more. During this time the narrower posterior end of the larva is extended into the opening at the skin surface, where it exchanges air in respiration (Fig. 19.26). After the larva matures, it enlarges the opening and drops to the ground to pupate. The pupal stage lasts 25–132 days. The longer period occurs in the colder regions of Brazil and Argentina, where the pupae are able to overwinter (Ribeiro, 2007).

Other Cuterebrine Flies

The remaining cuterebrine flies are tropical and include only a few species. Most of them parasitize rodents and marsupials, and what is known about their biology has been described by Guimaraes and Papavero (1999) as separate genera. *Cuterebra baeri* parasitizes howler monkeys (*Alouatta* spp.) in Central and South America and has been implicated in reduced survival of juvenile monkeys. This is

FIGURE 19.24 Female mosquito, *Psorophora ferox*, with eggs of the bot fly *Dermatobia hominis* attached to the underside of her abdomen. Note the first-instar bot fly larva emerging from one of the eggs; French Guyana. *Photograph by Lawrence E. Reeves.*

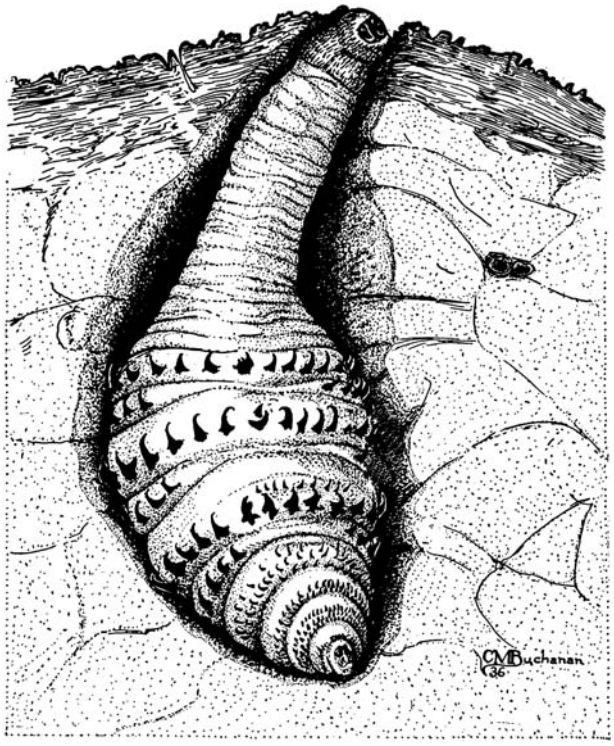


FIGURE 19.25 Tórsalo, or human bot fly, *Dermatobia hominis* (Oestridae, Cuterebrinae), mature larva in human skin, with posterior spiracles exposed through hole in skin surface. *From Craig and Faust (1940).*

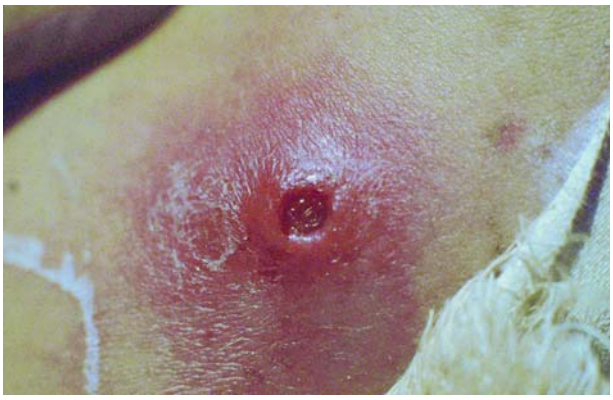


FIGURE 19.26 Tórsalo, or human bot fly, *Dermatobia hominis;* posterior end of maturing larva visible in hole made by larva in skin on human forearm. Note inflammation and swelling at site of wound. *Photograph by Ronald D. Cave.*

the only bot fly specific to a primate host. Warbles of this species usually are located in the cervical and axillary regions of their arboreal hosts.

Old World Skin Bot Flies (Hypodermatinae)

These flies are the Eurasian counterpart of the New World skin bots. There are nine genera and 27 species occurring in rodents, deer, goats, and cattle. All except two species are found only in Asia, Europe, and Africa. The most widespread and important species are in the genus *Hypoderma*, which includes six species, three causing myiasis in cervids—one in yaks, and two in bovids.

Cattle Grubs (Hypoderma spp.)

The **northern cattle grub** (*Hypoderma bovis*) and the **common cattle grub** (*H. lineatum*) (Fig. 19.27) are Holarctic in distribution, having been introduced wherever cattle are raised. They are major economic pests of domestic cattle. Losses include damage to hides and self-injury by hosts during headlong flights of panic, or gadding (Fig. 19.28), in futile attempts to escape ovipositing flies. Thus, the name **gad fly** is often used to refer to the adults. Adults of *Hypoderma* spp. also are called **heel flies**, referring to the defensive behavior of cattle in kicking up their hooves.

The biologies of *H. bovis* and *H. lineatum* are very similar. In late spring and early summer, adult females (Fig. 19.29) of both species glue their eggs directly to host hairs. *Hypoderma lineatum* usually deposits rows of eggs (Fig. 19.30) on the lower body regions of standing or even recumbent hosts, whereas *H. bovis* normally deposits single eggs in the same regions on active hosts. Presence of the latter species is what causes cattle to gad. After an incubation period of 3−7 days, eggs hatch and the first instars crawl to the base of the hairs on which the eggs were glued. They then penetrate the host skin using proteolytic enzymes. A 4-to-6-month period of migration and overwintering follows as the larvae make their way between sheets of connective tissue and fascial planes within the host, or along nerve pathways for *H. bovis*. During the winter, first instars of *H. lineatum* can be found in the esophageal submucosa, whereas the larvae of *H. bovis* can be found within the epidural fat along the spinal column. With the onset of spring the larvae of both species leave these "resting" sites and move to the host's back where they cut a hole, the warble pore, and develop through two subsequent larval instars. A boil-like warble develops around the enlarging maggot (Fig. 19.31). The cuticle of mature grubs is black, and these mature larvae back out of the warble pore and drop to the ground to pupate. As with all bots, it is at this time of dropping from the host that grubs are most vulnerable to predation by birds, rodents, and insectivores. The pupal stage lasts 1−3 months, depending on ambient temperature. Adult flies live only 3−5 days and fly quickly after emerging to mate and oviposit throughout late spring and early summer.

Another *Hypoderma* species worthy of note is *H. tarandi* (formerly *Oedemagena tarandi*). In the arctic and subarctic regions, *H. tarandi* causes cutaneous myiasis in the backs of reindeer and caribou, similar to that caused by cattle grubs. Heavier infestations generally occur in yearling fawns rather than in other age groups. Over time, infested animals slowly develop partial immunity to these parasites. The other three

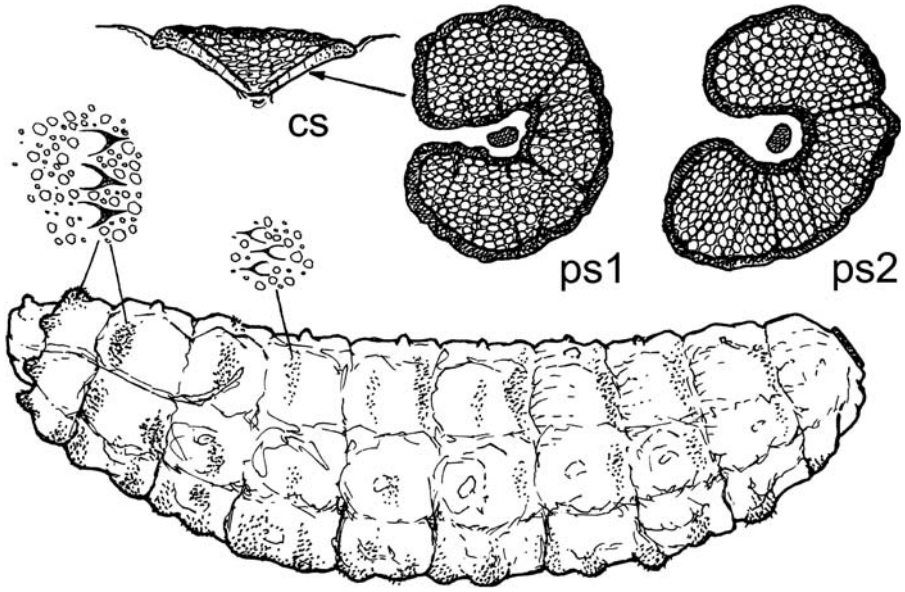


FIGURE 19.27 Cattle grubs, *Hypoderma* spp. (Oestridae, Hypodermatinae), third instars. *cs*, cross section of posterior spiracle of *H. bovis*, showing depressed center which contrasts with flat spiracle of *H. lineatum*; *ps1*, northern cattle grub (*H. bovis*), third instar; *ps2*, common cattle grub (*H. lineatum*), third instar. *Original by E. Paul Catts.*

hypodermatine species (*H. actaeon*, *H. diana*, and *H. sinensis*) responsible for warbles in cervids and yaks are also Holarctic in distribution and have similar biologies. *Oestromyia* spp. (Fig. 19.32), *Portschinskia* spp., and *Ochotonia lindneri* commonly cause cutaneous myiasis in wild rodents and lagomorphs in Europe, Asia, and the Far East. The remaining five hypodermatid genera (*Strobiloestrus*, *Pallasiomyia*, *Oestroderma*, *Pavlovskiata*, and *Przhevalskiana*) have been reported from African antelopes and lechwes, Saiga antelopes, Asian pika, Asian gazelles, and Middle Eastern and African sheep, goats, and gazelles, respectively.

A new genus/species of Hypodermatinae was recently described from a single male specimen collected from a region of Iran. *Gruninomyia mira* has morphological characteristics that place its subsequent larvae in either a lagomorph or artiodactyl host. Its *COX1* gene sequence, while provided, does not assist in its current placement.

Nose Bot Flies (Oestrinae)

This subfamily of oestrid flies includes nine genera and 34 species that parasitize members of the mammalian orders Artiodactyla, Perissodactyla, and Proboscidea (elephants).

FIGURE 19.28 Gadding behavior of calf in response to attack by heel fly (*Hypoderma bovis*), with tail raised and calf kicking up heels of hind legs while running frantically about. *Photograph by J. Weintraub, Agriculture and Agri-Food Canada.*

FIGURE 19.29 Heel fly, *Hypoderma bovis* (Oestridae, Hypodermatinae), adult female. *Courtesy of Agriculture and Agri-Food Canada.*

FIGURE 19.30 Eggs of heel fly, *Hypoderma lineatum* (Oestridae, Hypodermatinae), attached to body hair of cow. *Courtesy of Agriculture and Agri-Food Canada.*

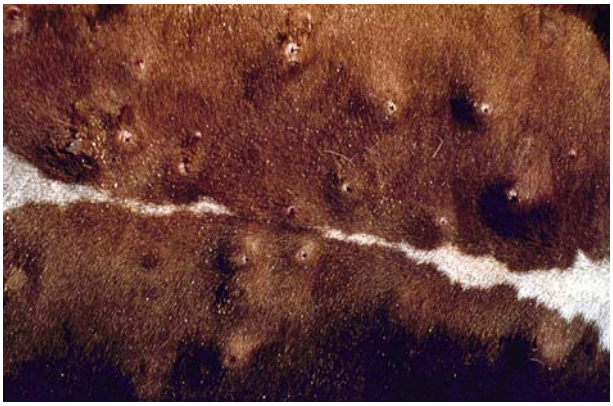


FIGURE 19.31 Multiple warbles along back of cow, caused by *Hypoderma bovis* (Oestridae, Hypodermatinae). *Photograph by J. Weintraub, Agriculture and Agri-Food Canada.*

Most nose bot flies are African or Eurasian in distribution; an exception is the Holarctic genus *Cephenemyia*. The genera *Oestrus* (six species) and *Rhinoestrus* (11 species) comprise most species in this group. Another species, *Tracheomyia macropi* (Fig. 19.33), develops in the trachea of the red Kangaroo in Australia and is the only native bot of that continent.

Nose bot flies differ from other bots in that their eggs develop in utero. First instars are ejected by the hovering female directly into the muzzle or eye of the host. The larvae crawl down the throat to enter tracheal branches of the lungs but soon return to the nasal sinuses or pharyngeal region of the host to complete their development. As with other bots in native hosts, there is little pathology at moderate parasite levels. However, purulent mucous exudates associated with an abundance of maggots may lead to respiratory complications or to secondary fly attack. While hosts are under attack by adult nose bots, they stop grazing and attempt to thwart the attack by pushing their muzzles into bushes or clumps of grass. Following development, mature larvae are sneezed from the nostrils of the host, causing some temporary suffering during this time. Occasionally a few larvae may become lodged in the nasal sinuses, which can cause the death of their host. After a pupal period of 4–6 weeks, adults emerge and seek a mate at aggregation sites. Adults generally are univoltine in cold regions and at least bivoltine in tropical and warm temperate areas. This indicates that larval development can be delayed during winter and accelerated during summer. Overwintering takes place within the host, as is the case of most other bot flies.

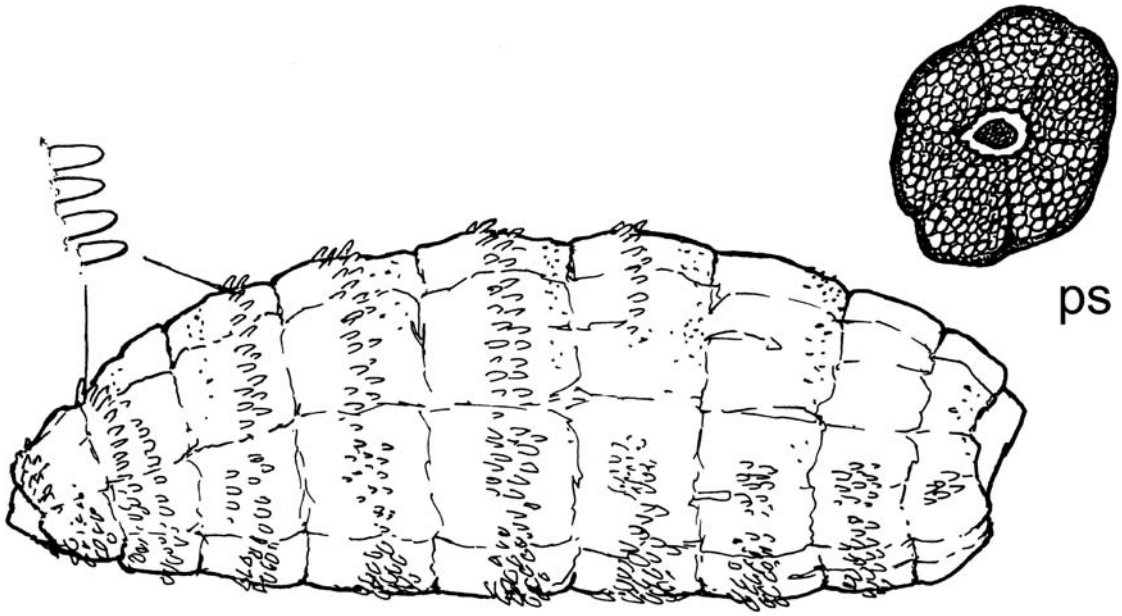


FIGURE 19.32 *Oestromyia* sp. (Oestridae, Hypodermatinae), third-instar larva; causes myiasis in wild rodents and lagomorphs. *ps*, posterior spiracular plate. *Original by E. Paul Catts.*

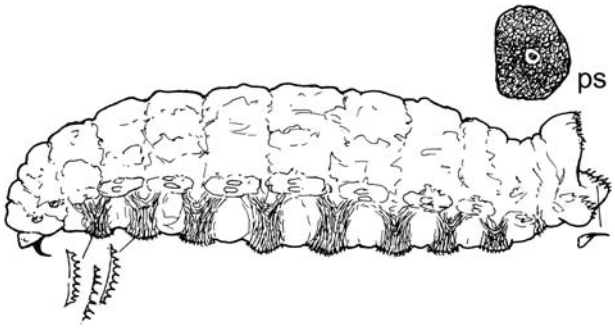


FIGURE 19.33 Kangaroo throat bot, *Tracheomyia macropi* (Oestridae, Oestrinae), third-instar larva; develops in trachea of the red kangaroo, Australia. *ps*, posterior spiracular plate. *Original by E. Paul Catts.*

The most widely distributed and economically important species is the **sheep nose bot** (*Oestrus ovis*), which parasitizes domestic and wild sheep and goats (Figs. 19.34 and 19.35). Females produce about 500 progeny that they larviposit in batches of 10−25 at a time. The usual host burden is 12−24 maggots, with gradual attrition reducing this number to fewer than 10 survivors by the time the maggots mature. Other *Oestrus* species parasitize antelopes and wild goats in Africa and Asia.

The **horse nose bot** (*Rhinoestrus pupureus*) is distributed widely throughout Eurasia, Africa, and the Orient. It is most prevalent in Asia, where high population levels of these bots in domestic horses have been recorded (>700 maggots in a single host). High parasite loads can cause death of the host. Ocular myiasis in people who live near, or handle, horses is not uncommon. The general life history of the horse nose bot is like that of *O. ovis*. *Rhinoestrus* includes 11 species, four in equids and seven others in

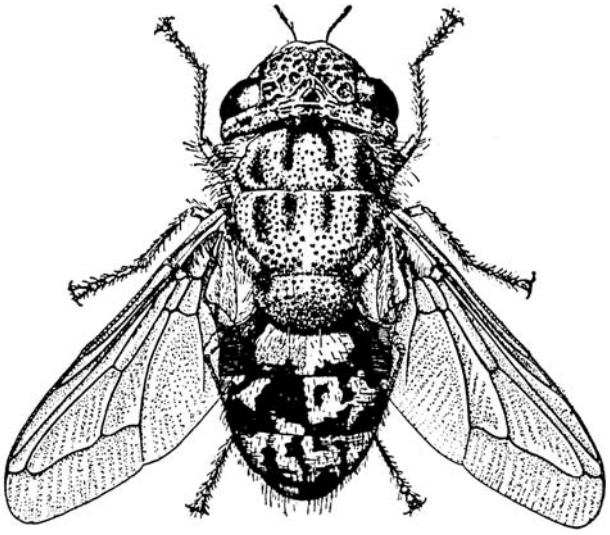


FIGURE 19.34 Sheep bot fly, *Oestrus ovis* (Oestridae, Oestrinae), female. *From James (1947).*

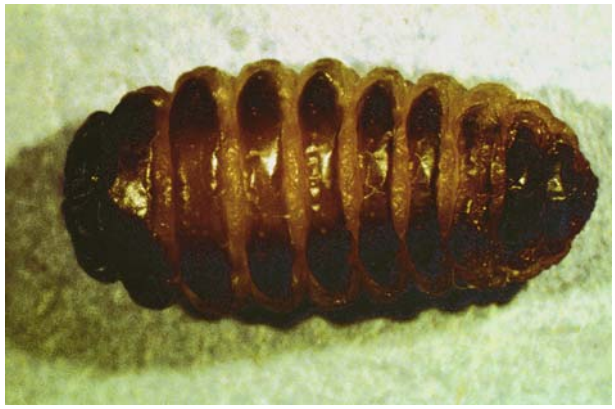


FIGURE 19.35 Sheep bot, *Oestrus ovis* (Oestridae, Oestrinae), third-instar larva. *Courtesy of Philip J. Scholl.*

specific nonequine hosts (e.g., giraffe, antelope, bush pig, hippopotamus, and warthog).

Eight species in the genus *Cephenemyia* (Fig. 19.36) parasitize cervids (deer). Only one, *C. trompe*, is distributed throughout the northern Holarctic region. The others are confined to either the Nearctic or Palearctic areas. All are **deer nose bots**. The four common species in North America are *C. phobifer* to the east of the Continental Divide and *C. apicata*, *C. jellisoni*, and *C. pratti* to the west. Although *C. trompe* is named the **reindeer throat bot**, it occurs in deer, moose, and caribou as well as reindeer. The life history of these bots is like that described for other nose bots. Mature maggots usually crowd the retropharyngeal pouches in the throat of their host. On completing their development, they crawl to the anterior nasal passages, where they are expelled by sneezing and pupate on the ground under surface debris.

The incidental occurrence of several oestrine species in the eyes of atypical hosts suggests that the orbit also may serve as a target for certain larvipositing bot flies. In Africa, first instars of *Gedoelstia cristata* regularly occur in the eyes of native wildebeest and hartebeest hosts. Later instars are found in the nasal cavities. Ocular invasion by this species in domestic sheep, goats, cattle, and horses

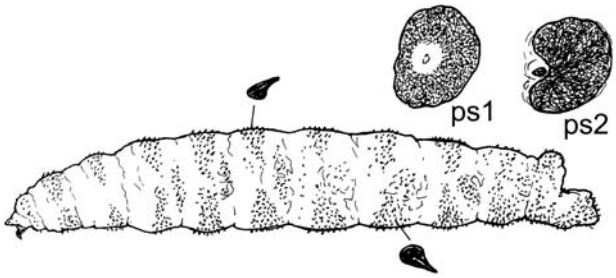


FIGURE 19.36 Deer nose bot, *Cephenemyia jellisoni* (Oestridae, Oestrinae), third-instar larva. *ps*, posterior spiracular plate. *Original by E. Paul Catts.*

produces gross pathological lesions and high levels of mortality. *Cephalopina titillator* (Fig. 19.37) is the nose bot of camels and dromedaries from Central Asia to Africa. Its life history is like that of other nose bots.

Stomach Bot Flies (Gasterophilinae)

Adult flies of this group, represented by 17 species in five genera, resemble honey bees in their general size and color. The largest genus is *Gasterophilus*, the **horse stomach bot flies**, with nine species, three of which have worldwide distribution (*G. intestinalis*, *G. nasalis*, and *G. haemorrhoidalis*). These parasites are common companions of horses and donkeys wherever these hosts occur. Few horses live to old age without having carried their load of stomach bots along the way.

Following oral entry, developing maggots attach to the gastrointestinal mucosa (Fig. 19.38) causing inflammation, sloughing of tissue, and ulcerations. They do not cause warble formations. Burrowing of first instars in the mouth lining, tongue, and gums can produce pus pockets, loosen teeth, and cause loss of appetite of the host. A gadding response to hovering, ovipositing females can cause self-inflicted injuries as well. However, *G. nigricornis* does not hover but alights on the host's cheek to oviposit, and *G. pecorum* oviposits on leaves and stems of potential host forage.

Most gasterophiline flies glue their eggs to the hair shafts of the host's body. Because of their brief life span of a few days at most, females can deposit all their eggs within a couple of hours if hosts are available and the weather is mild. Their larvae develop in the stomach and intestines of equids, elephants, and rhinoceroses, suggesting a very old coevolutionary relationship with these distantly related mammalian groups. Eggs that are ingested with forage or wetted by self-grooming of the front legs hatch after a brief incubation. Eggs located on the host's head hatch spontaneously. Upon burrowing into the oral tissues for a brief period, first instars eventually are swallowed and attach to the stomach or intestinal wall. The site of attachment is specific for each fly species. Larvae overwinter in the gastrointestinal tract. After larval development is completed, the mature larvae are expelled with the host feces during the warmer seasons. Pupation occurs in the soil soon after larvae drop from the host, with the pupal stage lasting about 3 weeks. Adult flies emerge, mate, and quickly resume activity near potential equine hosts. If hosts are not available, the bot flies move to high points to aggregate, then the females initiate a longer-distance search for hosts.

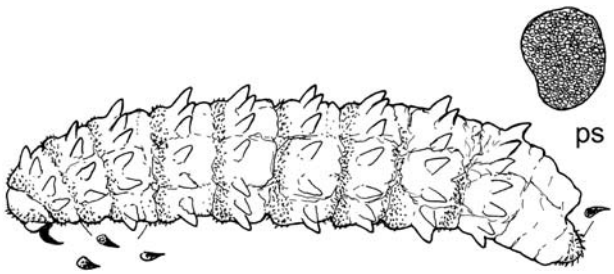


FIGURE 19.37 Camel nose bot, *Cephalopina titillator* (Oestridae, Oestrinae), third-instar larva. *ps*, posterior spiracular plate. *Original by E. Paul Catts.*

FIGURE 19.38 Common horse stomach bot, *Gasterophilus intestinalis* (Oestridae, Gasterophilinae); larvae attached to mucosa and inner surface of stomach of heavily infested horse. *Courtesy of The Natural History Museum, London.*

The **common horse stomach bot fly** (*Gasterophilus intestinalis*) (Figs. 19.39 and 19.40) is worldwide in distribution and is the predominant species in North America. It prefers to oviposit on the lower forelegs of horses. The two other species in North America are the **throat horse bot**, (*G. nasalis*) and the rarer **horse nose bot** (*G. haemorrhoidalis*). The former oviposits on the hairs of the chin and lower jaw and the latter on the hairs of the nose and lips. The **dark-winged horse bot** (*G. pecorum)* is the most commonly encountered species in Eurasia and Africa. It is the most pathogenic fly in this genus and can cause host fatalities resulting from constricted swelling of the esophagus due to attached maggots.

Other genera of stomach bots include *Gyrostigma* (three species) in rhinoceroses and *Cobboldia* (three species) parasitizing African and Indian elephants. Their life histories are similar to that of *Gasterophilus*. Two other obligate myiasis fly species are associated with the African elephant. Both are cutaneous parasites. *Ruttenia loxodontis* develops in warble-like skin boils on the buttocks and flanks of elephants, whereas *Neocuterebra squamosa* develops in shallow ulcers in the skin crevices of the elephant's feet. Both produce a **pseudo warble** during their development. The phylogenetic relationship of these unplaced genera to other bot flies is uncertain but recently these genera have been placed with the gasterophilines (Colwell et al., 2006). This placement agrees with Zumpt (1965), who suggested that both species represent early gasterophilines.

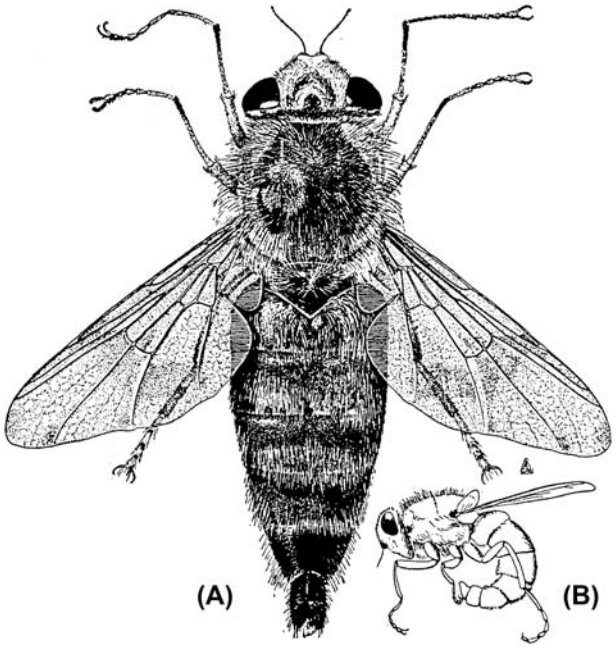


FIGURE 19.39 Common horse bot fly, *Gasterophilus intestinalis* (Oestridae, Gasterophilinae), adult female. (A) Dorsal view. (B) Lateral view. *From James (1947).*

Other Oestroid Flies

There are three other families of flies included in the Oestroidea in which the species do not parasitize vertebrate animals: Tachinidae, Rhinophoridae, and Mystacinobiidae. All members of the **Tachinidae** are obligate parasitoids of other insects. None are involved in myiasis. The **Rhinophoridae** are a small sister group of the tachinids that parasitize isopods. **Mystacinobiidae**, a sister group of the calliphorids, includes but a single species in New Zealand that is coprophagous on bat guano and phoretic on the bats themselves. None of these families include myiasis-causing species.

PUBLIC HEALTH IMPORTANCE

Myiasis is a relatively uncommon ailment among people worldwide, occurring only seasonally and sporadically in temperate regions and associated with wet seasons in the tropics. The notable exception occurs in South America

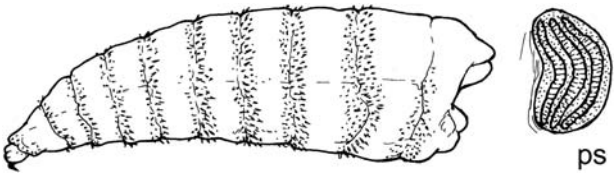


FIGURE 19.40 Common horse stomach bot, *Gasterophilus intestinalis* (Oestridae, Gasterophilinae), third-instar larva. ps, posterior spiracular plate. *Original by E. Paul Catts.*

where several hundred cases of infestation in humans by the New World screwworm, *Cochliomyia hominivorax*, have been recorded, most often in individuals in a severely debilitated condition (Guimaraes and Papavero, 1999). In some African countries such as Nigeria, a recently published study showed a prevalence of 82%–88% of furuncular myiasis caused by *Cordylobia anthropophaga* (Kasiemobi et al., 2012). Most human cases of facultative or obligatory myiasis are only temporary or are aborted because humans are unsuitable hosts (e.g., nose or stomach bots). Maggots normally do not complete their development in people because they usually are interrupted by self-grooming or medical intervention. Infants and infirm or debilitated older age groups are generally at greater risk because of associated difficulties in maintaining a minimal level of personal hygiene. Soiling of bedding or clothing with excrement can result in invasion through urogenital or anal sites. **Gastrointestinal myiasis** usually results from the ingestion of eggs or maggots with infested foods, commonly causing general malaise, nausea, vomiting, cramps, and diarrhea. Although such cases are seldom serious, adverse effects can be prolonged and may require purging of the alimentary tract to resolve the problem.

Facultative or obligatory **wound myiasis** is more serious because of the potential for rapid and indiscriminate destruction of tissues. Infirm persons are at particular risk, as are people who work and live in close proximity to livestock operations where carrion and suitable hosts are more available. Myiasis involving other flesh flies and bot flies occasionally occurs in people. The former usually involves infants, and the latter, livestock handlers and field workers. **Ophthalmic myiasis** is the most common form of human myiasis, for which numerous cases are recorded. It is usually caused by the sheep nose bot when a gravid female larviposits directly into an eye while hovering. As many as 50 first-instar *O. ovis* maggots have been removed from the eye following the strike of a single fly. Although the larvae do not actually penetrate the eye tissues, they can cause extreme irritation as they crawl about on the eyeball. They are best removed by flushing the eye with fluids or by gentle suction.

An aberrant type of **cutaneous myiasis** in humans is caused by first instars of *Gasterophilus* and *Hypoderma* species. In such cases, larvae hatching from eggs laid on a person or rubbed off by contact with the animal's skin (i.e., riding bareback or milking) penetrate the skin and wander in the epidermis, producing visible, sinuous, inflamed tracks accompanied by irritation and pruritus. This is called **cutaneous larval migrans**. If cases involving horse bot flies (*Gasterophilus* spp.) are not treated, the larvae eventually die. However, cattle grubs (*Hypoderma* spp.) and reindeer grubs can cause much more severe pathology in humans, sometimes with fatal results. The larvae are able to penetrate and burrow into deeper tissues with the potential

for serious consequences because they are not well adapted to human hosts. More cases involve *H. bovis* than *H. lineatum* and tend to be more severe in children. Both have been reported as causing ophthalmic myiasis and skin warbles, the latter usually in the neck and shoulders. Several human cases involving prolonged, but temporary, paralysis have been reported.

New World bot flies also parasitize humans. More than 60 cases of human cutaneous myiasis caused by *Cuterebra* spp. have been documented in North America. Clinical signs appear about 10−14 days after the bot fly eggs have been deposited on the skin in outdoor situations. The individual's body heat stimulates the eggs to hatch, after which the first instars, after contacting the host, enter the skin through cuts, abrasions, or natural body openings. Larval development usually progresses to the second and, occasionally, the third instar. The danger is that in the meantime the wandering first instars may invade deeper tissues, leading to dire consequences if they should penetrate the cranial cavity, eyes, or vital organs.

The tórsalo, or **human bot fly**, causes cutaneous myiasis in people throughout its range in Central and South America. Lesions generally occur on the exposed extremities but also may involve the scalp, forehead, external ears, eyelids, lower back, and thighs. Skin lesions at the site of larval development are relatively minor during the early stages. As the larva grows, however, the site becomes intensely itchy and exudes serosanguinous fluids. About a week before emergence of the mature larva, the lesion becomes tender, moderately swollen, and generally more painful due to the spines of the larva irritating the skin as it moves or rotates within (Fig. 19.26). Upon emergence of the larva the lesion usually heals spontaneously in about a week. If the larva dies before emergence or the wound becomes infected, serious complications can result. These include foul discharges and fetid odors that may attract other skin-invading parasites (e.g., screwworms), leading to cases of secondary myiasis. Occasionally, *D. hominis* infestations of the head have resulted in penetration of brain tissue, particularly in infants and young children, causing convulsions and death (Rossi and Zucoloto, 1972).

Larvae of *D. hominis* can be extracted by physically squeezing them out or killing them by injection with an anesthetic and surgically removing them. They also can be induced to exit the skin by blocking off their air supply with strips of pork fat ("bacon therapy"), lard, petroleum jelly, soft beeswax, and other materials applied to the lesion. In attempts to reach air, the larvae penetrate the material and are extracted when it is removed.

Clinical Use of Maggots

A few myiasis-causing flies feed only on necrotic tissue as they develop. They are species such as *Lucilia sericata* that are usually associated with carrion. In hospitals the larvae may be deliberately used in the treatment of deep wounds and sores in situations in which surgery is impractical. This approach has been used by people since ancient times for treating deep, chronic, or extensive surface wounds. Anthropological evidence shows that such widely located cultures as the Mayans of Central America, the aboriginals of Australia, and the hill people of Myanmar (formerly North Burma) all used maggots for such **wound therapy**. Later, with the advent of explosive artillery shells, bombs, and grenades in warfare, battlefield injuries became much more difficult to treat using conventional surgery. During the Napoleonic War, American Civil War, and World War I, military physicians observed that wounds inadvertently infested with maggots healed quickly with minimal scarring. This led to the deliberate use of maggots, hatched from surface-sterilized eggs, to remove necrotic tissue in treating chronic wounds, bed sores, severe burns, and bone infections. The procedure was called **maggot therapy** and gained wide use. Maggot therapy was a common procedure in medicine until the general use of antibiotics replaced the practice in the 1940s. Now, following a 50-year hiatus, the clinical use of **surgical maggots** in treating such maladies is being practiced again in cases where antibiotics are ineffective and where surgery is either impractical or is refused by the patient.

Three species of blow flies have been commonly used in treating wounds: *Lucilia illustris*, *L. sericata*, and *Phormia regina,* with *L. sericata* being the species of choice. Care must be taken to assure that maggots from the population selected for colonization feed only on dead tissues. Once the laboratory colony is established, eggs are sterilized in an antiseptic bath (e.g., sodium hypochlorite, formalin, or hydrogen peroxide) and held for hatching. First-instar maggots are transferred to the patient's wound where they are confined with a sterile screen-dressing fastened to the surrounding healthy tissue with adhesive.

The maggots act as microsurgeons, removing necrotic tissue along with debris and associated microorganisms. In moving about the wound while feeding, maggots stimulate the formation of desirable granulation tissue and secrete calcium carbonate, ammonia, and allantoin (a substance that promotes wound healing). Together they produce a more alkaline milieu less conducive to bacterial growth. The maggots develop at a predictable rate and can be removed and counted as they mature. Healing of maggot-treated wounds is very rapid with less scarring than conventional surgery. There is no need to use anesthesia during this process because the active maggots cause little discomfort except for occasional tingling sensations. Another advantage is the much lower cost of the entire procedure even when including the cost of maintaining a fly colony.

VETERINARY IMPORTANCE

Livestock and wildlife are at greater risk of attack by myiasis-causing flies than are humans. This is a result of greater exposure and the tendency to have more untreated open wounds as sites for fly exploitation. The seasonal incidence of myiasis in nonhuman hosts increases during calving, foaling, and lambing when young animals are less resistant in their behavioral and humoral response to maggot invasion. Also, the availability of placental carrion and the exposed umbilicus of newborn animals enhance related fly activity. Open wounds associated with shearing, branding, dehorning, and castrating operations increase the vulnerability of animals to attack. Blow flies also oviposit on fecal- or urine-soiled pelage. The exploitation of scrapes, scratches, and other skin wounds by myiasis-causing carrion flies, flesh flies, and screwworms can have a major economic impact on livestock operations throughout the world, especially in tropical and warm temperate latitudes.

The sheep blow flies, *Lucilia cuprina* (=*Phaenicia pallescens*, *Phaenicia cuprina*) and *Lucilia sericata* (=*Phaenicia sericata*) are very important causes of myiasis in sheep in Australia, South Africa, New Zealand, and Great Britain. Such an infestation is commonly referred to as **blow fly strike**. These flies especially attack Merino and other breeds that have numerous skin folds and crusted wool that retain excrement, causing **fleece rot**. Common sites of attack are the anal and genital regions of sheep. Prophylactic measures include keeping these body regions shorn, or surgically removing skin in these areas to limit fouling, termed **Mule's operation** or **mulesing,** which is a cruel, controversial husbandry practice banned in New Zealand in 2010. *Lucilia sericata* generally invades only fresh carrion at warm latitudes where it is not associated with myiasis. However, in Australia, a race of this species is known to cause secondary facultative myiasis in sheep. This race of fly also exhibits a greater developmental success when infesting live sheep than when infesting dead sheep as carrion. Annual economic losses to blow fly myiasis in Australia and other sheep-raising countries are very substantial. **Pissle strike** is another form of myiasis in Australia in which blow flies infest the urethral orifice of a ram's penis, attracted by the surrounding dense, urine-soaked fleece. Sheep strike in the northern Holarctic Region is most often associated with two important and closely related calliphorids:*Phormia regina* and *Protophormia terraenovae* (Wall and Shearer, 1997).

In terms of gross pathology, mortality, and economic losses, the most important myiasis-causing flies worldwide are the **screwworms**. Untreated screwworm cases nearly always lead to death of the host. The adult flies produced in one infested animal serve as a source for subsequent cases. Fatalities are due to the invasion of vital organs, septicemia resulting from large maggot-infested wounds, or predation of the debilitated host. Before the development and implementation of screwworm control measures, annual losses attributed to screwworms amounted to several hundred million dollars annually in North America alone. Because wild vertebrates also are subject to screwworm attack (Fig. 19.41), they constitute important reservoirs of these parasites for domestic species. Any open wound, no matter how small, is a potential site for oviposition by primary screwworms. The treatment of wounds, whether to prevent or remove screwworms, is a costly procedure in time and labor, especially when it involves livestock on open range.

The **tórsalo** is an important pest of domestic cattle throughout tropical areas of Central and South America. Horses and other equids, however, are seldom bothered. Clusters of furuncles can occur anywhere on the host, but only rarely are they secondarily invaded by other myiasis-causing flies. Even then they only invade after the premature death of the larva inside the furuncle. The most important injury is hide damage, causing significant economic losses to the very important leather industry of South America. Heavily infested cattle may become weak and emaciated and have difficulty walking because of *D. hominis* larvae infesting their legs and feet. Animals can become seriously crippled when wounds become infected, especially when retaining dead larvae. Deaths of heavily infested cattle and dogs have occurred in such cases. Certain cattle breeds (e.g., Brahman, Zebu) appear to be differentially resistant to tórsalo infestations and show lower infestation levels than do British and dairy breeds.

Economic losses to **cattle grubs,** primarily due to hide damage, reduced weight gain, meat trim, and reduced milk

FIGURE 19.41 Massive shoulder infestation of Key deer by screwworms (*Cochliomyia hominivorax*), Big Pine Island, Florida, USA. *Courtesy of United States Department of Agriculture/Animal and Plant Health Inspection Service/Wildlife Services; USDA/APHIS/Wildlife Services.*

production, were estimated to be as high as $100 million annually in North America in the 1980s (Drummond et al., 1988) and have recently been estimated to be as high as $200−$400 million annually. This figure does not include the cost of controlling cattle grubs. A grub burden of less than 50 larvae per host appears to have little effect on the vitality or weight increase of infested cattle. Economic losses are primarily caused in cattle when open warbles and scars of healed warbles along the host's backline decrease the value of hides used in making leather. The green, or raw, hide constitutes about 10% of the total carcass value, and **shot-holed hides** (warble-scarred) can result in considerable reduction in grower profits. Warbles caused by cattle grubs and tórsalo larvae in the backs of slaughtered animals require excessive trimming of discolored flesh and jellied fat surrounding the warbles, further contributing to decreased profits due to labor costs and loss of meat. Horses also are commonly infested by *Hypoderma* first instars, especially *H. bovis*. Grubs in horses, being in an abnormal host, produce abscesses when the larvae make their way to the back and begin to open their breathing holes. Because of the discomfort caused to the backs of infested horses, the use of these animals as saddle mounts is difficult, if not precluded, during certain times of the year.

Gadding by cattle in response to *Hypoderma* females, either in enclosures or on open range, can result in serious injuries and even the death of animals. Such injuries in turn are open to invasion by other myiasis-causing flies. Headlong galloping by panic-stricken cattle can bruise udders and, in the case of pregnant cows, cause spontaneous abortion. From a management perspective, gadding of receptive females during the critical spring breeding season has the effect of greatly increasing the later synchrony of calving. The accidental death of an animal killed in a fall incurred while fleeing from a bot fly attack has been blamed at times on large predators subsequently discovered feeding on the remains. In the Neotropics, because of the egg-porter phenomenon, hosts take no evasive action to avoid adult tórsalo flies.

Nose bots in sheep cause relatively low economic losses compared with cattle grubs. Unless the nose bots are very numerous, the value gained by the costly handling of sheep to administer controls is questionable. At times individual sheep may die as a result of unusually large numbers of maggots (e.g., 100−300 per host), on the order of 10 times the usual host burden. Significant weight gains and increased fleece length have been reported in Russia and South Africa as a result of controlling nose bots in lambs. Organophosphate and macrocyclic lactone insecticides, such as the avermectins, are effective against nose bots.

Horse stomach bot flies are ubiquitous parasites of horses throughout the world. Chronic and repeated infestations can result in loosened teeth due to larval tunneling in the gums and in ulcerations of the gastric lining. Eventually this will interfere with forage ingestion and digestion. However, most horses tolerate a maggot burden of a hundred or so with no apparent ill effects. Depending on the value of the individual animal, whether a race horse or cow pony, a family pet or a "candidate for the glue factory," the economic loss is widely variable. Different breeds of the domestic horse also react differently to egg laying by the hovering adults of the common stomach bot fly. Thoroughbreds, American saddle horses, and Arabians, for example, are much disturbed by the hovering fly and will take evasive action that can cause self-injury. At the other extreme, Shetland ponies, Morgans, and draft breeds appear to ignore the ovipositing flies. All breeds, however, react in wild panic to ovipositing *G. nasalis* females.

Among the horse stomach bot flies, *G. pecorum* is the most pathogenic. Its maggots often attach *en masse* in the oral cavity and upper alimentary tract. This can result in obstruction caused by the enlarging maggots attached to the soft palate and esophagus and the associated inflammation. Heavy populations hinder swallowing, prevent food passage, and can cause severe digestive problems in the host, sometimes with fatal results.

The possible use of cuterebrine bots as biocontrol agents of rodents has been suggested. However, the associated pathology in native hosts is minimal, and bot populations appear to have little effect on rodent numbers.

PREVENTION AND CONTROL

There are three major approaches for controlling myiasis: avoiding contact between potential hosts and myiasis-causing flies; early treatment of wounds to prevent myiasis; and reduction or elimination of myiasis-fly populations. In human health, there is general reliance on hygiene and medical or surgical intervention. In veterinary cases the most common approach is the use of insecticides, especially systemic compounds that target the parasitic larvae, administered to the host.

Preventing unnecessary or avoidable outdoor exposure of humans during fly seasons is one obvious way to control facultative or obligatory myiasis. Attractive odors associated with urine, feces, vomitus, nasal secretions, and purulent sores invite fly strike. Healthy adults are able to react to flies attempting to feed or oviposit, but infants and elderly individuals often are either unaware or unable to take evasive measures. Myiasis-causing flies sometimes enter buildings when attracted by similar odors. Management of geriatric care facilities should routinely include measures such as screening, diligent resident hygiene, and treatment of sores to avoid attraction and contact with flies. Securing foods in fly-proof containers, under screens, or under refrigeration is the best means of preventing gastrointestinal myiasis. Foods that have been exposed, even

when fly eggs are present, can be made safe by freezing or thorough cooking.

The risk of human myiasis is greater in livestock areas. People working or residing close to livestock operations (e.g., stables, dairies, feedlots, and poultry houses) are most at risk. Flies or bee-like insects hovering around a person or alighting at open sores should be suspect. Immediate evasive responses usually will drive the fly away. Where the tórsalo fly occurs in the Neotropics, insect repellents can be used to reduce the attacks of blood-seeking insects carrying tórsalo eggs.

Human myiasis can be prevented by early treatment of open wounds with an antiseptic salve and protective dressing. Wound dressings should be changed regularly and not be allowed to become excessively soiled by wound exudates. *Dermatobia* larvae in furuncles can be induced to back at least part way out of the warble by covering the pore with a thick smear of petroleum jelly. The protruding maggot then can be grasped with forceps or fingers and slowly extracted. Sometimes surgical removal is necessary; this entails snipping the rim of the warble pore to allow greater ease of extraction. Maggots in eyes, nasal sinuses, or the auditory meatus should be removed by flushing with saline or a diluted antiseptic such as carbolic acid. Gastrointestinal myiasis can be treated with purges or emetics, although several treatments may be necessary to remove all maggots.

Preventive measures for the control of myiasis in livestock, zoo animals, and wildlife mainly involve the removal of carrion that may attract flies, early treatment of open wounds to prevent fly strike, and minimizing injuries during the peak fly season. Husbandry practices that can help to reduce the incidence of myiasis include regular inspection of animals to detect and treat all wounds; deep burial (ca. 1.5 m) or cremation of carrion; limiting of dehorning, castrating, and branding to non-fly seasons; removal of sharp objects and other materials than can cause wounds (e.g., horns, barbwire, thorny shrubs); and effective control of biting flies and ticks. The application of salvelike ointments or "smears" can help to prevent fly strike or kill maggots that are already present. Smears contain larvicides such as benzol or Lindale and repellents such as pine-tar oil. Hydrogen peroxide has been infused directly into *Hypoderma* spp. warbles to antiseptically remove the larvae and avoid the consequences of crushing the larvae inside the warble, which can produce an anaphylactic response.

For many years **dipping vats** have been used throughout the world to treat livestock for arthropod pests, including cattle grubs, screwworms, and other myiasis-causing flies. This entails forcing animals to swim or otherwise move through a water-filled pit containing insecticides or acaricides. This approach, however, has been largely replaced by other methods such as insecticide-impregnated **ear tags**. The tags slowly release low levels of chemicals that afford protection for extended periods. Another slow-release device developed for cattle grub control was the insecticide-impregnated **ankle band** attached around the back legs of cattle, which proved to be difficult to prevent soiling, thus quickly losing efficacy. Systemic insecticides such as chlorinated hydrocarbons, organophosphates, carbamates, and sulfur compounds kill cattle grubs before they reach the back of the host. These materials have been almost entirely replaced by the macrocyclic lactones, including the avermectins and milbemycins. Systemic materials can be administered by **injection**, applied to the back as **pour-ons** to be absorbed by the bloodstream, as **boluses** placed in the animal's stomach to provide slow release of systemic compounds, or in **mineral blocks** (salt licks) for ad libitum consumption by livestock and wildlife species. Whereas systemic organophosphates are effective only against pre-warble larvae, macrocyclic lactones are effective against all larval instars.

Other methods used to control myiasis-causing flies that attack livestock are **low-volume sprays**, **oral drenches**, **power dusters**, and self-treatment devices such as suspended **dust bags** and **back rubbers**. In the case of "fleece worm" blow flies, the topical application of insecticides including organophosphates, synthetic pyrethroids, insect growth regulators, and the tetracyclic-macrolide compound spinosad to body areas subject to soiling with maggot secretions is an effective, albeit labor intensive, practice. Typically these applications are made using a high-pressure spray, called **jetting**. The use of **biocontrol agents**, such as parasitic pteromalid wasps that attack fly pupae and the physical removal of adult flies by **trapping**, can also play a role in integrated pest management programs for some muscoid species that cause myiasis.

The use of **genetically altered flies** has met with some success in reducing local populations of certain myiasis-causing calliphorid species. This approach shows promise in Australia where *Lucilia cuprina* (=*Phaenicia cuprina*) causes annual losses estimated at $280 million (Australian) due to secondary myiasis in sheep. Radiation treatment has been used to induce genetic translocations in chromosomes of a laboratory strain of *L. cuprina* that makes mutant male flies impotent. The fewer daughters that are produced from mutant-male and wild-female matings are able to reproduce normally. However, the second-generation daughters carry about 85% of the induced mutations. The result has been a reduction of *L. cuprina* populations by about half in the first generation and by increased proportions in subsequent generations.

Screwworm Eradication Program

The idea of using **sterilized males** to eradicate populations of wild flies was first conceived by E. F. Knipling in the 1930s. It was not until 1954–1955, however, that the first

successful field test was achieved with the eradication of the primary screwworm *Cochliomyia hominivorax* on the island of Curação (Netherlands Antilles). This entailed the mass production of sterile males from gamma-irradiated pupae, which were then released to mate with wild females. Eggs resulting from these matings do not hatch, thereby reducing fly populations. Subsequent successes in eradicating *C. hominivorax* were achieved in the southeastern United States in 1959, the rest of the United States by 1966, and in Puerto Rico in 1975. Following the establishment of the Joint Mexico–United States Commission on Screwworm Eradication in 1972, a cooperative program was begun in 1975 to eliminate *C. hominivorax* in Mexico as far south as the Isthmus of Tehuantepec. Hundreds of millions of flies were mass-reared weekly in facilities at Mission, Texas (USA), and Tuxtla Gutierrez, Chiapas (Mexico), for aerial releases in the targeted areas. By 1985 screwworms had been successfully eliminated north of the Isthmus of Tehuantepec in Mexico, advancing to the Guatemalan border by 1991, and the rest of Central America by 2001 with flies produced at the Tuxtla Gutierrez plant. For example, *C. hominivorax* was detected for the first time in Libya in 1988 where it was eradicated within a few months following the release of sterile males produced in this facility. A sterile male buffer zone has been successfully maintained at the Darien Gap, the isthmus at the border between Panama and Colombia by the Screwworm Barrier Maintenance Program in Panama (COPEG), managed by the United States and Panamanian governments. COPEG produces millions of flies in its Panama laboratory and production facility; sterilizes these flies using cobalt; and releases them by airplane over a barrier zone running through Eastern Panama and neighboring areas of Colombia. Additionally, sterile males produced in Panama are being successfully used in areas where *C. hominivorax* has reinvaded former eradication zones or where it has been introduced for the first time. In October 2016, *C. hominivorax* was detected in Key deer from the National Key Deer Refuge in Big Pine Key, Florida (Fig. 19.41). Approximately 3 million sterile flies were released twice a week in the affected area of the Florida Keys. In principal, as the population of sterile screwworm flies increases, the population of fertile screwworm flies decreases until the population dies out, which normally takes 4–6 months to complete in small areas. The Key deer infestation was deemed officially eradicated as of March 2017. For further details on the history of the screwworm eradication program, see Meyer (1996) and Wyss (2000).

Another approach to screwworm control has been the development of an attractant called **Swormlure** that simulates the odor of animal wounds (Snow et al., 1982; Cunningham et al., 1992). This attractant, combined as a bait with pesticides in a pelletized form, can be applied by aircraft to attract and kill gravid females. Called the Screwworm Adult Suppression System (SWASS), this approach has been successfully used as a complement to sterile-male release efforts by reducing the wild fly populations before the release of sterilized flies. Swormlure and other blow fly volatile attractants have also been used as the basis for the development of an attractant for the old screwworm fly *Chrysomya bezziana*. Bezzilure B has been proven to be effective as a bait in sticky traps with certain promising success (Sulston et al., 2014).

For oestrid species, the use of physical and chemical attractants in development of traps, focused on adult monitoring and control, has received some attention. *Hypoderma tarandi*, *Cephenemyia trompe*, and other *Cephenemyia* spp.have been reported to be attracted to CO_2-releasing devices. Lack of knowledge of the olfactory physiology of oestrids, together with a generally low population density of adults, complicates experimentation with baited traps.

Cattle Grub Control

The best approach to areawide control of cattle grubs is integrated management programs enlisting the cooperation of all producers in the targeted region. The most effective method to date is the use of **endectocides** (e.g., macrocyclic lactones), systemic compounds that kill both internal and external parasites. Unlike traditional systemic insecticides that kill only the migrating larvae of cattle grubs, macrocyclic lactones are also effective in killing second and third instars after they have formed warbles. The other characteristic of these compounds, especially the avermectins, is their high efficacy against migrating *Hypoderma* first instars even at dramatically low dosage levels (Drummond, 1985). This high efficacy of microdoses of avermectins has been used with success in some of the European eradication programs (Boulard, 2002). However, because these compounds are effective against both internal and external parasites, there are concerns that reducing the dosage for one target could lead to reduced efficacy, or even resistance, with other target species (Leaning, 1984).

Sterile-male release technology also has shown promise but is limited by inherent logistic problems in the mass rearing of *Hypoderma* spp. for this purpose. By combining the use of systemic compounds and sterile-male releases, cattle grub populations and the associated economic losses were dramatically reduced in the area covered by the joint United States–Canada Cattle Grub Project initiated in 1982 (Kunz et al., 1984; Scholl et al., 1986) and evaluated by Klein et al. (1990) and Kunz et al. (1990). Likewise, significant reductions in cattle grub problems have been achieved in Great Britain and several European countries (Boulard et al., 1984; Wilson, 1986) using combinations of strict compulsory treatment, movement control, serological monitoring, and, in some countries, microdoses of avermectins. Although experimental vaccines against

Hypoderma spp. have been developed, they have not been widely field tested. They may, however, play a greater role in the future as an important component of integrated management programs, especially in the face of increasing instances of the loss of chemical control of livestock nematodes resulting from drug resistance. For further information on the biology and control of cattle grubs, see Scholl (1993), Colwell (2001), and Colwell et al. (2006).

Myiasis and Molecular Advances

Molecular advances in traumatic (wound) myiases were reviewed recently by Hall et al. (2016). New molecular studies have so far been focused on taxonomy, phylogenetics, species identification, and epidemiology. The mitochondrial DNA *COX1* (cytochrome oxidase 1) and the nuclear 28S (rDNA) gene sequence have been used to study the population variation in some species of myiasis and forensic interest. Molecular techniques are also valuable to test the compatibility of fly strains for the sterile fly control, and transgenic insect techniques may be applied to improve the breeding performance of sterilized males in the field. Another significant advance provided by molecular studies is understanding the development of organophosphate resistance in blow fly species. The *αE7* gene encoding esterase 3 enzyme in the blow fly *L. cuprina* was identified with two mutations providing resistance to these insecticide chemicals.

REFERENCES AND FURTHER READING

Baird, C. R., Podgore, J. K., & Sabrosky, C. W. (1982). *Cuterebra* myiasis in humans: Six new case reports from the United States with a summary of known cases (Diptera: Cuterebridae). *Journal of Medical Entomology, 19*, 263–267.

Baird, J. K., Baird, C. R., & Sabrosky, C. W. (1989). North American cuterebrid myiasis. *Journal of the AmericanAcademy of Dermatology, 21*, 763–772.

Baumgartner, D. L. (1988). Review of myiasis (Insecta: Diptera: Calliphoridae, Sarcophagidae) of Nearctic wildlife. *Wildlife Rehabilitation, 7*, 3–46.

Boulard, C. (2002). Durably controlling hypodermosis. *Veterinary Research, 33*, 455–464.

Boulard, C., & Thornberry, H. (Eds.). (1984). *Warble fly control in Europe*. Rotterdam/Boston: Balkema.

Brewer, T. F., Wilson, M. E., Gonzalez, E., & Felsenstein, D. (1993). Bacon therapy and furuncular myiasis. *Journal of the American Medical Association, 270*, 2087–2088.

Broce, A. B. (1985). Myiasis-producing flies. In R. E. Williams, R. D. Hall, A. B. Broce, & P. J. Scholl (Eds.), *Livestock entomology* (pp. 83–100). New York: John Wiley.

Carvalho, C. J. B., & Ribeiro, P. B. (2000). Chave de identificação das espécies de Calliphoridae (Diptera) do Sul do Brasil. *Brazilian Journal of Veterinary Parasitology, 9*, 169–173 (in Portuguese).

Catts, E. P. (1982). Biology of New World bot flies: Cuterebridae. *Annual Review of Entomology, 27*, 313–338.

Catts, E. P. (1994). Sex and the bachelor bot fly. *American Entomologist, 40*, 153–160.

Cepeda-Palacios, R., Angulo Valadez, C. E., Scholl, P. J., Ramirez-Orduna, R., Jacquiet, P., & Dorchies, P. (2011). Ecobiology of the sheep nose bot fly (*Oestrus ovis* L.): A review. *Revue de Medicine Veterinaire, 162*, 503–507.

Cepeda-Palacios, R., & Scholl, P. J. (1999). Gonotrophic development in *Oestrus ovis* (Diptera: Oestridae). *Journal of Medical Entomology, 36*, 435–440.

Cepeda-Palacios, R., & Scholl, P. J. (2000). Intra-puparial development in *Oestrus ovis* (Diptera: Oestridae). *Journal of Medical Entomology, 37*, 239–245.

Chernin, E. (1986). Surgical maggots. *Southern Medical Journal, 79*, 1143.

Clarke, K. J. (2013). Myiasis (fly disease) and insectal disease generally are causing mental illness. *Medical Hypotheses, 81*, 360–365.

Colwell, D. D. (2001). Bot flies and warble flies (Order Diptera: Family Oestridae). In W. M. Samual, M. Pybus, & A. Kocan (Eds.), *Parasitic diseases of wild mammals* (2nd ed., pp. 46–71). Ames, Iowa: Iowa StateUniversity Press.

Colwell, D. D., Hall, M. J. R., & Scholl, P. J. (2006). *The oestrid flies: Biology, host–parasite relationships, impact and management*. Wallingford, UK: CABI Publishing.

Craig, C. F., & Faust, E. C. (1940). *Clinical parasitology* (2nd ed.). Philadelphia: Lea & Febiger.

Cunningham, E. P., Abusowa, M., Lindquist, D. A., Sidahmed, A. E., & Vargas-Teran, M. (1992). Screwworm eradication programme in North Africa. *Revue d'Elevage et de Médicine Vétérinaire des Pays Tropicaux, 45*, 115–118.

Curran, C. H. (1965). *The families and genera of North American Diptera* (2nd ed.). Woodhaven, NY: Tripp.

Cuyler, C., White, R. R., Lewis, K., Soulliere, C., Gunn, A., Russell, D. E., et al. (2010). Are warbles and bots related to reproductive status in West Greenland caribou?. In *The 13th North American caribou workshop. Winnipeg, Manitoba, Canada*.

Dear, J. P. (1985). A revision of the New World Chrysomyini (Diptera: Calliphoridae). *Revista of Brazilian Zoology, 3*, 109–169.

Drummond, R. O. (1985). Effectiveness of ivermectin for control of arthropod pests of livestock. *Southwest Entomologist Supplement, 7*, 34–42.

Drummond, R. O., George, J. E., & Kunz, S. E. (1988). *Control of arthropod pests of livestock: A review of technology*. Boca Raton, FL: CRC Press.

El-Badry, A. A., Salem, H. K., & El-Aziz Edmardash, Y. A. (2014). Human urinary myiasis due to larvae of *Clogmia* (*Telmatoscopus*) *albipunctata* Williston (Diptera: Psychodidae) first report in Egypt. *Journal of Vector Borne Diseases, 51*, 247.

Ferrar, P. (1987). A guide to the breeding habits and immature stages of Diptera Cyclorrhapha. In *Entomography* (Vol. 8, pp. 83–98). Leiden: Brill.

Furman, D. P., & Catts, E. P. (1982). *Manual of medical Entomology* (4th ed.). Cambridge, UK: Cambridge University Press.

Graham, O. H. (1985). *Symposium on eradication of the screwworm from the United States and Mexico*. Miscellaneous Publication No. 62. College Park, MD: Entomological Society of America.

Greenberg, B. (1971). *Flies and disease. Vol. 1. Ecology, classification and biotic associations*. Princeton, NJ: Princeton University Press.

Greenberg, B., & Singh, D. (1995). Species identification of calliphorid (Diptera) eggs. *Journal of Medical Entomology, 32*, 21–26.

Guimarães, J. H., & Papavero, N. (1999). *Myiasis in man and animals in the neotropical region; bibliographic database*. São Paulo, Brazil: Pleiade/FAPESP.

Hall, D. G. (1948). *The blow flies of North America*. College Park, MD: Thomas Say Foundation, Entomological Society of America.

Hall, M. J. R., & Wall, R. (1995). Myiasis of humans and domestic animals. *Advances in Parasitology, 35*, 257−334.

Hall, M. J. R., Wall, R., & Stevens, J. R. (2016). Traumatic myiasis: A neglected disease in a changing world. *Annual Review of Entomology, 61*, 159−176.

Hendrix, C. M., King-Jackson, D. A., Wilson, M., Blagburn, B. L., & Lindsay, D. S. (1995). Furunculoid myiasis in a dog caused by *Cordylobia anthropophaga*. *Journal of the American Veterinary Medical Association, 207*, 1187−1189.

James, M. T. (1947). *The flies that cause myiasis in man*. U.S. Department of Agriculture, Miscellaneous Publication 631. Washington, DC: U.S. Government Printing Office.

Kettle, D. S. (1995). *Medical &veterinary entomology*. Oxford, UK: Oxford University Press.

Klein, K. K., Fleming, C. S., Colwell, D. D., & Scholl, P. J. (1990). Economic analysis of an integrated approach to cattle grub (*Hypoderma* spp.) control. *Canadian Journal of Agricultural Economics, 38*, 159−173.

Kunz, S. E., Drummond, R. O., & Weintraub, J. (1984). A pilot test to study the use of the sterile insect technique for eradication of cattle grubs. *Preventive Veterinary Medicine, 2*, 523−527.

Kunz, S. E., Scholl, P. J., Colwell, D. D., & Weintraub, J. (1990). Use of the sterile insect technique for control of cattle grubs (*Hypoderma bovis* and *Hypoderma lineatum*) (Diptera: Oestridae). *Journal of Medical Entomology, 27*, 523−529.

Lane, R. P., & Crosskey, R. W. (1993). *Medical insects and arachnids*. London: Chapman & Hall.

Leaning, W. H. D. (1984). Ivermectin as an antiparasitic agent in cattle. *Modern Veterinary Practice, 65*, 669−672.

Leclercq, M. (1969). *Entomological parasitology-the relation between entomology and the medical sciences* (Chapter 5, Myiases). Oxford: Permagon.

Liu, D., & Greenberg, B. (1989). Immature stages of some flies of forensic importance. *Annals of the Entomological Society of America, 82*, 80−93.

McAlpine, J. F., et al. (Eds.). (1981, 1987, 1989). *Manual of Nearctic diptera* (Vols. 1−3). Quebec: Research Branch Agriculture Canada, Canadian Government Printing Center. Monographs 27, 28, and 32, respectively.

Meyer, N. V. (1996). *History of the Mexico-United States screwworm eradication program*. New York: Vantage Press.

Michener, G. R. (1993). Lethal myiasis of Richardson's ground squirrels by the sarcophagid fly *Neobellieria citellivora*. *Journal of Mammology, 74*, 148−155.

Milton, K. (1996). Effects of bot fly (*Alouttamia baeri*) parasitism on a free-ranging howler monkey (*Aloutta palliata*) population in Panama. *Journal of Zoology, London, 239*, 39−63.

Norris, K. R. (1965). The bionomics of blow flies. *Annual Review of Entomology, 10*, 47−68.

OISTROS. *An irregularly published newsletter dealing exclusively with current research on the Oestroidea*. Stockholm, Sweden: SwedishMuseum of Natural History.

Panadero-Fontán, R., & Otranto, D. (2015). Arthropods affecting the human eye. *Veterinary Parasitology, 08*, 84−93.

Papavero, N. (1977). *The world Oestridae (diptera), mammals and continental drift*. The Hague: Junk.

Pape, T. (2001). Phylogeny of Oestridae (Insecta: Diptera). *Systematic Entomology, 26*, 133−171.

Pape, T. (2006). Phylogeny and evolution of bot flies (Chapter 3). In D. D. Colwell, M. J. R. Hall, & P. J. Scholl (Eds.), *The oestrid flies: Biology, host−parasite relationships, impact and management*. Wallingford, UK: CABI Publishing.

Pape, T., Piwczynski, M., Wyborska, D., Akbarzadeh, K., & Szpila, K. (2016). A new genus and species of hypodermatine bot flies (Diptera: Oestridae). *Systematic Entomology*. https://doi.org/10.1111/syen.12220.

Pezzi, M., Cultrera, R., Chicca, M., & Leis, M. (2015). Furuncular myiasis caused by *Cordylobia rodhaini* (Diptera: Calliphoridae): A case report and a literature review. *Journal of Medical Entomology, 52*, 151−155.

Reames, M. K., Christensen, C., & Luce, E. A. (1988). The use of maggots in wound debridement. *Annals of Plastic Surgery, 21*, 388.

Ribeiro, P. B. (2007). Miíase. In S. Riet-Correa, Lemos, & Borges (Eds.), *Doenças de Ruminates e Equideos* (Vol. 1, pp. 551−564). Santa Maria: Pallotti (in Portuguese).

Ribeiro, P. B., & Carvalho, C. J. B. (1998). Pictorial key to Calliphoridae genera (Diptera) in southern Brazil. *Brazilian Journal of Veterinary Parasitology, 7*, 137−140.

Rosen, I. J., & Neuberger, N. (1977). Myiasis *Dermatobia hominis*, Linn: Report of a case and review of literature. *Cutis, 19*, 63−66.

Rossi, M. A., & Zucoloto, S. (1972). Fatal cerebral myiasis caused by the tropical warble fly, *Dermatobia hominis*. *The American Journal of Tropical Medicine and Hygiene, 22*, 267−269.

Sabrosky, C. W. (1986). *North American species of* Cuterebra*, the rabbit and rodent bot flies (Diptera: Cuterebridae)*. College Park, MD: Thomas Say Publications, Entomological Society of America.

Sabrosky, C. W., Bennett, G. F., & Whitworth, T. L. (1990). *Bird blowflies (*Protocalliphora*) in North America (Diptera: Calliphoridae)*. New York: Random House (Smithsonian Institution Press).

Sancho, E. (1988). *Dermatobia*, the Neotropical warble fly. *Parasitology Today, 4*, 242−246.

Sandeman, R. M., Bowles, V. M., & Colwell, D. D. (2014). The immunobiology of myiasis infections − whatever happened to vaccination? *Parasite Immunology, 36*, 605−615.

Scholl, P. J. (1993). Biology and control of cattle grubs. *Annual Review of Entomology, 39*, 53−70.

Scholl, P. J., Colwell, D. D., Weintraub, J., & Kunz, S. E. (1986). Area-wide systemic insecticide treatment for control of cattle grubs, *Hypoderma* spp. (Diptera: Oestridae): Two approaches. *Journal of Economic Entomology, 79*, 1558−1563.

Scholl, P. J., & Weintraub, J. (1988). Gonotrophic development in *Hypoderma lineatum* (Villers) and *H. bovis* (L.), with notes on reproductive capacity. *Annals of the Entomological Society of America, 81*, 315−324.

Scott, H. G. (1963). *Myiasis: Epidemiologic data on human cases (North America north of Mexico: 1952-1962 inclusive)*. Atlanta, GA: U.S. Department of Health, Education and Welfare, Centers for Disease Control and Prevention.

Sherman, R. A., & Pechter, E. A. (1988). Maggot therapy: A review of the therapeutic applications of fly larvae in human medicine, especially

for treating osteomyelitis. *Medical and Veterinary Entomology, 2*, 225–230.

Shewell, G. E. (1950). A new species reared from the Columbian ground squirrel (Diptera: Sarcophagidae. *The Canadian Entomologist, 82*, 245–246.

Slansky, F. (2007). Insect/mammal associations: Effects of cuterebrid bot fly parasites on their hosts. *Annual Review of Entomology, 52*, 17–36.

Slansky, F., & Kenyon, L. R. (2003). *Cuterebra* bot fly infestation of rodents and lagomorphs. *Journal of Wildlife Rehabilitation, 26*, 7–16.

Smith, K. G. V. (1986). *A manual of forensic entomology*. London: British Museum (Natural History).

Snow, J. W., Siebenaler, A. J., & Newell, F. G. (1981). *Annotated bibliography of the screwworm,* Cochliomyia hominivorax *(Coquerel)*. U.S. Department of Agriculture. ARM-S-14. Agricultural Reviews and Manuals, Southern Series No. 14.

Snow, J. W., Coppedge, J. R., Broce, A. B., Goodenough, J. L., & Brown, H. E. (1982). Swormlure: Development and use in detection and suppression systems for adult screwworm (Diptera: Calliphoridae). *Bulletin of the Entomological Society of America, 28*, 277–284.

Stehr, F. W. (1991). *Immature insects* (Vol. 2). Dubuque, IA: Kendall/Hunt.

Stone, A., Sabrosky, C. W., Wirth, W. W., Foote, R. H., & Coulson, J. R. (1965). *A catalogue of Diptera of America north of Mexico*. Agricultural Handbook 276. Washington, DC: US Department of Agriculture.

Sulston, E. C. J., Wardhana, A. H., Hall, M. J. R., Logana, J. G., Gezand, S. A., & Camerona, M. M. (2014). Combining cattle and wound-derived synthetic attractants, POC and Bezzilure B, for sampling *Chrysomya bezziana* in Indonesia. *Acta Tropica, 138*, S69–S75.

Vanin, S., Mazzariol, S., Menandro, M. L., Lafisca, A., & Turchetto, M. (2013). Myiasis by *Megaselia scalaris* (Diptera: Phoridae) in a python affected by pulmonitis. *Journal of Medical Entomology, 50*, 209–211.

Wall, R., French, N. P., & Morgan, K. L. (1995). Population suppression for control of the blowfly *Lucilia sericata* and sheep blowfly strike. *Ecological Entomology, 20*, 91–97.

Wall, R., & Shearer, D. (1997). *Veterinary entomology* (Chapter 5, Myiasis). London: Chapman and Hall.

Whitworth, T. (2006). Keys to the genera and species of blow flies (Diptera: Calliphoridae) of America north of Mexico. *Proceedings of the Entomological Society of Washington, 108*, 689–725.

Wilson, G. W. C. (1986). Control of warble fly in Great Britain and the European community. *The Veterinary Record, 118*, 653–656.

Wood, D. M. (1987). Oestridae. Monograph #28. In J. F. McAlpine (Ed.), *Nearctic Diptera–Vol. II* (pp. 1147–1158). Quebec: Research Branch Agriculture Canada, Canadian Government Printing Center.

Wyss, J. H. (2000). Screwworm eradication in the Americas. *Annals of the New York Academy of Science, 916*, 86–93.

Zumpt, F. (1965). *Myiasis in man and animals in the Old World*. London: Butterworth's.

This page intentionally left blank

Chapter 20

Louse Flies, Keds, and Bat Flies (Hippoboscoidea)

Will K. Reeves[1] and John E. Lloyd[2,†]

[1]U.S. Department of Agriculture, Animal and Plant Health Inspection Service, Biotechnology Resource Services, Western Compliance Assurance Branch, Fort Collins, CO, United States; [2]Department of Renewable Resources, University of Wyoming, Laramie, WY, United States

Members of the families Hippoboscidae, Streblidae, and Nycteribiidae are obligate, blood-feeding ectoparasites and, along with the Glossinidae, constitute the superfamily Hippoboscoidea. The Hippoboscoidea, excepting Glossinidae, are unique among the major families of obligate blood-feeding insects because they do not regularly feed on humans and are not known to transmit any agents that cause major diseases in humans. As a group they are primarily of interest as ectoparasites of domestic animals and wildlife, especially birds and bats.

The Hippoboscidae are variously called louse flies, bird flies, feather flies, spider flies, flat flies, tick flies, ked flies, and keds. The family probably originated in the Cenozoic along with some of their hosts (Lukashevich and Mostovski, 2003). Most species in this family are restricted to a narrow range of hosts. Approximately three-fourths of the known species are ectoparasites of birds, whereas the remainder occur on a variety of mammals other than bats. No members of the Hippoboscidae, Streblidae, and Nycteribiidae known to feed on cold-blooded vertebrates, such as reptiles or amphibians but some Glossinidae do. Members of the Streblidae, called the streblid bat flies or bat flies, and Nycteribiidae, called the nycteribiid bat flies, spider-like bat flies, or bat flies, are exclusively ectoparasites of bats, both Megachiroptera and Microchiroptera, and are encountered by individuals working with bats. These flies are rarely reported to bother or bite people even when bats infest attics or human made structures. For further information on the Hippoboscoidea, the reader is referred to the monographs and other works by Bequaert (1942, 1953-1957), Maa (1963, 1966, 1969, 1971), Maa and Peterson (1987), Theodor and Oldroyd (1964), Theodor 1967, Wenzel (1987), Wenzel and Peterson (1987), and Dick and Patterson (2006).

Although nearly worldwide in distribution, most species of Hippoboscidae are tropical and subtropical in both the Old and New Worlds. The Paleotropics is richer in hippoboscids than any other region. Some hippoboscids can be temporary summer residents of temperate regions due to the migratory habits of their hosts. A few species (e.g., the "grouse fly" *Ornithomya fringillina*) are restricted to temperate regions. There are few modern molecular taxonomic studies to confirm the species status of species with wide geographic ranges.

The bat flies have tracked the distribution and diversity of their hosts and most bat species are tropical. Members of the Streblidae are largely New World and tropical or subtropical in distribution. Relatively few species occur exclusively in temperate zones. Most members of the Nycteribiidae are found in the Oriental Region of the Old World where they might have originated and occur primarily in the tropical and subtropical regions.

TAXONOMY

Almost all collections of Hippoboscoidea must be made directly from host animals or near their nests (Fig. 20.1). Occasionally Hippoboscidae will swarm or can be collected in flight intercept traps. However, these collections are relatively rare and do not give a good representation of hippoboscoid diversity or present information on host associations. Some Hippoboscoidea will remain on dead hosts, such as animals killed by cars, for extended periods of time (Nelder and Reeves, 2005), but others will fly or

† Deceased.

Medical and Veterinary Entomology. https://doi.org/10.1016/B978-0-12-814043-7.00020-0

FIGURE 20.1 Bat being examined for batflies and other ectoparasites. *Photograph by Amanda D. Loftis.*

crawl away fairly soon. Species such as dog or sheep keds can be removed from domestic animals by hand. Collections of bat flies almost always involve mist netting or trapping bats and directly examining the bats for active flies. When collecting bat flies it is often a good practice to keep individual bats isolated in bags or held separately to avoid cross-contamination from one species or host to another (e.g., Martínez et al., 2016). Most Hippoboscoidea are delicate and will shrivel if stored dry, and many bat flies require slide mounting for identification.

As previously mentioned, the family level taxonomy of the Hippoboscoidea is open for interpretation and is contentious. Regardless of the status of the individual families, all are Order Diptera, Cyclorrhapha. Some authors include the Glossinidae in the Hippoboscoidea. Because of similarities in mechanisms of feeding and reproduction as well as specialized morphological characters, the Hippoboscidae, Streblidae, and Nycteribiidae were considered by Theodor (1964) to be monophyletic and were included in the group Pupipara. A molecular study by Peterson et al. (2007) would support the view of Theodor. The name Pupipara, however, is inappropriate for the group because the third-instar larva, not the pupa, is deposited by the female. These insects are therefore larviparous and not pupiparous. We consider Hippoboscidae, Streblidae, and Nycteribiidae as separate related families but acknowledge that the family-level status of the names is contentious.

The use of morphology to determine phylogenetic relationships within the Hippoboscoidea has been problematic, because of the loss of morphological structures to the parasitic way of life. According to Yeates et al. (2007) molecular analysis of the higher-level relationships of Hippoboscoidea support the monophyly of the Hippoboscoidea (including Glossinidae, Hippoboscidae, Streblidae, and Nycteribiidae), a sister-group relationship between the Hippoboscoidea and the remaining Calyptrata, and a sister-group relationship between Glossinidae and the remaining three families. Based on morphology, Streblidae and Nycteribiidae would appear to have common structural characters and a common origin, though Dittmar et al. (2006), using molecular techniques, indicated a strong possible paraphyly of the Streblidae and presented phylogenies that support the nycteribiids and some streblids as either a single group or that the Streblidae are paraphyletic. Further molecular studies should provide valuable insight into the phylogeny of these insects.

There are approximately 19-21 genera and 150-200 described species in the Family Hippoboscidae. Much of the taxonomic work on the Hippoboscidae is up to 50 years old and probably in need of modern revisionary consideration including molecular taxonomy. Thirteen genera containing 31 species and two subspecies have been reported from the Nearctic region (Maa and Peterson, 1987) with up to 40 species expected in the Neotropical region. Several of the species with a Holarctic distribution were introduced to the Nearctic from the Palearctic by humans. Pfadt and Roberts (1978) have presented a list of the louse flies recorded from the United States with their hosts and distribution, both in the United States and worldwide. Maa (1966, 1969) divided the Hippoboscidae into three subfamilies. The Ornithomyinae includes most of the hippoboscid species, with all but seven of them being parasitic on birds. The Lipopteninae contains approximately 34 species, all parasitic on mammals. The Hippoboscinae contains eight species of which seven infest mammals and one infests ostriches.

Overall there are more than 520 species of bat flies worldwide (Hurka and Soos 1986; Maa, 1989). Dick and Patterson (2006) recognize 25 genera and 156 described species of Streblidae in the New World and six genera and 71 described species in the Old World. It is interesting to note that no taxon — either genus or species — of streblid is represented in both the Eastern and Western Hemispheres. Dick and Patterson (2006) also recognize 12 genera and 275 described species of Nycteribiidae worldwide. While no species of nycteribiids are found in both the Eastern and Western Hemisphere, several genera are cosmopolitan in distribution.

MORPHOLOGY

All members of the Hippoboscoidea are morphologically adapted for an ectoparasitic existence among the hairs or feathers of their hosts. Certain parts of the exoskeleton have become modified, mainly by fusion and reduction or atrophy, in response to permanent ectoparasitism.

Hippoboscidae

Adults of this family (Fig. 20.2) vary in size from 1.5 to 12.0 mm. The body is dorsoventrally flattened, with a depressed head, thorax, and abdomen giving these insects their louselike appearance. The mouthparts are directed forward rather than downward. The abdominal integument is soft and flexible, allowing for stretching and distension of the abdomen while feeding and during larval development within the female.

The legs of hippoboscids are generally robust with enlarged femora, flattened tibiae, and short, compact tarsi with one or more basal teeth. The legs tend to be shorter and stouter with heavier tarsal claws in species that infest mammals than in those that infest birds. According to Bequaert (1953), hippoboscids that infest birds have legs that are adapted for scurrying swiftly forward, backward, and sideways amid the soft feathers. In species parasitizing mammals, the legs are adapted more for grasping and clinging to the skin and the coarse hairs of the pelt.

The compound eyes are generally well developed in those genera with functional wings. The eyes are greatly reduced in genera with small, nonfunctional wings and those that lose their wings after reaching the host. The sheep ked, *Melophagus ovinus*, which spends its entire life in the wool of sheep, has small compound eyes with relatively few ommatidia. Ocelli are present in several genera of Hippoboscidae but absent in others. The antennae are small and immovable and are located in deep antennal sockets.

The Hippoboscidae are vessel feeders (solenophages) with both sexes being obligate blood-feeders. The proboscis is strongly sclerotized. Its base is partially retracted into a pouch on the ventral side of the head when not in use. At rest, the distal portion is concealed in the palpal sheath. The structure of the proboscis resembles that of blood-sucking muscid flies. The labium is the principal piercing structure. The labrum and hypopharynx lie in a dorsal groove of the labium and together form the food channel. The labella at the tip of the labium are armed with teeth (Fig. 20.3). Female *Ascodipteron* (Strebliidae) are essentially endoparasites and are the exception to this trend.

Adults of most species of Hippoboscidae have relatively long, broad forewings; however, some break them off after locating a host. The hind pair of wings is represented by the halteres, a characteristic of dipterans. At rest, the forewings lie flat over each other on the abdomen like closed scissors blades, similar to tsetse. In both *Lipoptena* and *Neolipoptena* the newly emerged adult has fully functional wings; this winged adult is referred to as a volant. After reaching the host, the wings of these insects break off at the base leaving a stump. The first bloodmeal from the host stimulates physiological changes in the fly, including histolysis of flight muscles and growth of leg muscles to accommodate the subsequent parasitic life of the adult.

Both birds and mammals harbor a few species of Hippoboscidae with reduced wings that are not used for flight. Bequaert (1953) noted that at least four genera and 15 species have reduced wings (subapterous). The halteres, however, are not appreciably reduced in these species. The reduced wings are immovable and cover the halteres, which they probably help protect. Both the forewings and halteres

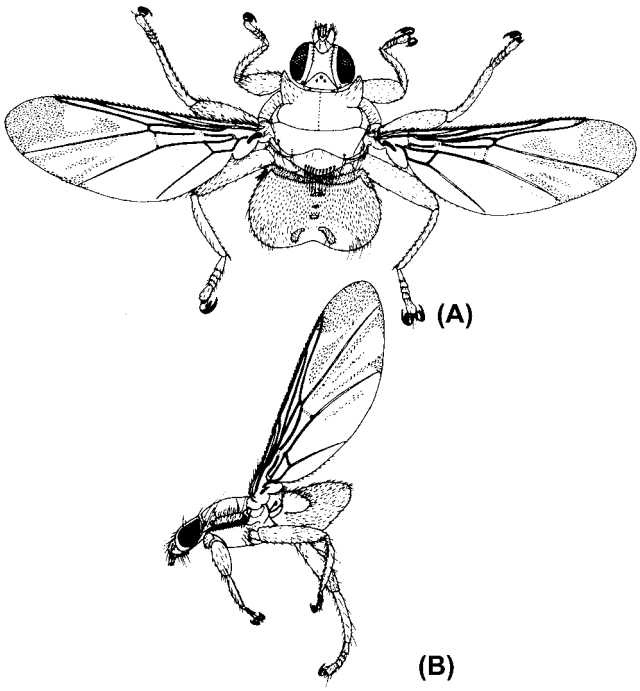


FIGURE 20.2 *Ornithomya avicularia*, adult female (Hippoboscidae). (A) Dorsal view, with wings spread. (B) Lateral view, showing dorsoventral flattening. *From Hutson (1984).*

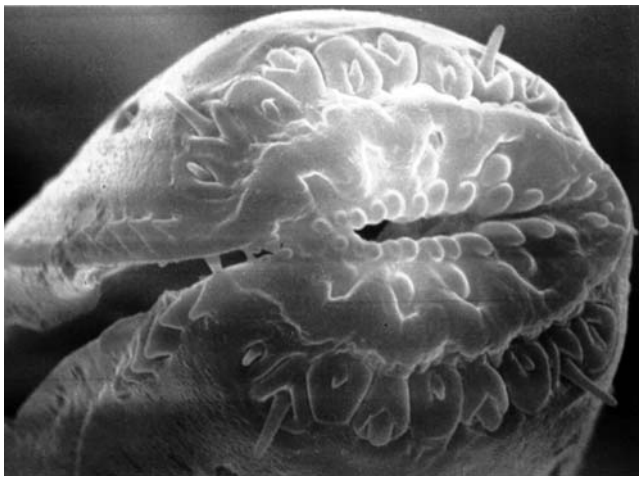


FIGURE 20.3 Tip of proboscis of the sheep ked, *Melophagus ovinus* (Hippoboscidae), showing labella armed with teeth for cutting into host tissue. *Photograph by John E. Lloyd.*

are reduced to small stumps and are nonfunctional in the genus *Melophagus*. This genus is possibly the most specialized in the family. The adults of *Melophagus* spp. emerge from the puparium with rudimentary, nonfunctioning flight muscles, which later atrophy.

Streblidae

This family includes a wide variety of morphologic forms, some of which exhibit highly specialized adaptations for parasitic life. The adults of most species are 1.5−2.5 mm in length; a few Neotropical species may be 0.75−5.0 mm (Wenzel and Peterson, 1987). Several genera are flattened dorsoventrally and superficially resemble hippoboscids or nycteribiids (Fig. 20.4). Species in the subfamily Nycterophiliinae are flattened laterally and resemble fleas. Members of the genus *Ascodipteron* demonstrate neosomy similar to some species of fleas. The female burrows into the skin of the host after shedding her wings.

Although most streblids are winged for at least part of their life, and very mobile, there may be different degrees of wing reduction even within a single genus. Flight is still possible in some species with reduced wings but others are clearly flightless. The function of reduced wings is poorly understood. The head is usually small in the Streblidae and the antennae are inconspicuous. Members of the Streblidae lack ocelli, and the compound eyes are absent or reduced to one or only a few facets. Compound eyes are often better developed in those species that actively fly among bats in roots.

Nycteribiidae

Species in this family vary in body length from 1.5 to 5.0 mm. They are more structurally modified than many of the streblids. The adults are wingless but still possess halteres. Antennae are moderately large in relation to the size of the head and, as in the Hippoboscidae, the antennae of nycteribiids are usually located in antennal pits. The eyes may be absent or are reduced to one, two, or, rarely, four facets. The marked reduction of the eyes of both Streblidae and Nycteribiidae appears to be associated with their occurrence on bats as nocturnal or cave-adapted hosts.

The dorsal plates of the thorax are reduced, and the head and legs of nycteribiids are displaced dorsally. This articular displacement of the legs, together with the complete loss of wings, gives the nycteribiids their spider-like appearance (Fig. 20.5). The head is narrower than those of the hippoboscids and streblids and, in the resting position, it is folded back so that its dorsal surface is in contact with the dorsum of the thorax. The head is rotated forward and downward, through about 180 degrees in order to feed.

LIFE HISTORY

Members of the Hippoboscoidea are **larviparous**. They exhibit a form of viviparity called **adenotrophic viviparity**. A single egg is passed to the uterus, where it embryonates and hatches. The egg contains sufficient yolk to nourish the embryo until hatching. The two subsequent larval instars remain in the uterus, where they are nourished by a pair of accessory glands, or **milk glands**, that empty into the uterus. The glands are very similar in structure to those in female tsetse flies, although their secretions are slightly different. Like other insects that feed exclusively on blood, the Hippoboscoidea rely on symbiotic microorganisms to supplement their nutrition. It is interesting that *Bartonella melophagi*, previously believed to be an endosymbiont has now been found infecting people and sheep. The symbionts of the Hippoboscoidea are passed to the offspring via milk glands in the uterus of the female.

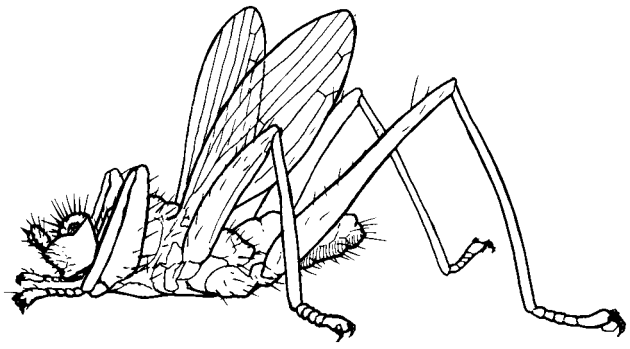


FIGURE 20.4 Representative adult female bat fly (Streblidae), ectoparasitic on bats. *From Furman and Catts (1982).*

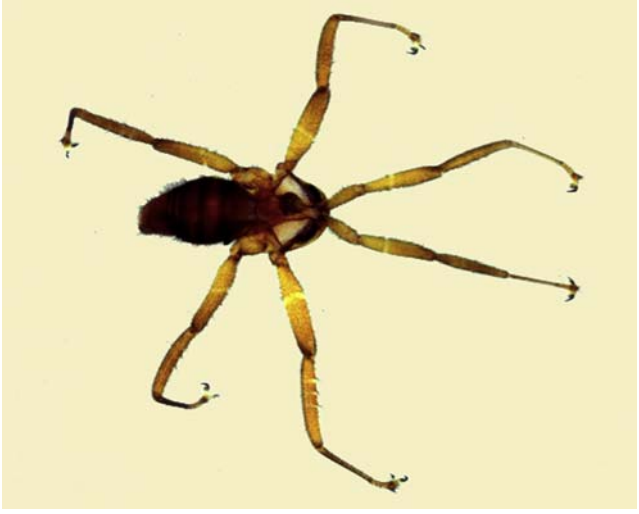


FIGURE 20.5 Spider-like bat fly, *Basilia boardmani* (Nycteribiidae), male, ectoparasite of North American bats. *Photograph by Jerry F. Butler.*

As with many obligate blood-feeding organisms, the Hippoboscoidea carry symbiotic microbes. Some of these have been cultured in the laboratory, and others are known only from microscopy and next-generation sequencing of the bacteriome of Hippoboscoidea. Symbiotic microbes were identified by Bequaert (1953) and other early workers. Later molecular studies focusing on specific microbes identified these bacteria as *Bartonella* spp. Bacteriome-based studies later identified several novel symbionts such as unnamed Gamma-Proteobacteria and *Enterobacteriales*, Candidatus *Arsenophonus* spp., *Rickettsia*, *Wolbachia*, and *Bartonella* (Reeves, 2005; Novakova et al., 2016; Wilkinson et al., 2016). Full genomes of the endosymbiont *Candidatus* Arsenophonus lipopteni have been sequenced and annotated (Novakova et al., 2016). These molecular genome studies could elucidate the roles that the endosymbionts play in physiology of the flies. Far more work has focused on the possibly relegated obligate symbionts of tsetse flies (*Glossina* spp.). Both *Wigglesworthia* spp. and *Sodalis glossinidius* are symbionts of tsetse that produce vitamins and have been the target of novel control strategies because, if they are killed, the host flies either die or suffer loss of fecundity (Sassera et al., 2013). Similar studies have not been directed at the obligate symbionts of Hippoboscoidea, but they could be useful in developing sheep ked control products. Studies of fungal, protozoa, and viral symbionts are lacking.

Parturition occurs when the larva is fully developed but prior to formation of the puparium (Fig. 20.6). The term **prepupa** has been applied to this stage because its structure is similar to that of the third-instar larva. It has ceased to feed, but histolysis of larval organs and formation of the true pupa have not yet started. Shortly after the larva emerges, its integument hardens to form the puparium. In some species, the larva may remain in the uterus until after internal pupal transformations have been initiated. Most puparia are deposited or dropped on the roost substrate, nest, bedding area, wall, or elsewhere in proximity to the host. The sheep ked is unusual in that the puparium is glued by the female to the fleece of the host. The adult fly emerges after a period of several weeks to several months, depending on the species and temperature. Although detailed life histories of streblids and nycteribiids are described for only a few species, Marshall (1970) and Overal (1980) have provided detailed life history descriptions, respectively, of *Basilia hispida*, a nycteribiid, and *Megistopoda aranae*, a streblid.

FIGURE 20.6 Deer ked, *Lipoptena mazamae* (Hippoboscoidea); adult, puparium, and third-instar larva on white-tailed deer. *Photograph by Nathan D. Burkett-Cadena.*

BEHAVIOR AND ECOLOGY

Both sexes of hippoboscoid flies feed as ectoparasites on the blood of birds or mammals; there are no species known to naturally feed on both mammals and birds. Nor, with the exception of *Hippobosca*, does any genus occur on both bird and mammal hosts. Host specificity varies considerably among different groups. Some are restricted to a single host species. Others are restricted to a genus or to several related genera of hosts, whereas still others are generalists that feed on a relatively wide range of host taxa. Almost all species were named prior to modern advances in molecular taxonomy, and there remains a strong possibility that species complexes exist where wide host or geographic ranges are involved.

Hippoboscidae occur on at least 18 orders of birds and five orders of mammals. Host specificity is more marked in species parasitic on mammals than in those parasitic on birds. Apterous species and those with reduced wings, or which have lost their wings altogether, tend to be most host specific. In addition, the more advanced or specialized species tend to be more host specific.

Members of the Streblidae and Nycteribiidae are exclusively parasites of bats (Order Chiroptera). No species of either family is known to occur naturally on both of the chiropteran suborders Megachiroptera and Microchiroptera. Host specificity varies widely within the streblid bat flies and nycteribiid bat flies from one to many host species. New World streblids, which tend to be host specific, have become adapted to living and feeding on particular body regions. Individual species are restricted to the wing membranes, head, or trunk. Some bats (e.g., *Phyllostomus hastatus*) commonly harbor three or four species at the same time, with most hosts having at least two species. In temperate regions, at least some bat flies remain physically active on hibernating bats. Some species apparently continue to be reproductively active despite the low body temperatures of their hosts.

The behavior of almost all bat flies is poorly documented. Nycteribiidae, and most species of Streblidae and Hippoboscidae, deposit their offspring away from their hosts. The fully developed third-stage larva is either

dropped to the ground, litter, or nesting material; deposited in a preferred site; or attached to the host or other substrate. Female nycteribiids and streblids leave their hosts to deposit larvae in the vicinity of bat roosts. This includes bat-roost surfaces, walls of caves, and branches or leaves of trees.

In the Hippoboscinae, the freshly deposited larva of *Melophagus* is covered with a secretion that hardens on drying and glues the puparium to the wool fibers of the host. *Neolipoptena* and *Lipoptena*, which shed their wings after reaching the host, also larviposit on the host. These larvae are not fastened to the host and eventually drop to the ground. The duration of the pupal stage of the sheep ked, within the wool of the host is, at most 45 days, whereas that of the deer ked on the soil may last several months. Most *Hippobosca* spp. larviposit away from the host in some favored location, as does the pigeon fly *Pseudolynchia canariensis*. Many species of Hippoboscidae that feed on nesting birds larviposit in nesting materials, from which their puparia may be collected.

Some streblids can fly and move readily within roosts by flight. However, streblids with small or poorly developed wings and most nycteribiids presumably travel less, but even the wingless species will move from one bat to another and can be seen on the walls of heavily infested bat roosts. Newly emerged *Lipoptena* and *Neolipoptena* often swarm in large numbers in search of a host at certain seasons. These volants have functional wings that break off near the base after the host is reached. Once on the host, adult hippoboscids move swiftly among feathers or hair and are difficult to collect. The relatively slow-moving sheep ked is an exception.

COMMON SPECIES OF HIPPOBOSCIDS

A number of louse flies in the genus *Hippobosca* are of particular interest to veterinary entomologists. Most occur in Europe, Africa, and Asia. Occasional introductions have been made into the United States with the importation of zoo animals. With the exception of the **ostrich louse fly** (*Hippobosca struthionis*), they are parasites of mammals. The sheep ked (*Melophagus ovinus*) is a parasite of sheep and is considered one of the most important insect pests of sheep in many areas of the world.

Sheep Ked (*Melophagus ovinus*)

The sheep ked (Fig. 20.7) is a wingless ectoparasite that spends its entire life on domestic sheep. It is worldwide in distribution except in tropical regions where it occurs only in the cooler highlands. It probably was introduced into the United States in the 15th century shortly after the European discovery of the New World. Often called the "**sheep tick**" by sheep producers, it is found on both range and farm flocks of sheep. The sheep ked is of considerable economic importance and is generally regarded as the most damaging ectoparasite of sheep in North America. A relative of the sheep ked, *Melophagus montanus*, occurs on Dall's sheep (*Ovis dalli*).

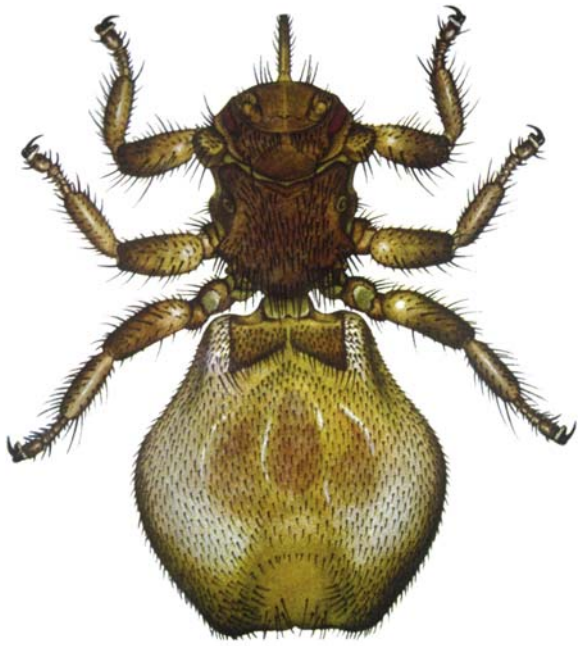


FIGURE 20.7 Sheep ked, *Melophagus ovinus* (Hippoboscidae), female, dorsal view. *Courtesy of Cornell University Agricultural Experiment Station.*

Much of what is known about the life history of hippoboscid flies is based on studies of *M. ovinus*. After a period of 7–8 days of feeding and growing in the uterus of the female, the fully developed larva is deposited and cemented to the sheep's wool. Members of the genus *Melophagus* are the only hippoboscids to attach their larvae to the host. The reddish, barrel-shaped puparium (Fig. 20.8)

FIGURE 20.8 Puparium of sheep ked, *Melophagus ovinus* (Hippoboscidae), adhering to sheep wool. *Photograph by John E. Lloyd.*

is fully formed within 12 h of parturition. In Wyoming (USA), Swingle (1913) determined that the duration of the pupal stage varied from 19 to 23 days in summer to 20–36 days in winter. The variation of this period was attributed to differences in temperature and the distance of the pupa from the skin of the host. Slightly shorter periods have been reported in geographical regions that are warmer than Wyoming. Swingle (1913) further indicated that the duration of the pupal stage is unlikely to be less than 19 days even in the warmest climate and may increase to 40–45 days in the winter.

Teneral females of the sheep ked mate within a day of eclosion, although the first larva is not deposited for at least 12 days. This period includes 6–7 days for larval maturation. Although one mating provides sufficient sperm for a lifetime, repeated mating usually occur when multiple males are present. A female sheep ked normally lives about 4 months and produces 10–12 larvae during her lifetime. Some, however, can live for 6 months or longer and can produce 15–20 larvae. The male life span is slightly shorter, approximately 2–3 months.

Larviposition by the sheep ked tends to occur on lower body parts, especially under the neck and in the breech area. In unshorn sheep, adults are consistently most numerous in the rib area. Contrary to popular belief, there is no daily or seasonal movement from one location to another on the host. In the spring and summer, sheep keds are more likely to be found on the underside of sheep that have been recently shorn and on young lambs with a very short fleece. On adults they can be found in tufts of longer fleece missed by the shears. Numbers of keds tend to be greater on younger animals.

Sheep keds generally live for only a few days if removed from the host. However, they may live up to 5 days in wool in the laboratory and, when kept under cool and moist conditions away from the host and fleece, they may live even longer. Their vigor and ability to relocate a host diminish the longer they remain separated from a host animal.

Although sheep keds that become dislodged from their host have the ability to locate a host from the ground, transfer of sheep keds is primarily by animal-to-animal contact. Newborn lambs become infested with keds directly from their mothers soon after birth. Within a flock, transfer occurs when sheep keds move to the tips of the fleece in response to increasing air temperature, and possibly in response to the brisk movement of sheep that accompanies flocking behavior. Air temperature usually must be 21°C (70°F) or above before many sheep keds are observed on the surface of the fleece. At 27–58°C (80–90°F) sheep keds are common on the outer wool surface. Thus transfer between animals is more likely, and occurs more rapidly, in summer than in winter.

Like many ectoparasites, populations of *M. ovinus* exhibit annual fluctuations in their numbers. Although minor variations have been reported from different parts of the world, populations of the sheep ked tend to be highest from late winter to early spring and lowest in summer. In Wyoming (USA) ked numbers on ewes tend to increase from September to February. Periods of high populations are extended on rams, pregnant ewes, and undernourished animals, with increases in numbers being prolonged for several weeks in pregnant ewes (Nelson and Qually, 1958). On newborn lambs, which receive only teneral keds from their mothers, numbers of sheep keds increase from their birth in early spring to a couple months later when populations begin their normal decline. Seasonal decline of sheep ked populations is attributed to acquired resistance (Nelson and Bainborough, 1963; Baron and Nelson, 1985). This resistance is apparently caused by a long-lasting, cutaneous, arteriolar vasoconstriction that cuts off much of the capillary blood flow to the upper dermis. Keds are unable to obtain sufficient blood and die of starvation.

Sheep keds feed approximately every 24–36 h, with the feeding time increasing to 2-day intervals as the keds become older. The feeding period of an individual ked is typically 5–10 min. Feeding is from larger vessels (30–100 μm) near the bases of the wool follicles and often near the apocrine glands or sweat glands associated with primary follicles (Nelson and Petrunia, 1969). Penetration of the dermis by the mouthparts is accomplished by rapid and continuous movement of prestomal teeth on the labellum, followed by movement of the entire haustellum. After piercing a blood vessel, the mouthparts are secured in place by the prestomal teeth, which are everted and serve to anchor the labella to the vessel wall.

Dog fly (*Hippobosca longipennis*)

The dog fly originally was a parasite of wild carnivores in East Africa. It has been recorded from members of the families Canidae (dogs, foxes), Viverridae (mongoose, civet), Hyaenidae (hyena), and Felidae (cats). It has since become widely distributed in association with domestic dogs from southern Europe and the Mediterranean region to China. It appears to be best adapted to warm and arid climates. Up to one-third of dogs in parts of Egypt are infested with this louse fly. It is found mainly on dogs in the Palearctic region and on wild carnivores in Africa.

In 1972, *H. longipennis* was introduced into North America on captive cheetahs from Africa. Subsequently the species has been detected in the United States on cheetahs at wild animal or safari parks in California, Texas, Georgia, and Oregon. Efforts were made by officials in each of the affected states to eradicate this ectoparasite before it escaped from its introduced host to domestic pets,

livestock, or wildlife. There is no evidence that this species has become established in the United States or elsewhere in the New World.

Hippobosca equina

This species (Fig. 20.9) is normally a parasite of Equidae (horse, donkey, ass) and is a facultative parasite of cattle. Although widespread in the Old World (Europe, northern Africa, western Asia), it does not occur on wild hosts. The original hosts are unknown. *Hippobosca equina* is a serious and common pest of a wide variety of domestic animals in Egypt (Hafez et al., 1977). It can torment its hosts with painful bites and possibly act as a vector of disease agents, including those that cause piroplasmosis of horses, Q fever, and other types of rickettsioses. A recent molecular study by Lee et al. (2016) detected some of these agents in other keds. In Britain, *H. equina* is called the "forest fly."

Hippobosca variegata

Tropical Africa is probably the center of distribution of this species from which it has spread northward to the Mediterranean and eastward into Asia. It is normally a parasite of the domestic horse and its relatives (*Equus* spp.) and cattle (*Bos* spp.). *Hippobosca variegata* is also reported from camels, dromedaries, and water buffalo in Africa and Asia, but these are considered facultative hosts.

Deer Keds (*Lipoptena* and *Neolipoptena* spp.)

Three species of *Lipoptena* and one of *Neolipoptena* are parasites of deer in North America, where they are called **deer keds**. In western North America, *L. depressa* and *N. ferrisi* are frequently found on the same host. The wings of deer keds are deciduous. They are fully developed and functional in the newly emerged adult, or volant, although the wing venation is greatly reduced. The wings are shed, probably within 48 h after the ked reaches a suitable host. Prepupae are deposited while the ked is on the host but eventually fall to the ground.

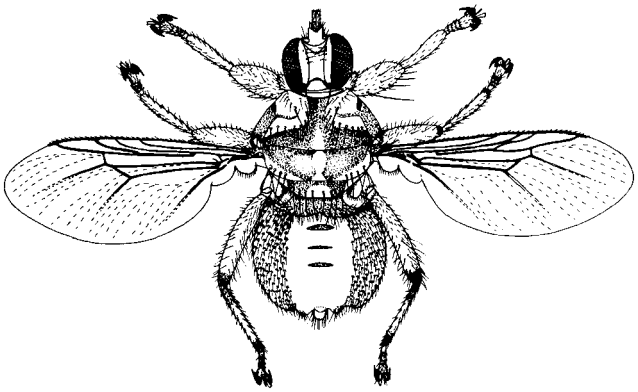


FIGURE 20.9 Horse ked, *Hippobosca equina* (Hippoboscidae), female, dorsal view. *From Hutson (1984).*

Lipoptena depressa

According to Bequaert (1957) *Lipoptena depressa* is the usual and common parasite of several subspecies of *Odocoileus hemionus*. These include the Rocky Mountain mule deer (*O. h. hemionus*), the Columbian black-tailed deer (*O. h. columbianus*), the California black-tailed deer (*O. h. californicus*), and probably the southern black-tailed deer (*O. h. fulginatus*). It probably parasitizes the Western white-tailed deer (*Odocoileus virginianus leucurus*). Both deer species are efficient breeding hosts for *L. depressa*. *Lipoptena depressa* is present along the Pacific Coast from southern British Columbia to southern California and as far inland as Alberta (Canada) and western South Dakota and Nebraska (USA). Its range includes most of the Rocky Mountain states but apparently not Arizona and New Mexico.

Lipoptena depressa has been divided into two subspecies: *L. d. depressa* and *L. d. pacifica* (Maa, 1969) and is an excellent example of a taxon that could be investigated by modern molecular tools. *Lipoptena d. depressa* is limited in its distribution to the eastern slope of the Rocky Mountain highlands in western Montana, northern Wyoming, southwestern South Dakota, and northwestern Nebraska (USA). The normal host is the Rocky Mountain mule deer. *Lipoptena d. pacifica* is found on the western slopes of the Rocky Mountain lowlands, including British Columbia (Canada), and the states of Washington, Oregon, Idaho, and California (USA). *Lipoptena d. pacifica* normally breeds on Columbian black-tailed deer and the western subspecies of white-tailed deer.

Lepoptena depressa volants will alight on any moving object. Most leave quickly, however, without dropping the wings if they land on an accidental host such as humans or horses. Westrom and Anderson (1992) found that *L. depressa* was a bivoltine species in California, with volants appearing in peak numbers in October and April. On the host, peak populations of apterous adults occurred in midsummer and early winter following adult flights. Thousands of *L. depressa* may be found on an individual deer, with populations being especially heavy in the fall.

Lipoptena cervi

This deer ked (Fig. 20.10) is a common parasite of the true elk (*Alces alces*), red deer (*Cervus elaphus*), and other species of deer in Europe, Siberia, and northern China. In the USA it was first reported from New Hampshire and Pennsylvania in 1907. It now occurs in New York, New

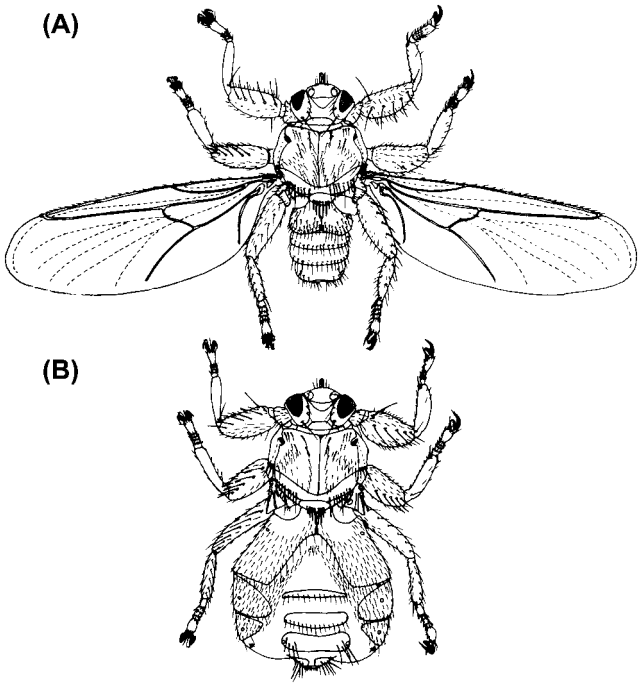


FIGURE 20.10 Deer ked, *Lipoptena cervi* (Hippoboscidae), females, dorsal view. (A) Winged (alate). (B) Wingless (dealate). *From Hutson (1984).*

Hampshire, Massachusetts, and Pennsylvania. Bequaert (1942) believed *L. cervi* was introduced into North America by humans with deer from Europe, and the species subsequently spread to white-tailed deer (*Odocoileus virginianus*). It also has been reported from another host native to the United States, the wapiti (*Cervus canadensis*).

Swarms of newly emerged, winged *L. cervi* appear in the fall, but apterous keds may be found on the host yearround. In Denmark, this deer ked was found mostly in the fine hair of the neck, anal region, groin, and axilla of the host (Haarløv, 1964). In Russia mass emergence is observed late in August and the first part of September. They are most active on warm, clear afternoons and are concentrated in low places protected from the wind, in young deciduous forests. The puparia remain on the ground in areas where their hosts are normally found until they emerge in September. Puparia may be particularly numerous at wallows and places where the hosts rub and shed their winter coats.

Lipoptena mazamae

The range of this tropical insect extends northward from Argentina through South and Central America, where it is a parasite of brocket deer (*Mazama* spp.), into the United States where it occurs on white-tailed deer in the states bordering the Gulf of Mexico and along the Atlantic coastal states to South Carolina. In surveys of white-tailed deer in southern Texas and Florida, it was the most prevalent ectoparasite (Samuel and Trainer, 1972; Forrester et al., 1996). Volants may be observed in every month from April through November and will land, crawl on, and even bite humans. Numbers on deer appear to be significantly lower in the fall than in the spring or summer. Low populations of this species have been attributed to mortality of the puparia in areas of flooding or high rainfall.

Neolipoptena ferrisi

This species occurs in the western United States from Canada to Mexico. Its range includes California, Oregon, Washington, the Rocky Mountain states, and as far east as South Dakota (USA). This deer ked occurs on three subspecies of *Odocoileus hemionus*: the Rocky Mountain mule deer (*O. h. hemionus*), the coastal black-tailed deer (*O. h. columbianus*), and the California black-tailed deer (*O. h. californicus*). Bequaert (1957) considered *O. hemionus* to be the only true breeding host of this insect, and regarded records from western white-tailed deer (*Odocoileus virginianus leucurus*) and prong-horn antelope (*Antilocapra americana*) as accidental occurrences.

In dual infestations, *N. ferrisi* is normally outnumbered by *L. depressa*. *Neolipoptena ferrisi* tends to be collected most frequently from the anterior regions of the body, with the highest population density on the head, whereas *L. depressa* is collected most frequently from the posterior regions of the body, including the tail (Westrom and Anderson, 1992).

Pigeon Fly (*Pseudolynchia canariensis*)

The **pigeon fly** (Fig. 20.11) is a winged hippoboscid that was introduced into North America at least a century ago. The earliest record in North America is 1896, when it was taken on pigeons at Savannah, Georgia (USA) (Knab, 1916). Its distribution now is nearly cosmopolitan. This is the only species of hippoboscid that parasitizes domesticated birds, with the exception of ostrich. In North America, it is found only on domestic pigeons (*Columba livia*). In the Old World it occurs both on domestic pigeons as well as birds of several other avian orders. Juvenile birds are more frequently attacked. Heavily infested birds become emaciated and susceptible to secondary infections. The pigeon fly can bite humans and can be irritating to individuals handling domestic pigeons; it can also carry phoretic mites and chewing lice (da Cunha, 2013).

PUBLIC HEALTH IMPORTANCE

Humans are not normal hosts of any hippoboscoid species. Occasionally, however, species such as the sheep ked and the pigeon fly bite humans and can be annoying to those

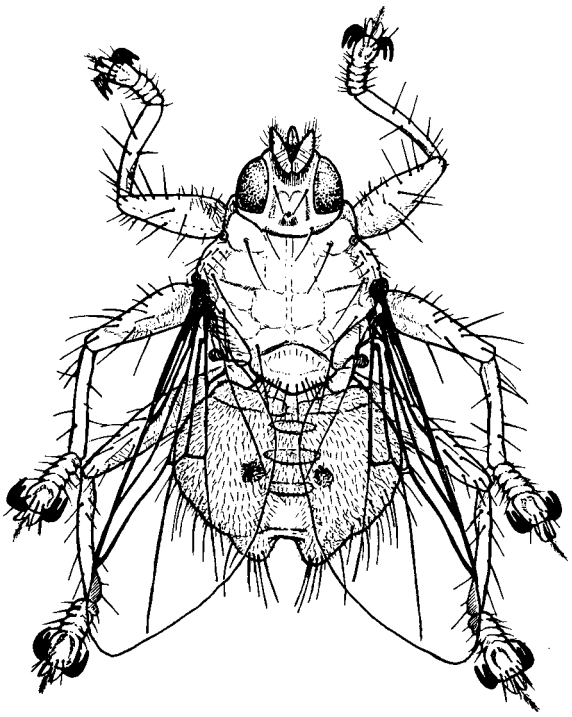


FIGURE 20.11 Pigeon fly, *Pseudolynchia canariensis* (Hippoboscidae), female, dorsal view. *From Furman and Catts (1982).*

routinely handling sheep or domestic pigeons, respectively. Pigeon flies also can be a problem to inhabitants of buildings that have become infested with feral pigeons. Hippoboscids that parasitize nondomesticated animals will bite, and even occasionally feed on a human host. *Olfersia coriacea*, normally a parasite of gallinaceous birds, has been reported attaching to and feeding on the back of the neck of a human in Panama (Harlan and Chaniotis, 1983). Deer keds constitute an annoyance in many areas in Europe because they swarm in large numbers, land on, and bite humans, getting into the hair and under clothing.

Human reactions to hippoboscid bites are variable. A redness and swelling at the bite site are reported by some individuals. Others report that the bite is painful or that there is subsequent irritation at the site of the bite. Individuals have even reported severe pain and swelling that required emergency medical attention. In India *M. ovinus* reportedly causes painful bites to shepherds engaged in shearing (Joseph et al., 1991). (On a personal note, JEL has been bitten on numerous occasions by the sheep ked and has experienced no associated pain or swelling.)

In Europe, feeding on humans by *L. cervi*, resulting in "deer ked dermatitis," has been well documented. In the area of St. Petersburg, Russia, dermatitis was reported among more than 300 individuals bitten by *L. cervi* during mass flights of volants in August and early September (Chistyakov, 1968). Reunala (1980) reported that the predilection areas were the scalp and upper back. Although the bite of *L. cervi* is barely noticeable, and initially leaves little trace, a hard, reddened welt develops within 3 days. The accompanying itch is intense and typically lasts 14−20 days. A pruritic papule may persist for 1 year. *Bartonella schoenbuchensis*, which colonizes the midgut of *L. cervi* and is transmitted to deer and humans by this ked, is the possible etiologic agent of "deer ked dermatitis" (Dehio et al., 2004; Hassler, 2005). *Bartonella schoenbuchensis* is maintained by vertical transmission in *L. cervi* (Bruin et al., 2015) and might be important for producing vitamins or nutrients for the ked. A novel *Bartonella* sp. that was 96% similar to *B. schoenbuchensis* was detected in *Lipoptena mazamae* collected form deer from Georgia and South Carolina (USA) and might cause similar deer ked dermatitis in hunters (Reeves et al., 2006). There are no reports of its presence in *L. cervi* in the United States. Rhinoconjunctival allergy to *L. cervi* has also been confirmed in Finland (Laukkanen et al., 2005).

The restriction of streblid and nycteribiid bat flies to bat hosts, as well as host specificity among some species of bat flies, minimizes the potential for transmission of pathogens to other animals, including humans. For example, even where humans in Africa come in close proximity to bats with *Bartonella* infections there was no evidence of human infection, and this was attributed to the host specificity of the nycteribiids that putatively transmit the pathogen (Mannerings et al., 2016). Dick and Patterson (2006) suggest it is theoretically possible that bat flies could transmit Ebola virus to humans as both nycteribiid and streblid bat flies are known to infest certain species of Old World fruit bats that may harbor this virus. However, this suggestion is not based on the detection or propagation of any *Filovirus* in bat flies and could be as epidemiologically meaningless and spurious as the suggestion that mosquitoes could transmit HIV.

VETERINARY IMPORTANCE

Louse flies directly affect their hosts by feeding on blood. Sometimes heavily infested animals become emaciated and susceptible to secondary infections. Juvenile birds and mammals are often more heavily infested with hippoboscids than older animals of the same species. The body conditions of wintering hosts, birds or mammals, may be worsened by infestation of these parasites. There are ecological data that support the theory that heavy hippoboscid infestations reduce the fitness of premigratory birds (Davis, 2015). The streblid bat fly, *Aspidoptera falcata*, has been shown to cause negative weight gains in its bat host (Linhares and Komeno, 2000). In addition to discomfort caused by their biting, louse flies can be annoying to their hosts simply by crawling about on the body. Louse flies also serve as vectors of pathogens and parasites (Table 20.1) and

TABLE 20.1 Species of Louse Flies (Hippoboscidae) in the United States, and Selected Species of Veterinary Importance From Other Regions of the World

Species	Hosts	Geographic Distribution	Parasites or Disease Agents
Subfamily: Hippoboscinae			
Hippobosca longipennis	Domestic dogs, hyenas	Southern Europe, northern Africa to China	*Acanthocheilonema dracunculoides* (filarial nematode)
Subfamily: Lipopteninae			
Lipoptena cervi	Deer, elk	Northeastern USA, Europe & Asia	*Bartonella schoenbuchensis* in Europe and Asia (bacterium)
Lipoptena depressa	White-tailed deer, mule deer	Western USA	*Corynebacterium lipoptenae* (bacterium; symbiote?)
Lipoptena mazamae	White-tailed deer, brocket	Southeastern USA to South America	*Bartonella* spp. in the USA (bacterium)
Melophagus ovinus	Domestic sheep	Worldwide	*Trypanosoma malophagium, Rickettsia melophagi* (rickettsia; symbiote?), Bluetongue virus?
Neolipoptena ferrisi	Mule deer	Western USA	None
Subfamily: Ornithomyinae			
Icosta albipennis	Egrets, ibises, and other wading birds (Ciconiiformes)	Widespread	None
Icosta americana	Owls, hawks, grouse, turkeys	Widespread	West Nile virus
Icosta angustifrons	Owls, hawks, falcons	Eastern USA	None
Icosta ardeae boutaurinorum	American bittern	Widespread	None
Icosta hirsuta	Quails, grouse, sage hens	Western USA	*Haemoproteus lophortyx*
Icosta holoptera holoptera	Rails	Eastern and central USA	None
Icosta nigra	Hawks, falcons	Widespread	None
Icosta rufiventris	Hawks, falcons, owls	Eastern and central USA	*Haemoproteus lopohortyx*
Microlynchia pusilla	Pigeons, doves, quails, roadrunner	Widespread	*Haemoproteus columbae, H. maccallumi*?, *H. sacharovi*?
Olfersia bisulcata	Vultures	Texas	None
Olfersia fumipennis	Osprey	Widespread	None
Olfersia sordida	Pelicans, cormorants	Widespread	None
Olfersia spinifera	Frigate birds	Florida and Louisiana	None
Ornithoctona erythrocephala	Hawks, pigeons, others	Widespread	None
Ornithoctona fusciventris	Warblers, flycatchers, others	Widespread	None
Ornithoica confluenta	Egrets, ibises, other wading birds	Florida	None
Ornithoica vicina	Owls, sparrows, others	Widespread	None

Continued

TABLE 20.1 Species of Louse Flies (Hippoboscidae) in the United States, and Selected Species of Veterinary Importance From Other Regions of the World—cont'd

Species	Hosts	Geographic Distribution	Parasites or Disease Agents
Ornithomya anchineuria	Hawks, crows, sparrows, others	Widespread	None
Ornithomya bequaerti	Small passerine birds	Widespread	None
Pseudolynchia brunnea	Whip-poor will, nighthawks	Widespread	None
Pseudolynchia canariensis	Domestic pigeons	Widespread	*Haemoproteus columbae, H. maccallumi, Trypanosoma hannai*?
Stilbometopa impresssa	Quails and related game birds	Western USA	*Haemoproteus lophortyx, Trypanosoma* sp.
Stilbometopa podopostyla	Wild pigeons, doves	California	*Trypanosoma avium*

Included are parasites and disease agents that they reportedly can transmit or have been associated with. Unless otherwise indicated, the parasites and disease agents are all blood protozoa.
Adapted from Baker (1967) and Pfadt and Roberts (1978).

as disseminators of certain ectoparasitic arthropods such as lice and mites. These include arboviruses, bacteria, mammalian trypanosomes and filarial worms, avian trypanosomes, hemosporina blood protozoa, lice, and mites.

Baker (1967) published a review of the role played by the Hippoboscidae as vectors of endoparasites. All known hippoboscid vectors of parasitic protozoa are members of two subfamilies: the Ornithomyinae on birds and the Lipopteninae on mammals. Host-specific hipppoboscid flies are thought to be important vectors for cervid and sheep trypanosomes (Rodrigues et al., 2006).

West Nile virus (WNV), which first appeared in the United States in 1999, has been detected in some nonculicine, blood-feeding flies including *Icosta americana*, a bird-feeding hippoboscid. Gancz et al. (2004) detected WNV RNA in 16 of 18 specimens of *I. americana* collected from owls during a WNV outbreak in Ontario, Canada. Farajollahi et al. (2005) detected WNV RNA from four of 86 specimens of *I. americana* collected from several species of raptors in New Jersey (USA). Because two of the four infected specimens had apparently not blood-fed, and the virus may have been in the hemocoel and tissue, these authors indicted that *I. americana* should be further studied as a potential vector of WNV.

Acanthocheilonema dracunculoides (synonym *Dipetalonema dracunculoides*), a parasitic filarial nematode of dogs and hyenas in the Old World, undergoes cyclical development in the dog fly (*Hippobosca longipennis*), which is thought to be its vector. Hippoboscids may transmit filariae of other mammals, particularly those of camels and lemurs, as well as ostriches and other birds. According to Pfadt and Roberts (1978), the role of hippoboscids as vectors of pathogens is probably much greater than presently known. This may be true of streblids and nycteribiids as well. Reeves et al. (2016) reported filarial nematodes in tropical streblids.

The sheep ked transmits *Trypanosoma melophagium*, a flagellate protozoan of sheep present wherever ked-infested sheep are found. Although it is distributed worldwide, this flagellate protozoan is rarely observed because it is present in relatively small numbers. The trypanosomes are ingested by the keds while feeding on sheep blood. The immature forms of the parasite develop in the posterior midgut of the sheep ked, while infective forms develop in the hindgut and are voided with the feces. They normally do not cross the gut wall into the hemolymph. Flagellates gain entry into the sheep when the keds or their feces are ingested. Nelson (1956) reported mortality in the keds as a result of blockage of the posterior midgut by large masses of the crithidial stage of *T. melophagium*. *Lepoptena capreoli*, an ectoparasite of domestic goats and the chamois goat in the Old World, transmits *Trypanosoma theodori* in a similar manner, as does *Ornithomya avicularia*, which transmits *Trypanosoma avium* found in corvid birds. *Pseudolynchia canariensis* and *Stilbometopa impressa* are possible vectors of other avian trypanosomes of pigeons (Family Columbidae) and quail (Family Phasianidae).

Several hippoboscid flies have been identified as vectors of *Haemoproteus* species, hemosporidian blood parasites that cause bird malarias. The importance of hippoboscids in the natural transmission of most species of *Haemoproteus* is unknown. In some wild birds such as magnificent frigate

birds, *Fregata magnificens*, infections with *Haemoproteus iwa* are most likely transmitted by hippoboscids and infections reduce host fitness (Quillfeldt et al., 2010). It is generally assumed that individual species of *Haemoproteus* are transmitted by either hippoboscid flies or *Culicoides* species (Ceratopogonidae) but not both. Many ecological or molecular studies infer that Hippoboscidae are primary vectors for numerous species of *Haemoproteus*.

Development of *Haemoproteus* in a hippoboscid vector is similar to that of the mosquito-borne malarial parasites in the genus *Plasmodium*. After microgamete production and fertilization of the macrogamete in the hippoboscid midgut, the zygote develops into a motile ookinete, which penetrates and encysts on the outside of the wall of the stomach. The oocyst enlarges and its contents differentiate into sporozoites. The enlarged oocyst bursts to release the sporozoites, some of which enter the salivary glands to be introduced into the next host on which the hippoboscid feeds (Baker, 1967).

The best known *Haemoproteus* species is *H. columbae*, which is parasitic in erythrocytes and visceral endothelial cells of the domestic pigeon (*Columba livia*). It is transmitted by the pigeon fly, *P. canariensis*. Infections can result in anemia and unthiftiness in pigeons and cause economic losses to pigeon breeders in the form of nestling mortality. Several other species of *Haemoproteus* are transmitted by hippoboscid flies to a variety of avian hosts (Table 20.1). Proven and presumed vectors of avian haemoproteids include species of *Pseudolynchia*, *Stilbometopa*, *Icosta*, *Ornithomya*, and other hippoboscid genera (Baker, 1967; Pfadt and Roberts, 1978).

The greatest diversity of Hippoboscoidea are ectoparasites of bats. However, because bats are wildlife and many tropical species live away from cities the number of studies on disease transmission by bat flies is limited. Nevertheless, because these flies feed exclusively on blood and feed repeatedly with high host fidelity often on multiple hosts in large colonies of bats, they meet almost all of the criteria to be efficient vectors. In addition, these flies are almost certainly eaten by bats during grooming, which could further permit pathogen transmission. Ecological studies of *Bartonella* infections in bats link ectoparasite numbers to infections (Olival et al., 2015). Members of both families, however, might be important in maintaining and spreading pathogens among bat populations. Streblids, which apparently will bite humans, are frequent blood-feeders that move readily between bats, and have the potential to quickly spread pathogens from one host animal to another.

Arboviruses and *Bartonella* spp. have been isolated from nycteribiids in Spain and Africa (Billeter et al., 2012; Aznar-Lopez, 2013). A rhabdovirus discovered in Spanish bats was detected in both bat saliva and in the nycteribiids. This implied that the virus infected both hosts. Streblids are also associated with possible pathogens of bats including *Bartonella* spp. (Reeves et al., 2005). In addition, DNA from a filiarial nematode, *Litomosoides* sp. (possibly *Litomosoides guiterasi*), was detected in *Trichobius intermedius* (Reeves et al., 2016). Bats are the definitive hosts of several *Litomosoides* spp., but the vectors are unknown and are currently suspected to be mites or bat flies (Bain and Chabaud, 1986).

A number of nycteribiid species are presumed to be vectors of *Poylchromophilus* spp., hemosporidian parasites of bats, primarily in the Old World. Oocysts and sporozoites of *P. murinus* have been found on the midgut and salivary glands, respectively, of the nycteribiid fly *Nycteribia kolenatii*. Developmental stages, mostly sporozoites, of other *Poylchromophilus* spp. have been reported in species of *Nycteribia*, *Penicillida*, and *Basilia* (Garnham, 1973; Gardner and Molyneux, 1988). Nycteribiids were also presumed vectors of *Trypanosoma vespertilionis* (Hoare, 1982).

Although they are neither mechanical nor biological vectors of any important disease agents of sheep, **sheep keds** have been shown to be capable of transmitting bluetongue virus in experimental studies (Luedke et al., 1965). While *Coxiella burnetii* has been detected in numerous blood-feeding flies, it was not reported from hundreds of keds removed from sheep in flocks that included infected animals, but this does not demonstrate that keds are refractory to *Coxiella* (Nelder et al., 2008). Such transmission, if it even occurs naturally, is probably only mechanical. In another study, sheep keds were unable to transmit *Anaplasma ovis*, the etiologic agent of ovine anaplasmosis, from infected to uninfected sheep (Zaugg and Coan, 1986). The deer ked (*Lipoptena fortisetosa*) was associated with probable endosymbiotic *Coxiella* spp. and *Theileria* spp., but there was no evidence that they transmitted these agents to their hosts (Lee et al., 2016).

Bartonella are bacteria that infect erythrocytes of vertebrates and are transmitted by blood-sucking arthropods. These bacteria are considered to be emerging pathogens in humans and animals, and it has been proposed that flies of the Family Hippoboscidae may be vectors of *Bartonella* to wild and domestic ruminants (Halos et al., 2004). The early work by Bequaert (1952-1953) probably noted a *Bartonella* as *Rickettsia melophagi* and Aschner (1943) probably detected a similar bacterial agent in *Eucampsipoda aegyptia* a nycteribiid. In that work it was described as a symbiont. Isolation of *Bartonella melophagi* from both sheep and in clinical blood samples from humans shows it is transmittable to vertebrate hosts (Bemis and Kania, 2007; Maggi et al., 2009; Kosoy et al., 2016). Wild-caught keds can be infected (Rudolf et al., 2016). Exposure to sheep keds was not considered a major factor in the reported human cases but published serological or molecular studies of sheep ranchers with ked exposure are lacking.

Feeding by the sheep ked can cause a defect in sheepskins called **cockle** or **rib cockle** (Fig. 20.12) (Everett et al., 1969; Laidet, 1969). Blemishes appear at the individual bite sites and are presumed to be the result of an allergic reaction to the salivary secretions of the feeding keds. The result is scattered, dense, brownish nodules in the grain layer of sheepskin, which seriously downgrades both grain and suede types of leather. The nodules of dense fibrous material cannot be flattened out and are impenetrable to dyes (Fig. 20.13). This defect, especially damaging in garment suede, causes economic losses of several million dollars to the leather industry in the United States each year. When sheep keds are eliminated, the skin recovers from the effect of the bites resulting in usable pelts. The length of time required for recovery by the living animal has not been determined but may be several weeks. A similar defect is caused by the sheep biting louse (*Bovicola ovis*) (Heath, 1994).

Results of studies of weight gains and wool growth of sheep parasitized by sheep keds are equivocal. Several reports in the literature indicate no adverse effects due to sheep ked infestations. In a study of the effect of keds on weight gains of feeder lambs in Wyoming (USA), for example, there was no significant difference in gain between ked-infested and uninfested lambs. In that study the number of keds infesting untreated lambs markedly decreased during the period of lamb feeding (Pfadt et al., 1953). In a study in Canada, Nelson and Slen (1968) found that ked-free lambs on various diets gained approximately 1.4−3.6 kg (3−8 pounds) more than infested lambs, and that uninfested yearling ewes produced about 11% more wool than infested ones. In a study in New Mexico, a 2% higher dressing percentage was observed in carcasses of uninfested lambs, and carcass weights were significantly heavier (0.9 kg per animal) (Everett et al., 1971). Fleece length was about 8% longer and percent clean fiber was approximately 7% greater in uninfested lambs, while the difference in clean, dry weight was about 20% in favor of uninfested animals.

FIGURE 20.12 Feeding damage (cockle) caused by the sheep ked, *Melophagus ovinus* (Hippoboscidae). Grain side of pickled sheepskin showing pitted surface, or cockle. *Photograph by John E. Lloyd.*

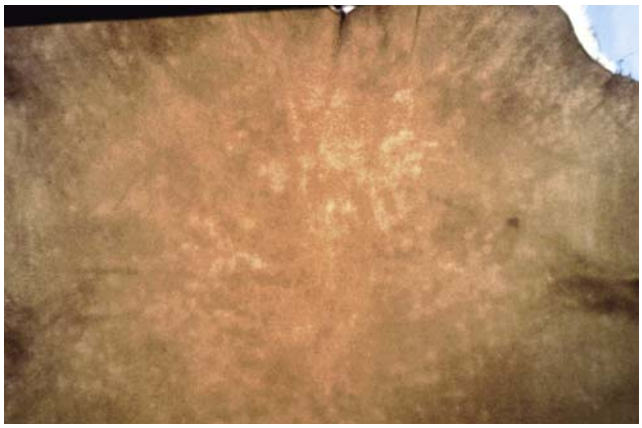


FIGURE 20.13 Discoloration and mottling of sheep leather due to feeding by sheep ked, *Melophagus ovinus* (Hippoboscidae). *Photograph by John E. Lloyd.*

Many ranchers in the western United States believe that heavy infestations of sheep ked contribute to the incidence of **back loss**. This term refers to the death of ewes that roll onto their backs in an apparent attempt to relieve irritation caused by keds. An animal that becomes stuck and remains on its back will eventually suffocate due to the pressure of its internal organs against the diaphragm.

PREVENTION AND CONTROL

Control technology has not been developed for the vast majority of the Hippoboscoidea, because they only feed on wildlife, and most of these parasites are to be endured. The few species that affect domestic animals and birds may be controlled through treatment of the host with insecticide formulations. The pigeon fly, for example, is controlled by periodic cleaning of the pigeon loft and, as necessary, dusting squabs with an insecticidal dust.

There are no specific treatments to control bat flies but if they were considered a threat to endangered bats or if they were shown to be reservoirs for significant zoonotic diseases there could be interest in controlling bat flies. There are isolated incidents where other ectoparasites of bats living in houses have been implicated in the transmission of human disease (Loftis et al., 2006). Because ectoparasites in bat roosts can become pests in human houses, some pest control companies already treat for free living ectoparasites such as *Cimex* spp. and possibly inadvertently kill bat flies.

The sheep ked is the only species for which an extensive control technology has been developed. Shearing prior to lambing can reduce sheep ked populations by approximately

75%. Shearing not only removes many pupal and adult keds with the wool but also kills many that are cut with the shears. If ewes are not shorn prior to lambing, substantial numbers of keds can transfer from full-fleeced ewes to their lambs where they are not subjected to the hazards of shearing.

Insecticidal treatment of sheep in the spring following shearing is a common and effective practice. Best results are achieved when ewes are treated following shearing but before lambing. Often, the ewes are treated as they leave the shearing shed. Several traditional treatment methods include whole-body **sprays**, **dusts**, and **dips**. Fall treatments tend to be less effective, possibly due to the greater length of the fleece at that time. Incidentally, applications of repellents to control biting midges also reduce ked populations (Reeves et al., 2010).

Other methods of treatment are the pour-on and low-volume spray applications. Particularly effective are pyrethroid insecticides, which have become popular because of their ease of application. The pour-on method entails applying a few ounces of insecticide along the backline of each animal (Fig. 20.14). The chemical may be poured from a calibrated dipper, or forcefully expelled into the fleece with an application gun. These methods are usually more effective if one waits a few weeks after shearing to permit growth of sufficient wool to retain the liquid formulation.

FIGURE 20.14 Pour-on application of insecticide for control of sheep keds. *Photograph by John E. Lloyd.*

In the low-volume application, less than an ounce of insecticide is applied to each animal at a pressure of about 50 psi. Large numbers of sheep may be treated in a short period of time by driving the sheep through a spray race equipped with one or more stationary spray nozzles. Animals can be driven through the spray race at approximately one animal per second, providing a treatment method well suited for large range flocks.

Several western states in the United States (Colorado, Idaho, Montana, North Dakota, South Dakota, Utah, and Wyoming) have adopted state-wide, voluntary ked-free programs. These programs involve regular surveillance of sheep, usually at shearing, and prompt treatment for sheep keds when necessary. These programs are normally coordinated by sheep-producer associations within each state. The first statewide ked-free program in the United States was implemented in 1986 by the Wyoming Woolgrowers Association and includes 80% of the sheep in that state.

REFERENCES AND FURTHER READING

Aschner, M. (1946). The symbiosis of *Eucampsipoda aegyptia* Mcq. (Diptera Pupipara: Nycteridiidae). *Bulletin de la Société Fouad Ier d'Entomologie, 30*, 1–6.

Atkinson, C. T. (1991). Vectors, epizootiology, and pathogenicity of avian species of *Haemoproteus* (Haemosporina: Haemoproteidae). *Bulletin of the Society for Vector Ecology, 16*, 109–126.

Aznar-Lopez, C., Vazquez-Moron, S., Marston, D. A., Juste, J., Ibanez, C., Berciano, J. M., et al. (2013). Detection of Rhabdovirus viral RNA in oropharyngeal swabs and ectoparasites of Spanish bats. *Journal of General Virology, 94*, 69–75.

Bain, O., & Chabaud, A. G. (1986). Atlas des larves infestantes de filaires. *Tropical Medicine & Parasitology, 37*, 301–340.

Baker, J. R. (1967). A review of the role played by the Hippoboscidae (Diptera) as vectors of endoparasites. *The Journal of Parasitology, 53*, 412–418.

Baron, R. W., & Nelson, W. A. (1985). Aspects of the humoral and cell-mediated immune responses of sheep to the ked *Melophagus ovinus* (Diptera, Hippoboscidae). *Journal of Medical Entomology, 22*, 544–549.

Bemis, D. A., & Kania, S. A. (2007). Isolation of *Bartonella* sp. from sheep blood. *Emerging Infectious Diseases, 13*, 1565–1567.

Bequaert, J. C. (1942). A monograph of the Melophaginae, or ked-flies, of sheep, goats, deer and antelopes (Diptera, Hippoboscidae). *Entomologica Americana, 22*, 1–220.

Bequaert, J. C. (1952–1953). The Hippoboscidae or louse-flies (Diptera) of mammals and birds. Part 1. Structure, physiology and natural history. *Entomologica Americana*, 32, 1–209 (1952), 33, 211–442 (1953).

Bequaert, J. C. (1954-57). The Hippoboscidae or louse-flies (Diptera) of mammals and birds. Part II. Taxonomy, evolution and revision of American genera and species. *Entomologica Americana*, 34, 1–232 (1954), 35, 233–416 (1955), 36, 417–611(1957).

Billeter, S. A., Hayman, D. T. S., Peel, A. J., Baker, K., Wood, J. L. N., Cunningham, A., et al. (2012). *Bartonella* species in bat flies (Diptera: Nycteribiidae) from western Africa. *Parasitology, 139*, 324–329.

de Bruin, A., van Leeuwen, A. D., Jahfari, S., Takken, W., Foldvari, M., Dremmel, L., et al. (2015). Vertical transmission of *Bartonella schoenbuchensis* in *Lipoptena cervi*. *Parasites & Vectors, 8*, 176–181.

Constantine, D. G. (1970). Bats in relation to health, welfare and economy of man. In W. A. Wimsatt (Ed.), *Biology of bats* (Vol. II, pp. 319–449). Academic Press.

Chistyakov, A. F. (1968). Skin lesions in people due to bite of *Lipoptena cervi*. *Vestnik Dermatologii I Venerologii, 42*, 59–62.

da Cunha Amaral, H. L., Bergmann, F. B., Silveira, T., dos Santos, P. R. S., & Krüger, R. F. (2013). *Pseudolynchia canariensis* (Diptera: Hippoboscidae): Distribution pattern and phoretic association with skin mites and chewing lice of *Columba livia* (Aves: Columbidae). *Journal of Natural History, 47*, 2927–2936.

Davis, A. K. (2015). Can a blood-feeding ectoparasitic fly affect songbird migration? Examining body condition and fat reserves of five bird species in relation to hippoboscid fly parasitism. *Ecologicla Parasitology and Immunology, 4*, 235907. https://doi.org/10.4303/epi/235907.

Dehio, C., Sauder, U., & Hiestand, R. (2004). Isolation of *Bartonella schoenbuchensis* from *Lipoptena cervi*, a blood-sucking arthropod causing deer ked dermatitis. *Journal of Clinical Microbiology, 42*, 5320–5323.

Dick, C. W., & Patterson, B. D. (2006). Bat flies: Obligate ectoparasites of bats. In S. Morand, B. R. Krasnov, & R. Poulin (Eds.), *Micromammals and macroparasites, from evolutionary ecology to management* (pp. 179–194). Tokyo: Springer-Verlag.

Dittmar, K., Porter, M. L., Murray, S., & Whiting, M. F. (2006). Molecular phylogenetic analysis of nycteribiid and streblid bat flies (Diptera: Brachycera, Calyptratae): Implications for host associations and phylogeographic regions. *Molecular Phylogenetics and Evolution, 38*, 155–170.

Evans, G. O. (1950). Studies on the bionomics of the sheep ked, *Melophagus ovinus* L., in West Wales. *Bulletin of Entomological Research, 40*, 459–478.

Everett, A. L., Roberts, I. H., & Naghski, J. (1971). Reduction in leather value and yields of meat and wool from sheep infested with keds. *Journal of the American Leather Chemistry Association, 66*, 118–130.

Everett, A. L., Roberts, I. H., Willard, H. J., Apodaca, S. A., Bitcover, E. H., & Naghski, J. (1969). The cause of cockle, a seasonal sheepskin defect, identified by infesting a test flock with keds (*Melophagus ovinus*). *Journal of the American Leather Chemistry Association, 64*, 460–476.

Farajollahi, A., Crans, W. J., Nickerson, D., Bryant, P., Wolf, B., Glaser, A., et al. (2005). Detection of West Nile virus RNA from the louse fly *Icosta americana* (Diptera: Hippoboscidae). *Journal of the American Mosquito Control Association, 21*, 474–476.

Forrester, D. J., McLaughlin, G. S., Telford, S. R., Jr., Foster, G. W., & McCown, J. W. (1996). Ectoparasites (Acari, Mallophaga, Anoplura, Diptera) of white-tailed deer, *Odocoileus virginianus*, from southern Florida. *Journal of Medical Entomology, 33*, 96–101.

Gancz, A. Y., Barker, I. K., Lindsay, R., Dibernardo, A., McKeever, K., & Hunter, B. (2004). West Nile virus outbreak in North American owls, Ontario, 2002. *Emerging Infectious Diseases, 10*, 2135–2142.

Gardner, R. A., & Molyneux, D. H. (1988). *Polychromohilus murinus*: A malarial parasite of bats: Life-history and ultrastructural studies. *Parasitology, 96*, 591–605.

Garnham, P. C. C. (1973). The zoogeography of *Polychromophilus* and description of a new species of a gregarine (*Lankestria galliardi*). *Annals of Parasitology (Paris), 48*, 231–242.

Haarløv, N. (1964). Life cycle and distribution pattern of *Lipoptena cervi* (L.) (Dipt., Hippobosc.) on Danish deer. *Oikos, 15*, 93–129.

Hafez, M., Hilali, M., & Fouda, M. (1977). Biological studies on *Hippobosca equina* (L.) (Diptera: Hippoboscidae) infesting domestic animals in Egypt. *Zeitschrift Fur Angewandte Entomologie, 83*, 426–441.

Halos, L., Jamal, T., Maillard, R., Girard, J., Guillot, B., Comel, B., et al. (2004). Role of Hippoboscidae flies as potential vectors of *Bartonella* spp. infecting wild and domestic ruminants. *Applied and Environmental Microbiology, 70*, 6302–6305.

Hare, J. E. (1945). Flying stages of the deer louse fly, *Lipoptena depressa* (Say), in California (Diptera, Hippoboscidae). *Pan-Pacific Entomology, 21*, 48–57.

Harlan, H. J., & Chaniotis, B. N. (1983). Report of *Olfersia coriacea* (Diptera: Hippoboscidae) feeding on a human in Panama. *The Journal of Parasitology, 69*, 1026.

Hassler, D., Kimmig, P., & Braun, R. (2005). *Bartonella schoenbuchensis* and the deer ked. *Deutsche Medizinische Wochenschrift, 130*, 13–14.

Heath, A. C. G., Cooper, S. M., Cole, D. J. W., & Bishop, D. M. (1994). Evidence for the role of the sheep biting louse *Bovicola ovis* in producing cockle, a sheep pelt defect. *Veterinary Parasitology, 59*, 53–58.

Hoare, C. A. (1972). *The trypanosomes of mammals*. Oxford, United Kingdom: Blackwell Scientific Publications.

Hurka, K., & Soos, A. (1986). Family Nycteribiidae. Family Streblidae. In A. Soos, & L. Papp (Eds.), *Catalog of Palaearctic Diptera Scatophagidae-Hypodermatidae* (Vol. 11, pp. 226–236). Amsterdam, Netherlands: Elsevier.

Hutson, A. M. (1984). Keds, flat-flies and bat-flies, Diptera, Hippoboscidae and Nycteribiidae. In *Handbooks for the identification of British insects* (Vol. 10, Part 7) Royal Entomological Society of London, 40 pp.

Jobling, B. (1929). A comparative study of the structure of the head and mouthparts in the Streblidae (Diptera: Pupipara). *Parasitology, 18*, 319–349.

Joseph, S. A., Karunamoorthy, G., Ramachandran, P. K., Sukumaran, D., & Rao, S. S. (1991). Studies on the haematophagous arthropods of zoonotic importance in Tamil Nadu. Entomology for defense services. In *Proceedings of the symposium held on 12–14 September 1990* (pp. 185–192). Gwalior, India: Defense Research & Development Establishment.

Keirans, J. E. (1975). A review of the phoretic relationship between Mallophaga (Phthiraptera: Insecta) and Hippoboscidae (Diptera: Insecta). *Journal of Medical Entomology, 12*, 71–76.

Knab, F. (1916). Four European Diptera established in North America. *Insecutor Inscitiae Menstruus, 4*, 1–4.

Kosoy, M., Baia, Y., Enscorea, R., Rizzoa, M. R., Benderb, S., Popovc, V., et al. (2016). *Bartonella melophagi* in blood of domestic sheep (*Ovis aries*) and sheep keds (*Melophagus ovinus*) from the southwestern US: Cultures, genetic characterization, and ecological connections. *Veterinary Microbiology, 190*, 43–49.

Laidet, M. (1969). L'orinine de la noisillure le Melophage. *Technicuir, 4*, 39–50.

Laukkanen, A., Ruoppi, P., & Makinen-Kiljunen, S. (2005). Deer ked-induced occupational allergic rhinoconjunctivitis. *Annals of Allergy, Asthma, & Immunology, 94*, 604–608.

Lee, S., Kim, K., Kwon, O., Ock, Y., Kim, T., Choi, D., et al. (2016). Novel detection of *Coxiella* spp., *Theileria luwenshuni*, and *T. ovis* endosymbionts in deer keds (*Lipoptena fortisetosa*). *PLoS One, 11*(5), e0156727. doi: 10:13871/journal.pone.0156727.

Legg, D. E., Kumar, R., Watson, D. W., & Lloyd, J. E. (1991). Seasonal movement and spatial distribution of the sheep ked (Diptera: Hippoboscidae) on Wyoming lambs. *Journal of Economic Entomology, 84*, 1532−1539.

Lenoble, B. J., & Denlinger, D. L. (1982). The milk gland of the sheep ked, *Melophagus ovinus*: A comparison with *Glossina*. *Journal of Insect Physiology, 28*, 165−172.

Linhares, A. X., & Komeno, C. A. (2000). *Trichobius joblingi, Aspidoptera falcata*, and *Megistopoda proxima* (Diptera Streblidae) parasitic on *Carolla perspicilllata* and *Sturnira lillum* (Chiroptera: Phyllostomidae) in southeastern Brazil: Sex ratios, seasonality, host site preference, and effect of parasitism on the host. *The Journal of Parasitology, 86*, 167−170.

Lloyd, J. E. (1985). Arthropod pests of sheep. In R. E. Williams, R. D. Hall, A. B. Broce, & P. J. Scholl (Eds.), *Livestock entomology* (pp. 253−267). John Wiley.

Lloyd, J. E., Olson, E. J., & Pfadt, R. E. (1978). Low-volume spraying of sheep to control the sheep ked. *Journal of Economic Entomology, 71*, 548−550.

Lloyd, J. E., Pfadt, R. E., & Olson, E. J. (1982). Sheep ked control with pour-on applications of organophosphorus insecticides. *Journal of Economic Entomology, 75*, 5−6.

Loftis, A. D., Gill, J. S., Schriefer, M. E., Levin, M. L., Eremeeva, M. E., Gilchrist, M. J., et al. (2005). Detection of *Rickettsia, Borrelia*, and *Bartonella* in *Carios kelleyi* (Acari: Argasidae). *Journal of Medical Entomology, 42*, 473−480.

Luedke, A. J., Jochim, M. M., & Bowne, J. G. (1965). Preliminary bluetongue transmission with the sheep ked *Melophagus ovinus* (L.). *Canadian Journal of Comparative Medical and Veterinary Science, 29*, 229−231.

Lukashevich, E. D., & Mostovski, M. B. (2003). Hematophagous insects in the fossil record. *Paleontological Journal, 37*, 153−161.

Maa, T. C. (1963). Genera and species of Hippoboscidae (Diptera): Types, snyonymy, habitats and natural groupings. *Pacific Insects Monographs, 6*, 1−186.

Maa, T. C. (1966). Studies in Hippoboscidae (Diptera). *Pacific Insects Monographs, 10*, 1−148.

Maa, T. C. (1969). Studies in Hippoboscidae (Diptera). Part 2. *Pacific Insects Monographs, 20*, 1−312.

Maa, T. C. (1971). Studies in batflies (Diptera: Streblidae, Nycteribiidae). Part I. *Pacific Insects Monographs, 28*, 1−248.

Maa, T. C., & Peterson, B. V. (1987). Hippoboscidae. In J. F. McAlpine, B. V. Peterson, G. E. Shewell, H. J. Teskey, J. R. Vockeroth, & D. M. Wood (Eds.), *Manual of Nearctic Diptera* (Vol. 2, pp. 1271−1281). Research Branch, Agriculture Canada, Monograph 28.

Maggi, R. G., Kosoy, M., Mintzer, M., & Breitschwerdt, E. B. (2009). Isolation of *Candidatus* Bartonella melophagi from human blood. *Emerging Infectious Diseases, 15*, 66−68.

Mannerings, A. O., Osikowicz, L. M., Restif, O., Nyarko, E., Suu-Ire, R., Cunningham, A. A., et al. (2016). Exposure to bat-associated *Bartonella* spp. among humans and other animals, Ghana. *Emerging Infectious Diseases, 22*, 922−924.

Marshall, A. G. (1970). The life cycle of *Basilia hispida* Theodor 1967 (Diptera: Nycteribiidae) in Malaysia. *Parasitology, 61*, 1−18.

Marshall, A. G. (1981). *The ecology of parasitic insects*. London: Academic Press, 459 pp.

Martínez, M. M. R., Lopez, M. P. I., Iñiguez-Dávalos, L. I., Yuill, T., Orlova, M. V., & Reeves, W. K. (2016). New records of ectoparasitic Acari (Arachnida) and Streblidae (Diptera) from bats in Jalisco, Mexico. *Journal of Vector Ecology, 41*, 309−313.

Nelder, M. P., & Reeves, W. K. (2005). Ectoparasites of road-killed vertebrates in northwestern South Carolina, USA. *Veterinary Parasitology, 129*, 313−322.

Nelder, M. P., Lloyd, J. E., Loftis, A. D., & Reeves, W. K. (2008). *Coxiella burnetii* in wild-caught filth flies. *Emerging Infectious Diseases, 14*, 1002−1004.

Nelson, W. A. (1956). Mortality in the sheep ked, *Melophagus ovinus* (L.) caused by *Trypanosoma melophagium* Flu. *Nature, 178*, 750.

Nelson, W. A. (1958). Transfer of sheep keds, *Melophagus ovinus* (L.), from ewes to their lambs. *Nature, 181*, 56.

Nelson, W. A., & Bainborough, A. R. (1963). Development in sheep of resistance to the ked *Melophagus ovinus* (L.). III. Histopathology of sheep skin as a clue to the nature of resistance. *Experimental Parasitology, 13*, 118−127.

Nelson, W. A., & Petrunia, D. M. (1969). *Melophagus ovinus*: Feeding mechanism on transilluminated mouse ear. *Experimental Parasitology, 26*, 308−313.

Nelson, W. A., & Qually, M. C. (1958). Annual cycles in numbers of the sheep ked, *Melophagus ovinus* (L.). *Canadian Journal of Animal Science, 38*, 194−199.

Nelson, W. A., & Slen, S. B. (1968). Weight gains and wool growth in sheep infested with the sheep ked *Melophagus ovinus*. *Experimental Parasitology, 22*, 223−226.

Nováková, E., Václav, H., Nguyen, P., Husník, F., & Darby, A. C. (2016). Genome sequence of *Candidatus* Arsenophonus lipopteni, the exclusive symbiont of a blood sucking fly *Lipoptena cervi* (Diptera: Hippoboscidae). *Standards in Genomic Sciences, 11*, 72. https://doi.org/10.1186/s40793-016-0195-1.

Olival, K. J., Dittmar, K., Bai, Y., Rostal, M. K., Lei, B. R., Daszak, P., et al. (2015). *Bartonella* spp. in a Puerto Rican bat community. *Journal of Wildlife Diseases, 51*, 274−278.

Overal, W. L. (1980). Host-relations of the bat fly, *Megistopoda aranea* (Diptera: Streblidae) in Panama. *University of Kansas Science Bulletin, 52*, 1−20.

Peterson, B. V., & Wenzel, R. L. (1987). Nycteribiidae. In J. F. McAlpine, B. V. Peterson, G. E. Shewell, H. J. Teskey, J. R. Vockeroth, & D. M. Wood (Eds.), *Manual of Nearctic Diptera* (Vol. 2, pp. 1283−1291). Research Branch, Agriculture Canada, Monograph 28.

Pfadt, R. E., Lloyd, J. E., & Spackman, E. W. (1973). *Control of insect and related pests of sheep*. University of Wyoming Agricultural Experiment Statin Bulletin 514R, 15 pp.

Pfadt, R. E., Paules, L. H., & DeFoliart, G. R. (1953). Effect of the sheep ked on weight gains of feeder lambs. *Journal of Economic Entomology, 46*, 95−99.

Pfadt, R. E., & Roberts, I. H. (1978). Louse flies (family Hippoboscidae). In R. A. Bram (Ed.), *Surveillance and collection of arthropods of veterinary importance* (pp. 60−71). U.S. Dept. Agric., Agric. Handbk. No. 518.

Philips, J. R., & Fain, A. (1991). Acarine symbionts of louseflies (Diptera: Hippoboscidae). *Acarologia, 32*, 377−384.

Quillfeldt, P., Martínez, J., Hennicke, J., Ludynia, K., Gladbach, A., Masello, J. F., et al. (2010). Hemosporidian blood parasites in seabirds - a comparative genetic study of species from Antarctic to tropical habitats. *Science and Nature, 97*, 809−817.

Reeves, W. K. (2005). Molecular genetic evidence for a novel bacterial endosymbiont of *Icosta americana* (Diptera: Hippoboscidae). *Entomological News, 116*, 263−265.

Reeves, W. K., Nelder, M. P., Cobb, K. D., & Dasch, G. A. (2006). *Bartonella* spp. in deer keds, *Lipoptena mazamae* (Diptera: Hippoboscidae), from Georgia and South Carolina, USA. *Journal of Wildlife Diseases, 42*, 391−396.

Reeves, W. K., Lloyd, J. E., Stobart, R., Stith, C., Miller, M. M., Bennett, K. E., et al. (2010). Control of *Culicoides sonorensis* (Diptera: Ceratopogonidae) blood feeding on sheep with long lasting repellent pesticides. *Journal of the American Mosquito Control Association, 26*, 302−305.

Reeves, W. K., Beck, J., Orlova, M. V., Daly, J. L., Pippin, K., Revan, F., et al. (2016). Ecology of bats, their ectoparasites, and associated pathogens on Saint Kitts Island. *Journal of Medical Entomology, 53*, 1218−1225.

Reunala, T., Rantanen, T., Vuojolahti, P., & Hackman, W. (1980). Deer ked (*Lipoptena cervi* L.) causes chronic dermatitis in man. *Duodecim, 96*, 897−902.

Rodrigues, A. C., Paiva, F., Campaner, M., Stevens, J. R., Noyes, H. A., & Teixeira, M. M. G. (2006). Phylogeny of *Trypanosoma (Megatrypanum) theileri* and related trypanosomes reveals lineages of isolates associated with artiodactyl hosts diverging on SSU and ITS ribosomal sequences. *Parasitology, 132*, 215−224.

Rudolf, I., Betášová, L., Bischof, V., Venclíková, K., Blažejová, H., Mendel, J., et al. (2016). Molecular survey of arthropod-borne pathogens in sheep keds (*Melophagus ovinus*), Central Europe. *Parasitology Research, 115*, 3679−3682.

Samuel, W. M., & Trainer, D. O. (1972). *Lipoptena mazamae* Rodani, 1878 (Diptera: Hippoboscidae) on white-tailed deer in southern Texas. *Journal of Medical Entomology, 9*, 104−106.

Sassera, D., Epis, S., Pajoro, M., & Bandi, C. (2013). Microbial symbiosis and the control of vector-borne pathogens in tsetse flies, human lice, and triatomine bugs. *Pathogens and Global Health, 107*, 285−292.

Schlein, Y. (1970). A comparative study of the thoracic skeleton and musculature of the Pupipara and the Glossinidae (Diptera). *Parasitology, 60*, 327−373.

Strickman, D., Lloyd, J. E., & Kumar, R. (1984). Relocation of hosts by the sheep ked (Diptera: Hippoboscidae). *Journal of Economic Entomology, 77*, 437−439.

Swingle, L. D. (1913). The life-history of the sheep-tick *Melophagus ovinus*. In *Bulletin no. 99*. University of Wyoming, Agricultural Experiment Station, 24 pp.

Theodor, O. (1964). On the relationships between the families of the Pupipara. In *Proc. 1st Congr. Parasit., Rome, Italy* (pp. 999−1000).

Theodor, O. (1967). *An illustrated catalogue of the Rothschild collection of Nycteribiidae (Diptera) in the British Museum (Natural History) with keys and short descriptions for the identification of subfamilies genera, species and subspecies*. British Museum (Natural History), London, Publ. 655, 506 pp.

Theodor, O. (1968). New species and new records of Nycteribiidae from the Ethiopian, Oriental, and Pacific regions. *Parasitology, 58*, 247−276.

Theodor, O. (1975). *Fauna Palaestina, Insecta I: Diptera Pupipara*. Jerusalem: Publ. Israel Acad. Sci. Human., Sect. Sciences, The Jerusalem Post Press, 168 pp.

Theodor, O., & Oldroyd, H. (1964). Hippoboscidae. In E. Lindner (Ed.), *Die fliegen der Palearktischen Region* (Vol. 65, pp. 1−70).

Wenzel, R. L., & Peterson, B. V. (1987). Streblidae. In J. F. McAlpine, B. V. Peterson, G. E. Shewell, H. J. Teskey, J. R. Vockeroth, & D. M. Wood (Eds.), *Manual of Nearctic Diptera* (Vol. 2, pp. 1293−1301). Research Branch, Agriculture Canada, Monograph 28.

Wenzel, R. L., Tipton, V. J., & Kiewlicz, A. (1966). The streblid batflies of Panama. In R. L. Wenzel, & V. J. Tipton (Eds.), *Ectoparasites of Panama* (pp. 405−675). Chicago: Field Museum of Natural History.

Westrom, D. R., & Anderson, J. R. (1992). The distribution and seasonal abundance of deer keds (Diptera: Hippoboscidae) on Columbian black-tailed deer (*Odocoileus hemionus columbianus*) in northern California. *Bulletin of the Society for Vector Ecology, 17*, 57−69.

Wilkinsona, D. A., Duronc, O., Cordonina, C., Gomarda, Y., Ramasindrazanaa, B., Mavinguib, P., et al. (2016). The bacteriome of bat flies (Nycteribiidae) from the Malagasy region: A community shaped by host ecology, bacterial transmission mode, and host-vector specificity. *Applied and Environmental Microbiology, 82*, 1778−1788.

Yeates, D. K., Wiegmann, B. M., Courtney, G. W., Meier, R., Lambkin, C., & Pape, T. (2007). Phylogeny and systematics of Diptera: Two decades of progress and prospects. *Zootaxa, 1668*, 565−590.

Zaugg, J. L., & Coan, M. E. (1986). Test of the sheep ked *Melophagus ovinus* (L.) as a vector of *Anaplasma ovis* Lestoquard. *American Journal of Veterinary Research, 47*, 1060−1062.

Chapter 21

Moths and Butterflies (Lepidoptera)

Gary R. Mullen[1] and Jennifer M. Zaspel[2]

[1]*Department of Entomology and Plant Pathology, Auburn University, Auburn, AL, United States;* [2]*Milwaukee Public Museum, Milwaukee, WI, United States*

Many moths and butterflies are recognized as economic pests of row crops, fruit and shade trees, ornamental shrubs, and other plantings on which their larvae feed. Others are household pests that infest cereals, grains, and other stored products or attack woolen fabrics and other materials of animal origin. Adult moths also can be a nuisance because of their attraction to lights, often entering homes at night. Butterflies, however, are seldom pests. The adults are generally viewed as colorfully attractive insects that are a pleasure to see in flight or visiting flowers for nectar. However, a number of lepidopteran species, notably, moths, can cause significant health problems for humans and other animals.

In most cases of a medical-veterinary nature, it is the larval stage that is involved. The larvae of many species are armed with toxic setae or spines, which, on contact with skin, can cause a stinging or burning sensation. Under certain circumstances, stinging caterpillars may be ingested by domestic animals resulting in gastrointestinal problems. This most commonly occurs when animals such as cattle and horses graze on infested forage. Adults also can contribute to health problems. The inhalation of wing scales and body hairs of adult moths can induce allergic reactions, whereas contact with the silk of certain species can cause allergic responses in sensitized individuals. The most unusual lepidopterans from a medical-veterinary perspective are species that, as adults, feed on animal fluids. Some moths feed about the eyes, whereas others are attracted to wounds and in some cases can actually penetrate the skin of humans and other animals to feed directly on blood.

The general term **lepidopterism** is applied to adverse reactions of humans and other animals to moths and butterflies. The term also is used in a more restricted sense for cases involving only adults. Reactions to larvae are called **erucism** (L. *eruca*, caterpillar). Most cases involve **urticaria** (L. *urtica*, nettle), a vascular reaction of the skin in the form of papules or wheals, caused by contact with the specialized defensive spines or setae of certain species. On rare occasions lepidopterous larvae invade animal tissues, causing **scoleciasis** (G. *scolec*, worm).

An excellent overview of the Lepidoptera including morphology, developmental stages, behavior, and higher taxonomic classification is provided by Scoble (1992). For information specifically on Lepidoptera-related problems of medical-veterinary significance, the following reviews are recommended: Delgado (1978), Southcott (1978, 1983), Kawamoto and Kumada (1984), Wirtz (1984), and Plotkin and Goddard (2013).

TAXONOMY

Classification of the Lepidoptera above the superfamily level is subject to debate among taxonomists. The simple separation of the Order into two groups, moths and butterflies, is no longer phylogenetically appropriate. Nonetheless, for the purpose of this chapter, the terms "moths" and "butterflies" will be used in the generally accepted context for the species discussed. Virtually all species of medical-veterinary importance are members of the following four superfamilies: Bombycoidea, Noctuoidea, Papilionoidea, and Zygaenoidea. For details on the higher classification of the Lepidoptera, see Kristensen (1984), Nielsen and Common (1991), Scoble (1992), and Nieukerken et al. (2011).

Among the more than 100 lepidopteran families, 14 include species that as larvae cause health-related problems. They represent more than 60 genera and 100 species worldwide. Members of at least 10 families and 42 genera are known to cause medically related problems as larvae in North America. The families most commonly encountered as problems are Limacodidae, Megalopygidae, and Saturniidae. Other important families in various parts of the world are the Erebidae (i.e., Arctiinae, Lymantiinae), Lasiocampidae, and Noctuidae.

Seven families of Lepidoptera that include species in which adults feed on animal wounds and various body secretions are Geometridae, Erebidae, Noctuidae, Notodontidae, Crambidae, Sphingidae, and Drepanidae (subfamily Thyatirinae). The species most commonly observed

Medical and Veterinary Entomology. https://doi.org/10.1016/B978-0-12-814043-7.00021-2

feeding on animals are *Lobocraspis griseifusa* and *Arcyophora* spp. (Nolidae), *Hypochrosis* spp. (Geometridae), *Filodes*, and *Microstega* spp. (Crambidae). The only species that are known to be capable of piercing vertebrate skin are members of the erebid genus *Calyptra*, in Southeast Asia.

MORPHOLOGY

Adult moths and butterflies are easily recognized by their scale-covered wings, wing venation, and long coiled proboscis, or feeding tube. The proboscis serves primarily as a means of imbibing fluids such as nectar, fruit juices, honeydew, and water (Fig. 21.1A). The process is facilitated by modifications of the inner surface of the distal portion of the proboscis to form a brush-like tip that significantly increases capillary action while feeding (Fig. 21.1B and C). A pharyngeal sucking-pump then draws liquids up through a porous channel formed by the tightly interlocked pair of elongate maxillae, each of which bears a longitudinal groove along its inner surface (Lehnert et al., 2017), before passing into the alimentary tract. In species that as adults feed on wounds and body fluids of animals, the primary differences in their feeding mechanisms are structural modifications near the tip of the proboscis (Fig. 21.2) for stimulating lachrymation and rasping or piercing tissues.

The larvae of moths and butterflies are called caterpillars. The larva is typically cylindrical with a well-developed head capsule, three pairs of thoracic legs, and five pairs of fleshy, unsegmented prolegs, one pair each on abdominal segments 3–6 and 10 (Fig. 21.3). These larvae are called **eruciform**. Prolegs usually bear tiny hooks called **crochets**, which aid them in clinging to various surfaces while moving about. The mouthparts are adapted for chewing plant material on which the larvae feed. The general body form of larvae is highly modified in some families to the extent that they may not be recognized as lepidopteran larvae by the nonspecialist. Examples include some of the medically important taxa such as puss caterpillars and hag moths.

Caterpillars that cause dermatitis on contact with vertebrate skin are protected by specialized hairs and spines. In some cases, these structures cause simple mechanical injury or irritation when they penetrate the skin. In other cases, they have associated poison glands that secrete toxic substances that elicit varying degrees of inflammation and swelling at the contact site. The location, numbers, and types of urticating hairs and spines vary significantly among different families and genera (Fig. 21.4).

Setae and spines are produced by specialized **trichogen cells**, literally "hair-forming" cells. These cells secrete multiple layers of cuticle to form the wall structure, differentiated from the surrounding epidermis. Each spine typically articulates with a socket formed by a **tormogen cell**, or "socket-forming" cell. Setae, or hairs, are generally formed by one or a few trichogen cells, whereas the larger, more robust spines are multicellular in origin and are produced by many trichogen cells. These setae and spines usually are innervated and associated with supporting cells and, depending on the taxon, may or may not have toxin-secreting cells and tracheoles.

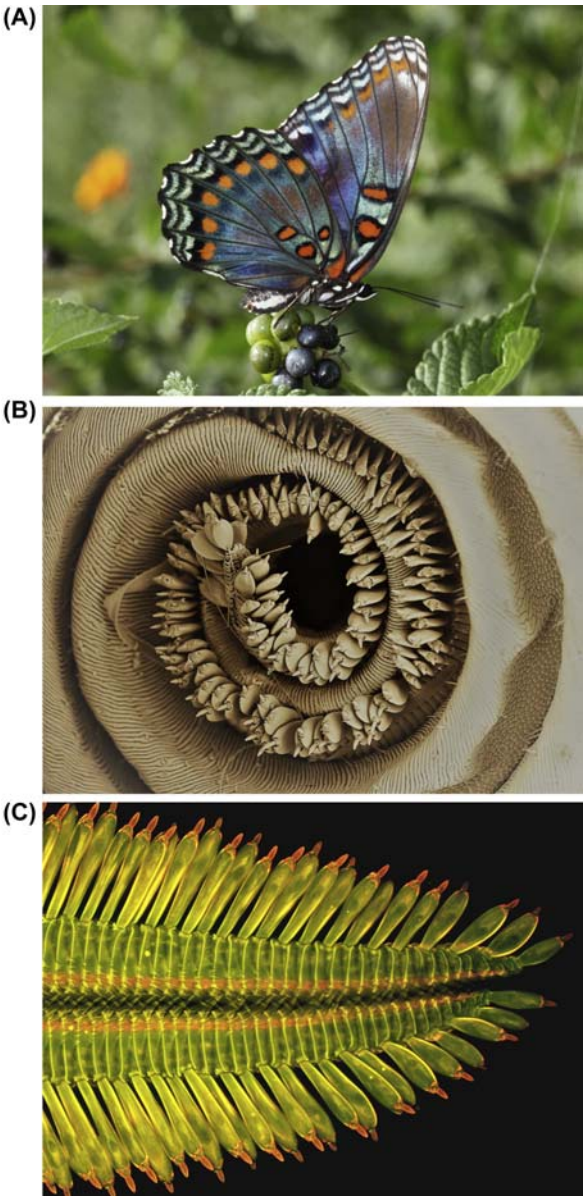


FIGURE 21.1 Red-spotted purple, *Limenitis arthemis astyanax* (Nymphalidae), a typical nectar-feeding butterfly. (A) Adult resting on lantana berries. (B) Distal portion of the coiled proboscis with elongated chemomechano receptors (sensilla styloconica) on the inner surface, which create a brushlike tip for increasing capillary action when feeding on wet surfaces; scanning electron micrograph. (C) Distal region of proboscis showing modified dorsal labial lobes (ligulae) and brushlike sensilla styloconica that provide capillary action when feeding; confocal microscope image. *(A) Photograph by John Flannery, Creative Commons. (C and D), photographs by Matthew S. Lehnert.*

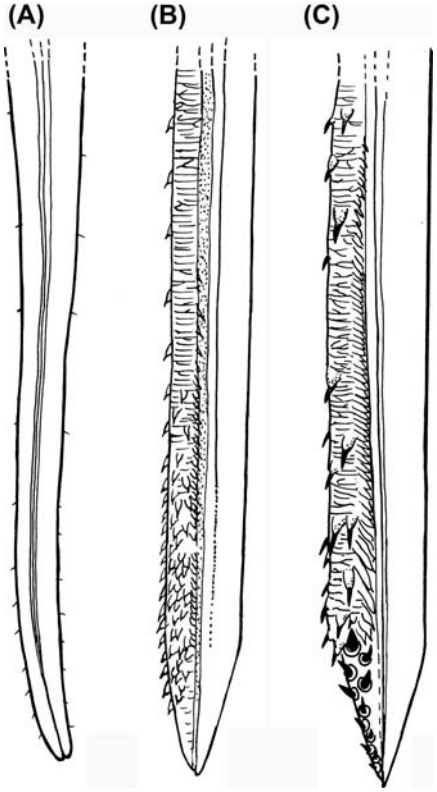


FIGURE 21.2 Modifications of the distal tip of the proboscis in fruit-piercing and skin-piercing moths. (A) Unmodified proboscis of nectar-sucking moth, *Autographa gamma* (Noctuidae). (B) Fruit-piercing moth, *Scoliopteryx libatrix* (Erebidae). (C) Skin-piercing, blood-sucking moth, *Calyptra eustrigata* (Erebidae). *Redrawn from Bänziger (1971).*

The specialized setae and spines of urticating lepidopterans are highly variable in structure. The following categories are based on Kawamoto and Kamada's (1984) modification of a classification proposed by Kano (1967, 1979), which includes types found in adults as well as larvae. According to their classification, there are two major groups of urticating structures: spicule hairs and spine hairs. **Spicule hairs** are detachable setae that are readily rubbed off or can become airborne to cause dermatitis on contact with animal skin. These hairs are easily shed and often are incorporated into the silk of the pupal cocoon. Although some of them cause only mechanical injury, others contain toxins that can affect vertebrates on contact. **Spine hairs**, on the other hand, cause urticaria only when the caterpillar makes direct contact with the skin. They often have associated poison glands and must be innervated in order for the toxins to be released. The following seven types of spicule hairs and four types of spine hairs are recognized.

Spicule Hairs

Type 1 (*Euproctis*-type). These are very small hairs (length 50–200 μm; diameter up to 5 μm), each of which has a pointed end that articulates with its own socket. The distal end has multiple, small barbs. On detaching, the pointed tip of the hair penetrates the skin. They occur as small clusters of 3–15 hairs each in cup-shaped papillae (Fig. 21.5A) on various parts of the caterpillar. Type 1 hairs are characteristic of the brown-tail moth (*Euproctis chrysorrhoea*) and other *Euproctis* spp. in the family Lymantriinae. The number of these papillae can be extremely large, as in *Euproctis similis* larvae with an estimated 600,000 spicule hairs and *E. subflava* larvae with more than six million.

Type 2 (*Thaumetopoea*-type). These spicule hairs are similar to type 1 in size and shape but are pointed at both ends. They are inserted point-downward into individual cuplike sockets and occur only in third-instar and older larvae. They are typical of the family Thaumetopoeidae. The processionary caterpillar (*Thaumetopoea processionea*) is estimated to have over 630,000 of these specialized defensive hairs.

Type 3 (*Dendrolimus*-type). These are relatively long (0.5–1.0 mm), slender spicule hairs, with blunt proximal ends and sharply pointed distal tips (Fig. 21.5B). These are loosely articulated with individual sockets and are easily broken off. In addition, to mechanical injury to the skin, they can cause localized reactions upon discharge of toxin when the spicule wall is broken. These urticating hairs occur in adults of the Lasiocampidae, notably, on the mesothoracic and metathorax of the genus *Dendrolimus*.

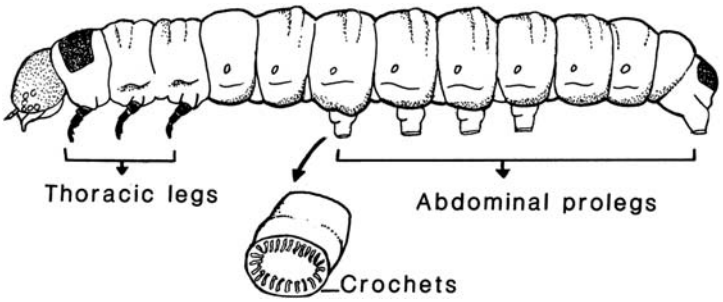


FIGURE 21.3 External morphology of typical lepidopteran larva, with enlargement of abdominal proleg showing tiny hooks, or crochets. *Redrawn from Romoser (1981).*

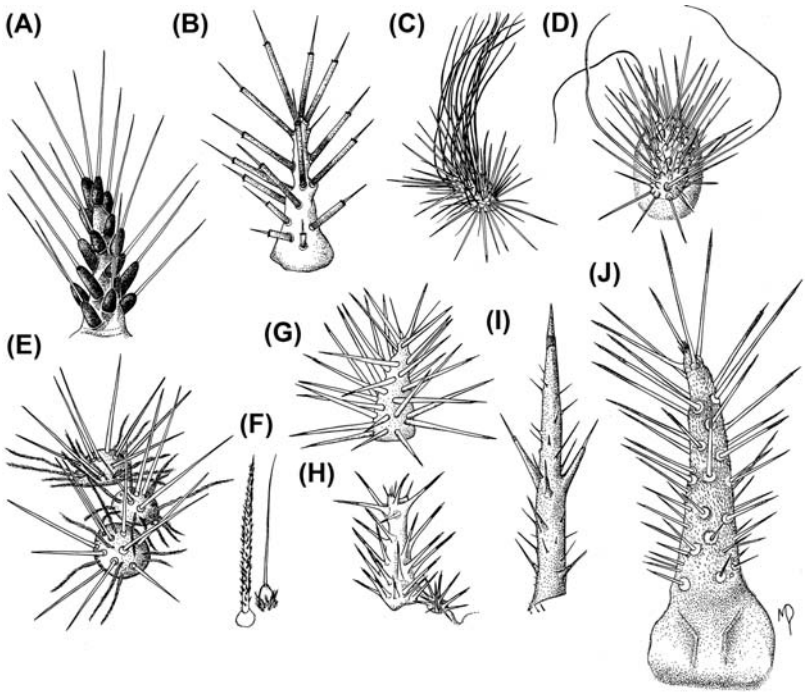


FIGURE 21.4 Urticating hairs and spines of North American caterpillars. (A) Pandora caterpillar, *Coloradia pandora* (Saturniidae). (B) Buck moth, *Hemileuca maia* (Saturniidae). (C) Puss caterpillar, *Megalopyge opercularis* (Megalopygidae). (D) White flannel moth, *Norape ovina* (Megalopygidae). (E) Smeared dagger moth, *Acronicta oblinita* (Noctuidae). (F) Hag moth, *Phobetron pithecium* (Limacodidae). (G) Io moth, *Automeris io* (Saturniidae). (H) Spiny oak-slug caterpillar, *Euclea delphinii* (Limacodidae). (I) Mourning cloak butterfly, *Nymphalis antiopa* (Nymphalidae). (J) Saddleback caterpillar, *Sibine stimulea* (Limacodidae). *Original by Margo Duncan.*

Type 4 (*Latoia*-type). These spicule hairs are relatively short (0.5−1.0 mm) and stout with a pointed distal tip and three or four basal barbs. They are multicellular in origin, typically having a large poison-secreting cell surrounded by supporting cells. They are characteristic of slug caterpillars of the genus *Latoia*, family Limacodidae, where they occur on the ninth segment of the last larval instar.

Type 5 (Starlike hair). These highly specialized urticating hairs are very small, compact, rhomboid-shaped structures (Fig. 21.5C), each produced by a single trichogen cell. Projecting from the outer wall are tiny, pointed spikes or prickles, which cause a nettling sensation on contact with skin. These spicule hairs are characteristic of many limacodid larvae, occurring primarily in clusters on the lateral tubercles.

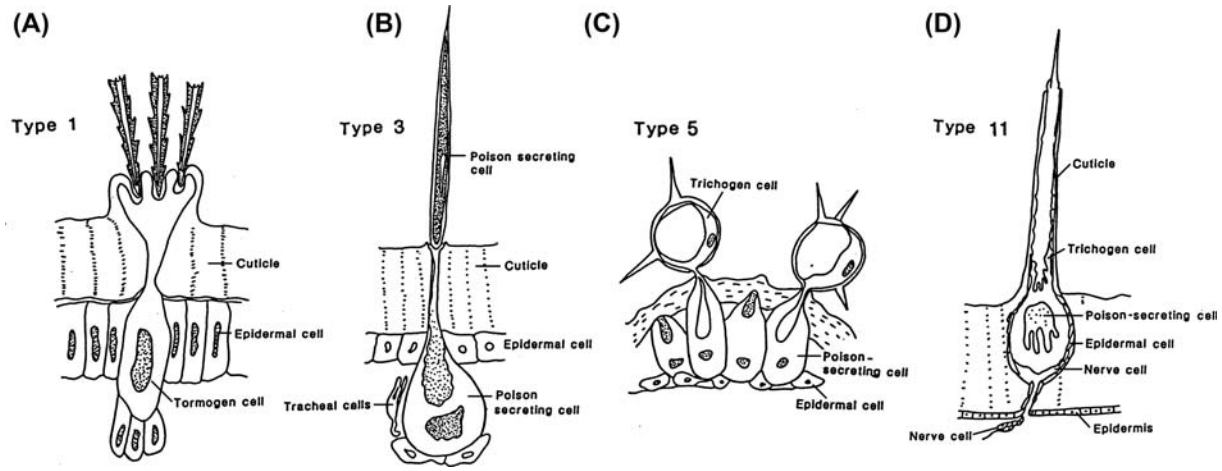


FIGURE 21.5 Representative types of spicule hairs and spine hairs in urticating caterpillars. (A) Type 1 spicule hair (*Euproctis*, Lymantriinae). (B) Type 3 spicule hair (*Dendrolimus,* Lasiocampidae). (C) Type 5 spicule hair (starlike, Limacodidae). (D) Type 11 spine hair (*Latoia*, Megalopygidae). *Redrawn and modified from Kawamoto and Kumada (1984).*

Type 6 (brush hair). These long (c. 4.0 mm), slender spicule hairs occur in dense brushlike clusters appearing as tufts on the abdominal segments. The basal end of each hair is sharply pointed with many barbed hooks. They are easily detached and commonly incorporated with the silk as larvae spin their cocoons. These urticating hairs are common in the Lymantriinae, including *Calliteara pundibunda* and the Douglas-fir tussock moth *Orygia pseudotsugata*. They also occur in the arctiine genus *Premolis* and the Australian anthelid known as the hairymarry caterpillar, *Anthela nicothoe*.

Type 7 (moth spicules). These spicule hairs occur only in adult moths. They are similar to type 2 hairs from which they differ primarily in their sockets being located in papillae. The spicule hairs themselves are very small (170 μm long, 3−5 μm diameter) with sharp tips and tiny barbs on the upper one-third of the shaft. They are characteristic of the genus *Hylesia*, family Saturniidae, and adults of several genera of the Thaumetopoeidae such as *Anaphe*, *Epanaphe*, *Epicoma*, and *Gazalina*.

Spine Hairs

Type 8 (primitive type). These are structurally similar to normal body hairs with sharply pointed tips and varying numbers of tiny barbs projecting from their surface. Dermatitis is usually limited to mechanical injury by these hairs because most species lack associated poison-producing cells. Exceptions occur in the genera *Chalcosia* and *Erasmia* (Zygaeneidae) in which poison glands are located at the base of the hairs. Type 8 spine hairs are characteristic of larvae of most species of Arctiinae and Noctuidae and of such butterflies as Nymphalidae that are known to cause urticaria.

Type 9 (simple poisonous spines). These are simple hairs similar to type 8 spine hairs except that each possesses a poison-secreting cell. Relatively little is known about the morphology of these spines despite their wide occurrence among lepidopterous larvae. They are probably the most common type of urticating spine in caterpillars and cause more severe skin reactions than simple mechanical injury.

Type 10 (*Balataea*-type). These are venomous spines characterized by a bulbous base containing the poison reservoir. The spines are multicellular in origin involving several trichogen cells, the poison cell, supporting cells, and a nerve cell. Toxin secreted by microvilli of the associated poison cell passes into the reservoir via a small duct. Apparently there is no opening at the tip of the spine; the toxin is released only when the tip of the spine breaks off or when the spines are broken upon penetrating the skin. Type 10 spines typically are found in caterpillars of the zygaeneid genera *Balataea* and *Illiberis*, the arctiine genera *Eilema* and *Lithosia*, and first-instar larvae of the gypsy moth *Lymantria dispar* (Lymantriinae).

Type 11 (*Latoia*-type). These spines are relatively short, stout, and conical with a bulblike base containing a large poison gland (Fig. 21.5D). There is no opening at the pointed tip. The structure of type 11 spine hairs is more complex than the others, involving not only multiple trichogen cells and poison-secreting cells but also tracheal and nerve cells. The distinguishing feature is the presence of a diaphragm against which the poison cell rests. When mechanically stimulated on contact with skin, the pressure of hemolymph acting on the diaphragm causes toxin to be ejected from the tip of each broken spine. The result can be some of the most severe dermal reactions associated with urticating caterpillars. Type 11 spine hairs occur most commonly in the Limacodidae, best represented by *Latoia* larvae. Other limacodid genera with this type of spine are *Microleon*, *Monema*, *Parasa*, and *Scopelodes*. Recent evidence suggests this condition originated once in the family and represents a derived trait (Zaspel et al., 2016). Similar urticarial spines are found in larvae of *Automeris io* and *Dirphia* (Saturniidae), *Catocala* (Erebidae), and the flannel-moth genera *Doratifera* and *Megalopyge* (Megalopygidae).

LIFE HISTORY

Moths and butterflies undergo holometabolous development. Adults typically deposit their eggs, either singly or in clusters, on host plants that serve as food for the larvae. Soon after egg hatch the larvae begin feeding. They grow rapidly, molting three to 10 times, depending on the species, as they consume increasing quantities of foliage or other plant material. When time for pupation nears, most species leave the host plant, crawling or descending on a strand of silk to the ground where they seek protected recesses in which to pupate. Others remain on the host plant. The larvae of most moths spin a protective silken cocoon in which pupation takes place. Cocoons may be attached to twigs and leaves or constructed under tree bark, under rocks or ground debris, in the soil or litter, and in other suitable sites. Larvae of moths that do not spin cocoons generally pupate in protected recesses or chambers that they excavate in the soil. The larvae of butterflies do not spin cocoons. Instead they transform into a naked pupa, or chrysalis, which usually hangs exposed on the host plant. Although most chrysalides are green, brown, or otherwise cryptically camouflaged, some are attractively colored or ornamented. Most lepidopterans produce one generation per year, with a few taking 2 or more years to complete their development. Overwintering usually occurs in the egg or pupal stage.

BEHAVIOR AND ECOLOGY

Only a few groups of moths have become adapted for feeding directly on secretions and body fluids of live animals. This behavior is likely derived from the commonly

observed habit of adult lepidopterans feeding on animal excretory products such as fecal material, urine-contaminated substrates, and animal secretions like saliva and nasal mucous smeared on vegetation or other objects. Imbibing fluids from wounds and the eyes of host animals is a behavioral modification requiring relatively minor morphological changes. In those species that feed on eye secretions, the proboscis is frequently moved over the sensitive eye region to irritate the conjunctiva and increase the flow of tears on which the moths feed. At least one species, *Hemiceratoides hieroglyphica* has been documented feeding on the tears of birds (Hilgartner et al., 2007). A related lineage, *Calyptra*, includes species that have the ability to pierce mammalian skin to feed directly on blood (Fig. 21.23). It has developed unique modifications of the proboscis, notably, tearing hooks and erectile barbs (Fig. 21.24). These are movable by internal hemolymph pressure that, aided by special proboscis motions and head musculature, enable the proboscis to penetrate vertebrate tissues.

Moths that are attracted to animals are called **zoophilous**. They have been observed feeding on the following animal fluids: lachrymal secretions from eyes, blood and skin exudates associated with wounds, nasal secretions, saliva, perspiration, urine, and droplets of blood extruded from the anus of mosquitoes feeding on host animals. Because lepidopteran adults, with few exceptions, lack proteinases and therefore cannot digest proteins from these fluids, this specialized feeding behavior is believed to serve primarily as a means of obtaining salts. The feeding time for most zoophilous species is usually a few minutes but in cases blood-feeding can be up to 20 min.

Species that feed about the eyes on tears from the lachrymal glands are called **lachryphagous**. They also are called tear drinkers and eye-frequenting moths. Lachryphagy is the most striking zoophilous behavior, as documented by Bänziger in Southeast Asia. Since the first observation in 1904 of a moth feeding on the eyes of a horse in Paraguay (Shannon, 1928), lachryphagous moths have been reported attacking a wide range of wild and domestic animals, particularly ungulates and elephants. Zebu and water buffalo tend to be particularly favored hosts; other common hosts include horses, mules, tapirs, rhinoceroses, kangaroos, deer, and humans. It appears that many species exhibit a fairly high degree of host specificity at the order level.

Most lachryphagous species settle on the host to feed; others tend to hover or continually flutter their wings, remaining partially airborne while feeding. The proboscis is used to feed on tears flowing from the eyes. In some cases, it is used to irritate the conjunctiva or cornea and to feed directly on the eye tissue itself. Many lachryphagous moths are capable of slipping the proboscis between the closed eyelids of sleeping or dozing animals. Some even continue to feed when the host animal tightly closes the eyelids or blinks as a defensive response to the associated irritation. Other moths may irritate eye tissues, particularly the sensitive inner surface of the lids, with their tarsal claws while they attempt to feed.

Some adult moths are attracted to open wounds where they imbibe blood and other host fluids. In most cases the feeding behavior is similar to drinking water and other fluids from the surface of mud, fresh animal feces, or honeydew. In other cases, the moths actually probe the wound, penetrating damaged tissues to feed on fresh blood (Fig. 21.22). They are variously called wound-feeding, hematophagous, and blood-sucking moths. As many as 10 taxa of skin-piercing moths are capable of piercing intact skin in order to feed. It is thought that the ability to pierce animal tissue and feed on blood in this group evolved from fruit piercing, an obligatory feeding behavior that both males and females of *Calyptra* exhibit. Although no specific morphological or molecular markers have been directly linked to the blood-feeding habit in these moths, recent evidence suggests hematophagous males may have a reduction in olfactory sensilla sensitive to animal odors (Hill et al., 2010).

Urticating Caterpillars

The three more important lepidopteran families with stinging caterpillars are Limacodidae, Megalopygidae, and Saturniidae. Other families include Erebidae (subfamilies: Lymantriinae and Arctiinae), Lasiocampidae, Noctuidae, Thaumetopeidae, and Nymphalidae. With the exception of the last family, all are moths as adults.

Megalopygidae

Members of this family are called **flannel moths**, referring to the densely hairy larvae and adults. They occur in the Palearctic and Nearctic regions but especially in South America and the West Indies where the urticating larvae are known as *tataranas* ("like fire"), *cuy machucuy*, and **fire caterpillars**. All megalopygid larvae are protected by poisonous spines concealed beneath the more conspicuous long, fine hairs. These are generally type 11 spine hairs that cause a nettling sensation on contact with skin. Some species can cause particularly severe reactions and present occupational hazards for tree-plantation workers in parts of South America.

Among the 11 species in North America, the **southern flannel moth** (*Megalopyge opercularis*) is the most commonly encountered by humans. The larva is called a **puss caterpillar**, referring to its hairy appearance (Fig. 21.6). Other colloquial names include the asp, or asp caterpillar; and *gusano pollo* in South America. The dense, fine, silky hairs vary in color from tan to dark brown or charcoal gray with one or more pairs of small, dorsolateral patches of white setae. The hairs at the posterior end form a

FIGURE 21.6 Puss caterpillar, *Megalopyge opercularis* (Megalopygidae). *Photograph by Sturgis McKeever.*

tail-like tuft, whereas the head is concealed beneath the mouselike pelage. It occurs primarily in the southeastern and south central United States where the larvae feed on oaks, hackberry, persimmon, apple, orange, almond, pecan, roses, and other trees and shrubs. The short, toxic spines are arranged in radiating clusters (Fig. 21.4C) on three pairs of elevated, longitudinal ridges along the mid-dorsum and sides of the body. The tips of these spines break off on contact with the skin, releasing toxin from the bulbous cavity at their base. All instars are capable of stinging.

The puss caterpillar causes the most painful and severe reactions among urticating species in the United States. Reactions typically include an initial burning sensation, commonly followed by numbness and occasional localized swelling, nausea, and vomiting. Reddened blotches or mottling develop at the contact site, often associated with a glistening appearance as cell fluids are released at the skin surface. Edema may occur, especially if the wrist or lower arm is involved; in such cases, the entire limb from hand to shoulder may become swollen. This is often accompanied by inflammation of lymphatic vessels and dull, throbbing aches involving the axillary nodes that may persist for 12 h or longer. Stings on the neck can be particularly severe. Occasionally, when populations of *M. opercularis* are unusually high, large numbers of people may be affected. Two such instances have occurred in Texas (USA). One involved hundreds of children that resulted in the closing of public schools (Bishopp, 1923); the other was a widespread outbreak in which more than 2,100 cases were reported (Keegan, 1963).

Other megalopygid species that as larvae cause urticaria in North America are the **crinkled** or **black-waved flannel moth** (*Lagoa crispata*), **yellow flannel moth** (*Lagoa pyxidifera*), and **white flannel moth** (*Norape ovina*) (Fig. 21.7).

Limacodidae (Cochlidiidae, Eucleidae)

This is a large, mostly tropical and subtropical family of moths that occurs widely throughout the Neotropical,

FIGURE 21.7 White-flannel moth, *Norape ovina* (Megalopygidae). *Photograph by Sturgis McKeever.*

Ethiopian, Indo-Australian, and Palearctic regions. Approximately 50 species occur in North America. The larvae are called **slug caterpillars** or **nettle grubs**, referring to their unusual shape and the fact that most species have stinging spicule hairs. The larvae are usually somewhat flattened or sluglike, with a small retractable head, short thoracic legs, and reduced abdominal prolegs that are modified as suckers. They move in a gliding motion, suggestive of slugs. The poisonous setae are usually type 4 or type 5 spicule hairs, often in the form of starlike clusters of prickles borne on cone-shaped protuberances; the spines break off easily on contact to cause a nettling sensation.

Six species of urticating slug caterpillars occur in North America. The most commonly encountered is the **saddleback caterpillar** (*Sibine stimulea*). It is easily recognized by a dorsal, brown oval spot with a white border, in turn surrounded by a green area suggesting a saddle and saddle blanket (Fig. 21.8). Urticating hairs are borne on two pairs of large, dark brown, fleshy protuberances (Fig. 21.4J), one pair at each end, and on smaller prominences along the sides. In addition, two pairs of rounded lobes bearing specialized, deciduous setae called **calytropes**, which cause irritation to skin, are located at the caudal end. The stinging reaction consists of a burning

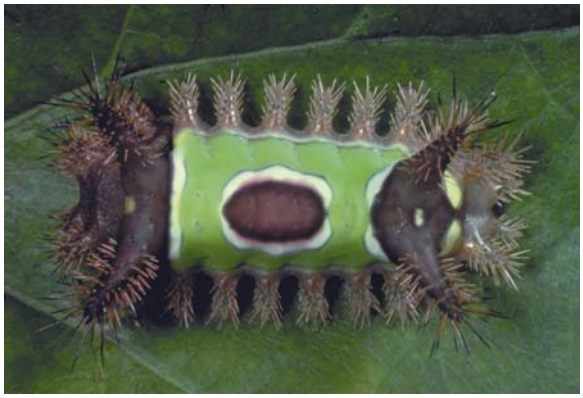


FIGURE 21.8 Saddleback caterpillar, *Sibine stimulea* (Limacodidae). *Photograph by Sturgis McKeever.*

sensation and erythematous lesion which is usually much less severe than that of the puss caterpillar. The saddleback caterpillar is found on oaks, elms, dogwoods, linden, corn, ixora, asters, blueberries, grapes, and a number of fruit trees such as apple, citrus, pear, plum, and banana.

Larvae of the **hag moth** (*Phobetron pithecium*) are sometimes called **monkey slugs**. Their unkempt, haglike appearance is attributed to their lateral fleshy processes of variable lengths that are covered with fine, brown or grayish, plumose hairs (Fig. 21.9). The relatively few, tuberculate stinging hairs (Fig. 21.4F) are located at the tips of the processes and laterally on each segment. Contrary to some reports in the literature, the urticarial reaction to hag moth larvae is usually mild, at most. The larvae feed on ashes, birches, hickories, oaks, chestnut, willows, apple, and persimmon.

Another urticating species is the **spiny oak-slug caterpillar** (*Euclea delphinii*) in the eastern United States (Fig. 21.10). The yellow-green larvae feed on a variety of woody plants including beech, cherry, maple, oak, redbud, sycamore, and willow. In addition, to urticating spines on the dorsal and lateral processes (Fig. 21.4H), they possess a pair of caudal patches of densely clustered, brown, deciduous setae (calytropes), which can be shed defensively causing skin irritation. These specialized setae also are incorporated into the silk used in spinning the cocoon, providing protection for the developing pupa.

Two closely related limacodid species with urticating larvae are the **stinging rose caterpillar** (*Parasa indetermina*) (Fig. 21.11) and the **smaller parasa** (*Parasa chloris*) (Fig. 21.12). *Parasa indetermina* is found on oaks, hickories, maples, poplars, apple, dogwood, and roses, whereas *P. chloris* occurs most commonly on apple, dogwood, elms, and oaks. Less commonly encountered are the stinging caterpillars of **Nason's slug moth** (*Natada nasoni*) (Fig. 21.13) and *Adoneta spinuloides*. *Natada nasoni* feeds on beech, hickory, hornbeam, chestnut, and oak, whereas *A. spinuloides* feeds on beech, birch, linden, willow, plum, and other trees and shrubs. *Isa textula* can cause a slight urticaria in some individuals; it is found on cherry, maples, and oaks.

FIGURE 21.10 Spiny oak-slug caterpillar, *Euclea delphinii* (Limacodidae). *Photograph by Sturgis McKeever.*

Slug caterpillars are common pests among agricultural workers, particularly in Latin America and Southeast Asia. They represent significant occupational hazards for workers in banana and rubber plantations, groves of coconut and oil palms, and other tree crops in the tropics.

Saturniidae

The members of this family are called **giant silk moths** and include some of the largest and most colorful of all adult moths. Two North American species in particular have urticating caterpillars, the io moth and the buck moth. The **io moth** (*Automeris io*) occurs throughout the eastern United States and Canada. The larvae feed on a wide range of host plants; these include trees such as oaks, willows, maples, birches, and elms, in addition, to other plants like corn, clover, and ixora. The caterpillar is pale green and fairly stout with lateral stripes varying in color from yellow to reddish or maroon (Fig. 21.14). The stinging spines are usually yellow with black tips and are borne on fleshy

FIGURE 21.9 Hag moth, *Phobetron pithecium* (Limacodidae). *Photograph by Sturgis McKeever.*

FIGURE 21.11 Stinging rose caterpillar, *Parasa indetermina* (Limacodidae). *Photograph by Gary R. Mullen.*

FIGURE 21.12 Smaller parasa, *Parasa chloris* (Limacodidae). *Photograph by M. E. Epstein.*

tubercles along the back and sides (Fig. 21.4G). The sharply pointed tips break off easily on contact, allowing the toxin to penetrate the skin. Other *Automeris* spp. cause urticaria in South America, especially in Brazil and Peru.

The **buck moth** (*Hemileuca maia*) caterpillar is dark, sometimes almost black, with conspicuous black spines borne on dorsal and lateral tubercles (Figs. 21.4B and 21.15). Contact with the skin causes an immediate nettling sensation, often followed by local puffiness. The histamine-induced edema resulting from punctures of the skin by individual spines commonly coalesces to form pronounced wheals. This species occurs in the central and eastern United States, where it feeds most commonly on oaks. The adults are unusual in being active fliers during the daytime. Other North American *Hemileuca* spp. with urticating caterpillars are the **New England buck moth** (*H. lucina)*, **Nevada buck moth** (*H. nevadensis*), and *H. oliviae*.

In South America, two species of saturniids are involved in urticarial cases: *Lonomia achelous* in Venezuela and other northern countries and *L. oblique* (Fig. 21.16), particularly in southern Brazil. The larvae are gregarious, feeding at night in trees and moving to the trunk or lower branches during the day. Their toxin contains a potent proteolytic

FIGURE 21.14 Io moth, *Automeris io* (Saturniidae). *Photograph by Sturgis McKeever.*

enzyme that breaks down fibrinogen in human blood, interfering with its ability to clot. Dermal contact with *Lonomia* larvae causes an immediate burning sensation, often followed within hours by generalized discomfort, weakness, and headache. This is accompanied by hemorrhaging of capillaries near the skin surface (ecchymosis). In severe or untreated cases, there may be profuse bleeding from the nose, ears, intestinal tract, and vagina within 2−10 days after onset of symptoms. Fatalities have been documented, involving cerebral hemorrhages and kidney failure. An antivenin has been developed for *A. oblique*.

Other saturniid species that as larvae cause urticaria are *Dirphia multicolor* and *D. sabina* in Brazil and Peru.

Erebidae

Subfamily Lymantriinae

Members of this subfamily are called **tussock moths**. The common name refers to the larvae, which are typically hairy with prominent tufts of hairs, or tussocks, on the back. The family occurs throughout the world but mainly in the Nearctic, Palearctic, and Indo-Australian regions. A relatively few species possess urticating hairs. When present they occur as modified, simple hairs arising from

FIGURE 21.13 Nason's slug moth, *Nadada nasoni* (Limacodidae). *Photograph by Jerry F. Butler.*

FIGURE 21.15 Buck moth, *Hemileuca maia* (Saturniidae). *Photograph by Lacy L. Hyche.*

FIGURE 21.16 *Lonomia oblique* (Saturniidae), larval aggregation on tree trunk, Brazil. *Photograph by Germano Woehl, Jr.*

cuticular cups clustered in groups at the base of the tussocks. The two most common species that cause urticaria in North America are the brown-tail moth and the gypsy moth. Both are introduced species from Europe that have become serious forest pests in the northeastern United States and the maritime provinces of Canada.

The **brown-tail moth** (*Euproctis chrysorrhoea*) is so called because of the brownish tip of the abdomen that contrasts sharply with the otherwise white body of the adult. It was introduced from Europe into Massachusetts in 1897, where it became a major defoliator of New England woodlands. The larvae feed on many species of trees and shrubs, especially members of the rose family. Host plants include apple, pear, plum, cherry, hawthorns, oaks, willows, bayberry, and roses. The larva has a light-brown head and dark-brown, almost black, body with broken lines on either side; two prominent dorsal red spots are located near the posterior end. Numerous tubercles on the back and sides bear long, barbed setae with shorter, brown setae in between. Barbed, stinging setae are borne on tubercles amidst the longer body hairs. These specialized hairs break off easily and cause a nettling sensation when they contact skin. They also are incorporated into the silken cocoon that the larva spins, thereby protecting the pupa from potential enemies. The adult female possesses similarly, specialized hairs on her abdomen, which she uses to cover her egg masses while ovipositing. Thus, all developmental stages of the brown-tail moth are provided with stinging hairs that can cause urticaria in humans and other animals.

The **gypsy moth** (*Lymantria dispar*) is similar in many respects to the brown-tail moth. Following its introduction to the United States from Europe about 1868, in an unsuccessful effort to use this species for developing a silk industry in Massachusetts, the gypsy moth escaped and became established in New England. It since has spread widely throughout much of northeastern and Great Lakes area of the United States. The larvae feed on a wide range of deciduous trees, causing extensive damage when their populations are high. They prefer oaks but also attack apple, basswood, alder, birches, boxelder, poplars, willows, hazelnut, mountain-ash, sumac, witch-hazel, and roses. The larvae are quite hairy, with a pair of blue tubercles on each of the thoracic and first two abdominal segments; a pair of red tubercles are present on the next six abdominal segments (Fig. 21.17).

Gypsy moth larvae possess two types of defensive setae. One causes irritation to the skin primarily due to mechanical damage by tiny projections on the long, slender shaft of each seta. The other type is represented by shorter, smoothly tapered setae that arise from a ball-in-socket joint; they are connected with poison glands that apparently produce histamine. Reactions to these stinging hairs vary from mild to moderately severe pruritus, with accompanying erythema and papule formation. The onset of discomfort is usually noticed within 8−12 h after contact, often becoming more pronounced 1−2 days later. Most cases resolve in a few days or up to 2 weeks. Delayed hypersensitivity reactions sometimes result in irritation to the eyes, inflammation of the nasal passages, and shortness of breath. This is especially common in the case of airborne hairs of adult gypsy moths, or contact with clothes hanging on outdoor lines when this moth is locally abundant. A major infestation of the gypsy moth in the northeastern United States in 1981 resulted in thousands of cases of pruritic dermatitis being reported that year. Like the brown-tail moth, female gypsy moths cover their egg masses with specialized body hairs that can cause urticaria on contact with skin.

Other lymantriine species that cause urticaria in North America are the **whitemarked tussock moth** (*Orgyia leucostigma*) and the **yellow-tailed moth** or **mulberry tussock moth** (*Euproctis similis*). Wind-dispersed hairs of *E. similis* resulted in an estimated 500,000 human cases of pruritic dermatitis in Shanghai, China, in 1981. A similar outbreak involving the **Oriental tussock moth** (*Euproctis flava*) affected more than 200,000 people in Japan in 1955. The airborne setae are believed to have originated from larval hairs woven into the cocoons that adhered to the adult moths as they emerged. The **pale tussock moth** (*Dasychira pudibunda*) has been reported as the cause of "hop dermatitis" in Europe.

Subfamily Arctiinae

The adults of this subfamily are known as **tiger moths**. The larvae usually are covered with fairly dense hairs of varying

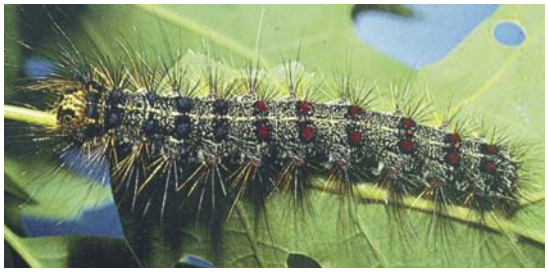


FIGURE 21.17 Gypsy moth, *Lymantria dispar* (Lymantriinae). *Courtesy of U.S. Department of Agriculture, Forest Service.*

colors arising from raised warts, in contrast to the normally bare, shiny head. Included in this group is the familiar "wooly bear" caterpillar of the Isabella tiger moth (*Pyrrharctia isabella*), which, like most arctiines, does not possess urticating hairs or spines. In larvae that cause skin irritation, type 8 spicule hairs are borne on dorsal tufts, partially concealed by the longer body hairs. Members of the following six genera in North America include urticating caterpillars: *Adolia*, *Callimorpha*, *Euchaetes*, *Halysidota*, *Lophocampa*, and *Parasemia*. Most of these species are relatively uncommon and only occasionally involved in urticarial cases. However, larvae of the **hickory halysidota** (*Halysidota caryae*), a common species on hickory, pecan, and walnut trees in the United States, have been responsible for a significant number of cases of mild urticaria, particularly in children. The **milkweed moth** (*Euchaetes egle*) is perhaps the best known. Its larvae are common on milkweed and are distinguished by dense tufts of black, yellow, and white hairs.

Although the family is cosmopolitan, the largest diversity of arctiine moths occurs in the Neotropical and Oriental regions. Where abundant, they can cause occupational erucism among field workers, as in the case of *Premolis semirrufa* in South America.

Subfamily Catocalinae

Moths in the genus *Catacola* are commonly referred to as underwing moths due to their relatively drab fore wings and brightly colored hindwings. Caterpillars of many species feed on the new leaves of woody plants such as oak, walnut, and willow (Wagner, 2005). Larvae of several species of **underwing moths** (*Catocala* spp.) are protected by stinging hairs.

Lasiocampidae

Members of this family are commonly called **tent caterpillars** or **lappet moths**. The larvae are usually very hairy, often colorful with longitudinal stripes. In the case of lappet moths, the larvae are somewhat flattened with hair-covered, fleshy lobes (lappets) on the sides of each segment. They are typically gregarious, forming communal silken webs or "tents" in trees for protection from natural enemies. Their specialized defensive hairs cause only mild, transient discomfort on contact with skin. Urticating hairs are used for strengthening and protecting cocoons and represent another source of contact for humans.

Lasiocampid larvae of only a few North American species reportedly possess urticating hairs. These include the **eastern tent caterpillar** (*Malacosoma americanum*) commonly found on apple and cherry trees, and two lappet moths of the genus *Tolype*: the **large tolype** (*T. velleda*) on apple, oak, ash, elm, birch, plum, and other trees, and the **small tolype** (*T. notialis*) on conifers.

Several reports in the older literature refer to the larvae of *Bombyx* spp. (family Bombycidae) causing urticaria in humans. These include species in Great Britain (Sharp, 1885; Jenkyns, 1886; and Long, 1886) and in India, Sri Lanka, and Africa (Castellani and Chalmers, 1913). The species reported in Great Britain as *Bombyx rubi* and *B. quercus* are now recognized as the lasiocampids *Macrothylacia rubi* and *Lasiocampus quercus*. Both are said to cause a nettling sensation and small white blisters in some individuals who handle them. The likelihood is that other old reports of bombycid larvae causing urticaria also refer to lasiocampid species.

Noctuidae

Adults in this family are known as **owlet moths** or simply noctuids. Only a relatively few species are known to possess stinging hairs or spines. Spine hairs are type 8 with sharp tips that break off and penetrate the skin; the spines may be branched and sometimes form brushes. Urticating species are primarily members of the subfamily Acronictinae (dagger moths). The larvae of dagger moths are commonly covered with tufts of long hairs, superficially resembling the Arctiinae.

In North America only larvae of the genus *Acronicta* cause urticaria. The **smeared dagger moth** (*Acronicta oblinita*), the larva of which is known as the **smartweed caterpillar**, is a pest of apple and other fruit trees; in addition, it is found on elms, oaks, pines, willows, cotton, corn, clover, smartweed, strawberry, and grasses. The **cottonwood dagger moth** (*Acronicta lepusculina*) feeds on cottonwoods, poplars, aspens, birches, and willows.

Nolidae

Larvae of the gum-leaf skeletonizer (*Uraba lugens*), a pest of *Eucalyptus* forests in Australia, causes classic urticaria with associated itching and wheals. This species was introduced to New Zealand in 1992, where it is now established in the Auckland region.

Thaumetopoeidae

The larvae of this family are best known as **processionary caterpillars**. They typically live in communal webs that they leave at night to feed on foliage. When moving, they crawl one behind the other, forming rows or columns, and marching in an orderly fashion. They occur primarily in the Palearctic, Asian, Ethiopian, and Australian regions where they feed on pines and oaks, and less commonly on cedars and walnut. No processionary caterpillars occur in North America. The urticating hairs of their caterpillars are type 2 spicule hairs similar to those found in the caudal tufts of adult females. Toxin is drawn by capillary action into the tip of the seta from tiny glands in the epidermis. In adults,

contractions of the abdomen are sufficient to release these hairs from their sockets. Pruritic dermatitis and urticaria can result from contact with the larvae, airborne setae from the adults, or contact with egg masses in which the barbed hairs from the adult female have been incorporated as a protective covering.

The **oak processionary** (*Thaumetopoea processionea*) is widely distributed in Europe where it commonly causes urticaria. The larva is covered with long whitish hairs arising from reddish warts, contrasted against a blue-gray coloration above the line of the spiracles and a greenish gray below; velvety black, dorsal patches occur on most of the abdominal segments. The larvae feed primarily on oaks and sometimes on walnut. Other processionary caterpillars that cause discomforting rashes include *Anaphe infracta* in Europe, *Thaumetopoea wilkinsoni*, and several *Thaumetopoea* species in Africa and Madagascar.

Nymphalidae

The only nymphalid butterfly in the Nearctic region in which the larva possesses urticating hairs is the **mourningcloak** (*Nymphalis antiopa*). It is an introduced species from Europe that occurs throughout the eastern United States and Canada. The larvae are velvety black, speckled with tiny white dots, with a row of mid-dorsal red spots and several rows of long, branched spines (Fig. 21.18) that are capable of piercing skin. The urticating structures are typical type 8 spine hairs. Caterpillars are found on elm, hackberry, poplar, willow, rose, and other common host plants. Subfamily Morphinae includes the showy, brightly iridescent-blue **morpho butterflies** that occur only in the Neotropics. The larvae of at least seven species are known to cause urticaria: *Morpho achillaena*, *M. anaxibia*, *M. cypri*, *M. hercules*, *M. laertes*, *M. Menelaus*, and *M. rhetenor* (Rotberg, 1971). Most encounters involve accidentally brushing against the larvae feeding on plants in the families Leguminosae and Menispermaceae. Cases are relatively few. They can occur any time of the year but are most commonly seen during the summer when larval populations are highest. Little is known about the nature of the urticating structures.

LACHRYPHAGOUS MOTHS

More than 100 species of zoophilous moths have been observed feeding on lachrymal secretions (Figs. 21.19 and 21.20), primarily in Thailand, Malaysia, and other parts of Southeast Asia (see Bänziger references). Most of these moths are members of the Geometridae, Crambidae, and Notodontidae, with a few species of Erebidae, Noctuidae, Sphingidae, and Drepanidae (formerly Thyatiridae).

Erebidae

Despite a relatively high diversity of species in this family (more than 20,000), instances of lachryphagy are uncommon. The most noteworthy example of tear feeding occurs in the species *Hemiceratoides hieroglyphica*, which feeds on the tears of sleeping magpie robins in Madagascar (Hilgartner et al., 2007). While feeding on the tears of nonavian reptiles has been observed in Lepidoptera, this represents the first record of tear feeding on birds. Spines near the tip of the proboscis of this moth irritate the eye tissues, possibly stimulating the flow of tears on which the adult moth feeds (Figs. 21.21). Additional morphological modifications vary significantly from other moths that feed on animal secretions yet their precise functions remain unknown (Zaspel et al., 2011).

Geometridae

Members of only a few of the 2,000 genera of geometrid moths are reportedly zoophilous. Nonetheless this family includes the largest number of lachryphagous taxa, with more than 50 species in Southeast Asia. As in other zoophilous moths, except some Noctuidae, only the males are attracted to animals. Most of them feed on mammalian body fluids which either drop to the ground or are smeared

FIGURE 21.18 Mourningcloak, *Nymphalis antiopa* (Nymphalidae). *Photograph by Sturgis McKeever.*

FIGURE 21.19 Three species of moths feeding on eye secretions of zebu: *Hypochrosis irrorata* (Geometridae), *Filodes mirificalis* (Crambidae), and *Lobocraspis griseifusa* (Nolidae). *Photograph by H. Bänziger.*

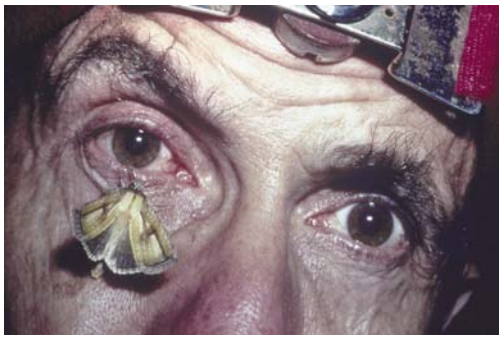


FIGURE 21.20 Tear-drinking moth, *Chaeopsestis ludovicae* (Drepanidae), feeding from human eye with tip of proboscis just inside lower eyelid. *Photograph by H. Bänziger.*

FIGURE 21.21 Distal tip of the Madagascar moth *Hemiceratoides heiroglyphica* (Erebidae), which feeds on eye secretions of birds as they sleep at night; note the harpoon-like, serrated armature that is thought to irritate the conjunctiva, stimulating the flow of tears on which the adult moth feeds. *Photograph by Jennifer M. Zaspel.*

on vegetation. They have been observed primarily in association with water buffalo, but other ungulates and elephants also are frequently visited. A few species have been reported imbibing droplets of blood extruded by mosquitoes as they feed on host animals. As a group they do not commonly frequent the eyes; however, *Hypochrosis hyadaria*, *H. flavifusata*, *Godonela eleonora*, and, to a lesser extent, other *Hypochrosis*, *Godonela*, *Scopula*, *Problepsis*, and *Zythos* spp. are among the more frequent tear drinkers. The only lachryphagous species that has been reported in the United States is the **pectinate euchlaena** or **forked euchlaena** (*Euchlaena pectinaria*) observed feeding on eye secretions of a horse in Arkansas (Selman, 1972).

Crambidae

Crambid moths are second only to the Geometridae in the number of species known to feed on lachrymal secretions. Members of the following genera are zoophilous and to various extents lachryphagous: *Botyodes*, *Epipagis*, *Hemiscopis*, *Lamprophaia*, *Pagyda*, *Pyrausta* and *Thliptoceras*. *Microstega homoculorum*, *Filodes mirificalis*, and *Paliga damastesalis* are among the more common visitors of human eyes, while *Thliptoceras* and *Hemiscopis* tend to imbibe human perspiration. Typically, they have been observed feeding on lachrymal and skin secretions of ungulates and elephants.

Notodontidae

Adult males of at least eight species of the genera *Tarsolepis*, *Togarishachia*, and *Pydnella* are lachryphagous. Elephants appear to be their preferred hosts; however, these moths feed on a wide range of other large mammals in Southeast Asia including water buffalo, zebu, tapir, rhinoceros, deer, and humans. Although they feed primarily on tears, they also have been seen imbibing saliva from around the mouth. They are persistent feeders, some causing only mild discomfort to their hosts, whereas others are very irritating.

Nolidae

Although only a few species of nolid moths are zoophilous, they are behaviorally the most advanced in terms of lachryphagy and locally can be the most frequent tear drinkers. The highly flexible proboscis is swept back and forth across the eye to induce tearing as the moth feeds. The extra length of the proboscis allows these moths to feed between the eyelids of dozing animals and reduces the risk of being dislodged by eyelid movements of wakeful hosts. Both males and females of *Arcyophora* and *Lobocraspis* species are lachryphagous and are the only known tear drinkers capable of digesting proteins contained in lachrymal fluids.

Sphingidae

Rhagastis olivacea in Thailand is the only sphingid moth confirmed as being lachryphagous. It feeds while hovering about the eyes of horses, mules, and humans. It also has been observed inserting its proboscis between the lips and into the nostrils of humans to feed on saliva and nasal secretions; the latter has been described as causing a tickling sensation. Only mild discomfort is experienced when they feed on eyes.

Drepanidae

This is a relatively small family with 122 described genera worldwide. Only a few species in the genera *Chaeopsestis* and *Neotogaria* in Thailand and China are known to be zoophilous. They tend to be avid tear drinkers on zebu, horses, and mules, although they also feed on wounds. *Chaeopsestis ludovicae* is the only drepanid known to feed on humans (Fig. 21.20). It has been observed imbibing perspiration on human skin and clothes, in addition to nasal mucous and saliva of human hosts. This moth is an aggressive feeder and can cause considerable pain due to irritation of the conjunctiva and inner surface of the eyelid with its tarsal claws. The discomfort has been likened to a grain of sand being rubbed between the eye and eyelid. Adding to the annoyance is its persistence in fluttering

about the eyes and repeated attempts to feed even when the eyelids are tightly closed.

WOUND-FEEDING AND SKIN-PIERCING MOTHS

The only lepidopterans known to be capable of piercing animal skin are members of the erebid genus *Calyptra* in Southeast Asia. Like many geometrids and other zoophilous moths, they tend to be attracted to wounds, open sores, cuts, scratches, scabs, and other skin lesions (Fig. 21.22). However, while these other moths imbibe only exposed wound fluids, *Calyptra* spp. are capable of piercing the underlying tissue to feed on blood (Fig. 21.23). Only males are hematophagous. Both males and females are believed to feed almost exclusively on fruits and are able to pierce the outer layers of ripening fruit to reach the sugar-rich juices within. Other moths closely related to *Calyptra* are fruit piercers, suggesting that blood feeding is a relatively recent development in this group, derived from fruit-piercing behavior (Zaspel et al., 2012).

A number of *Calyptra* spp. has been observed piercing mammalian skin under natural conditions. Five *Calyptra* spp. are known to feed on humans: *C. bicolor*, *C. fasciata*, *C. ophideroides*, *C. parva*, and *C. pseudobicolor*. *Calyptra* spp. also have been observed piercing the skin of elephants, water buffalo, zebu, Malayan tapir, rhinoceros, deer, antelope, mules, and pigs. Not surprisingly, they are often referred to as "vampire moths." At least three additional species have been observed piercing human skin and feeding on blood under experimental conditions: *C. eustrigata*, *C. fletcheri*, and *C. thalictri* (Figs. 21.23 and 21.24). The feeding times typically range from 3 to 15 min. The reaction to the proboscis penetrating the skin varies from being barely felt to causing locally intense pain accompanied by a burning sensation. The latter has been attributed to saliva that is introduced as the moth feeds, whereas the amount of pain is believed to depend on the number of pain receptors that are encountered by the piercing stylets. Other associated reactions include localized swelling that may persist for several hours, slight numbness or itching, pressure sensitivity at the bite site, and mild induration the following day.

FIGURE 21.22 Two wound-feeding moths, *Hypochrosis pyrrhophaeata* and *Zythos* sp. (Geometridae), feeding at site of host injury. *Photograph by H. Bänziger.*

FIGURE 21.23 A skin-piercing moth, *Calyptra thalictri* (Erebidae), feeding on human blood at the base of the fingernail on thumb, Vladivoskok, Russia. *Photograph by Jennifer M. Zaspel.*

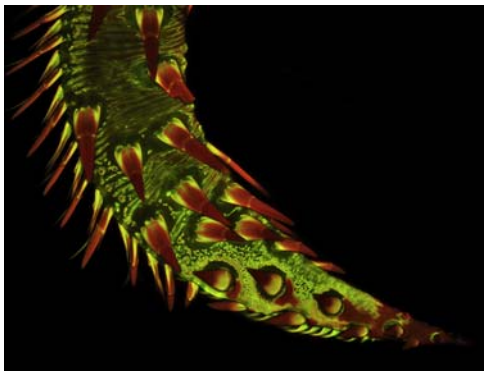


FIGURE 21.24 Distal tip of the proboscis of *Calyptra thalictri* (Erebidae); lateral view. Note the erectile barbs and tearing hooks that are adapted for piercing fruit and animal tissues; confocal microscope image. *Photograph by Matthew S. Lehnert.*

For further details on the biology and behavior of eye-frequenting and wound-feeding moths, see references by Bänziger and Zaspel.

PUBLIC HEALTH IMPORTANCE

The severity of reactions to **urticating caterpillars** is highly variable, depending on the species involved, degree of contact, and nature of the toxin. Toxic components commonly include histamine-like or histamine-releasing substances that cause edema and wheal formation at the site of contact; proteolytic enzymes and esterases; peptides and other substances that can increase vascular permeability, destroy blood cells, or cause local necrosis of tissues; and globulins with immunologic properties.

Many cases involve mild, localized dermatitis in the form of a nettling sensation or minor irritation, with transient puffiness or redness at the contact site. In more severe encounters, individuals often experience an intense burning sensation with associated wheal formation and persistent

erythema. Other cases may include localized numbness, formation of vesicles, nausea, vomiting, or fever. In cases such as those involving the saddleback caterpillar and puss caterpillar, the affected skin tends to glisten after several minutes as fluids from the damaged dermal cells appear on the skin surface. Without treatment, the burning sensation usually subsides within 30 min to 1 h, but may persist much longer. Radiating pains and lymphadenopathy may occur in more severe cases involving the limbs. The pain often extends proximally to the axillary or inguinal lymph nodes, sometimes with associated inflammation of the lymphatic vessels, which may be visible as reddened traces on the skin surface. Some cases result in dull, throbbing aches in the lymph nodes that can persist as long as 12–24 h. The contact site may remain discomforting and sensitive to touch for several days thereafter as the skin heals. The pattern of actual contact with the urticating spines may be evident for days, sometimes weeks.

Occupational erucism is a common occurrence in tropical countries, notably, in South America and Southeast Asia, where field workers are exposed to stinging caterpillars. In addition, to causing temporary discomfort and annoyance affecting primarily the arms and hands, chronic contact with some species (e.g., the arctiine *Premolis semirrufa* in Brazil) can result in persistent swelling and fibrous lesions of the joints of the hands and fingers. Workers in saw mills may experience dermatitis as a result of contact with urticating hairs in egg masses on the trunks of trees being cut for timber or stored at mill sites.

Reactions to adult moths usually occur as dermal irritation induced by airborne setae on contact with the skin, a condition called **moth dermatitis**. Most of the reported cases involve *Hylesia* spp. (Saturniidae). The response is similar to that caused by urticating larvae. The most commonly affected parts of the body are the face, neck, and upper limbs. As the setae are rubbed into the skin, they may release histamine-like substances that cause erythema and pruritus. The problem is further aggravated and spread by scratching and sweating, or by clothing and bedding contaminated with the irritating hairs. In severe cases, symptoms may persist for several days or longer, sometimes resulting in fever, insomnia, malaise, nausea, vomiting, or muscle spasms.

When airborne body setae or wing scales of moths are drawn into the respiratory tract, they can cause mechanical irritation and inflammation of the nasal passages, pharynx, and trachea. Individuals who become sensitized to these insect parts may develop **inhalation allergies** on subsequent exposure. Most reported cases involve *Hylesia* spp. in South America and the Caribbean region. Thousands of microscopic setae from the lateral and ventral areas at the end of the female abdomen become airborne during outbreaks of these moths. Clouds of setae may be released into the air when the moths are attracted to light at night and bump against windows and outdoor light fixtures. Several such instances involving thousands of human cases of moth dermatitis and inhalation allergies have been reported in Peru.

Another health-related problem is **silk-induced allergies**. This results from contact with the silk of certain moths, with sericin being the main allergen in the silk of *Bombyx mori* and *B. mandarina*. This can be an occupational problem among individuals involved in processing natural silk, resulting in skin irritation and conjunctivitis. Similar allergic responses are caused by contact with silk clothing and silk-filled bed quilts. In addition, inhalation of moth scales by workers in the silk industry can result in allergies, leading to coughing, allergic rhinitis, and asthma. Certain proteins present in silkworm pupae have been reported to cause sensitivity reactions in the sericulture regions of China where eating silkworm pupae is common.

The major health concern from lachryphagous moths feeding on humans is the irritation of eye tissues caused by microscopic lesions as the tip of the proboscis abrades the eyeball or inner surface of the eyelids. The most discomforting cases are caused by the tarsal claws of species that secure themselves to the eyelids while feeding. Actual damage to the eyes is usually minor, however, and generally heals without becoming infected. The risk of mechanically transmitted agents is greater in the case of ocular abrasions by the tarsal claws than by the proboscis, simply because the tarsi are more apt to become contaminated with infectious agents. They come in contact with a wider array of animal substances and potential pathogens. Nonetheless, there is no evidence that any human pathogen is transmitted by eye-frequenting moths.

The potential for mechanical transmission of pathogens is greatest in the case of skin-piercing moths. The relatively large size of these moths provides a greater surface area for picking up contaminants, while the deeper skin punctures and significant blood flow around the bite wound can be contributing factors. The fact that their bites are more painful than lachryphagous species increases the chances of interrupted feedings and attacks on more than one animal, enhancing prospects for transfer of pathogens. However, there is no documented evidence that any disease agent is transmitted to humans by these moths.

Treatment of urticarial cases includes the mechanical removal of urticating spines or hairs and the application of substances to alleviate the symptoms. The broken spines or hairs can be removed from the skin with fine-tipped forceps or by lightly applying sticky tape to the affected skin surface and lifting them away as the tape is removed. It is important not to rub the spines into the skin in the process of trying to remove them. They also may be removed by submerging the affected areas in warm water to float off the hairs, gently washing the skin surface with running water, or showering. Since components of the venom are generally water-soluble, water helps to dilute and destroy the toxins, thereby reducing their potency.

Other measures taken to relieve discomfort include the local application of ice packs, calamine lotion, and antihistamines. Soaking with warm bicarbonate of soda or solutions of household ammonia are generally ineffective, although relief has been reported in some cases. Mild analgesics, such as salicylic acid, do not usually provide much relief, either for the localized pain or accompanying headaches.

VETERINARY IMPORTANCE

Under certain conditions, lepidopterous species can cause veterinary problems. These usually involve the ingestion of urticating caterpillars or the caterpillars of nonurticating species that contain toxins in their body fluids. The result is irritation and inflammation of the lining of the digestive tract, variously called erucic stomatitis, erucic gastroenteritis, and erucic gastroenterocolitis. Reactions following ingestion may be immediate or delayed and range from mild to fatal. Although this type of erucism is most common in grazing cattle, horses, and other herbivores, it also has been reported causing severe stomatitis in dogs and cats. Canine and feline cases have followed ingestion of caterpillars and leaves contaminated with urticating hairs. However, severe oral lesions and tongue necrosis have been documented in dogs in the eastern Mediterranean following ingestion of larvae of the pine processionary moth (*Thaumetopoena wilkinsoni*) (Bruchim et al., 2005). In the United States, *Hemileuca maia* and other *Hemileuca* spp. have been abundant enough to pose serious threats to cattle (Caffrey, 1918). In other cases, cattle have died following ingestion of the larvae or pupae of cabbage butterflies (Pieridae) containing poisonous body fluids that can cause severe enteritis (Delgado, 1978).

Cases of urticaria in domestic animals are seldom reported but occasionally have been observed in horses. Other minor concerns are the suspected involvement of adult moths in transmitting the bacterial agent of bovine infectious keratitis, or pinkeye, in Uganda (Guilbride et al., 1959) and some moths serving as intermediate hosts for the rat tapeworm *Hymenolepis diminuta* (Belding, 1964). There also are reports of adult moths feeding on blood of chickens in coastal Ecuador where they are known by the local inhabitants as *chupa-gallina* (chicken suckers).

Wound-feeding and skin-piercing moths are not known to transmit any animal pathogens. However, their feeding can cause considerable irritation and discomfort, especially when associated moth populations are high and their attacks persistent. Most of the lachryphagous moths cause relatively mild discomfort and are generally considered more of an annoyance than a significant health problem. Nonetheless, it has been suggested that eye-frequenting moths could be vectors of trachoma virus, conjunctivitis, and pinkeye (Bänziger and Buttiker, 1969).

Caterpillar-Induced Equine Abortion

The caterpillars of certain moths have been implicated as the cause of equine abortions and stillbirths in North America and Australia. Although uncommon, high incidences of fetal losses have occurred in localized areas in years when populations of caterpillars are unusually large, leading to ingestion of the caterpillars by horses in fodder or forage. Fragments of barbed setae, which serve a defensive function in the caterpillars, penetrate the intestinal wall, and enter the bloodstream. From there they can be carried to various tissues and organs, including the developing fetuses of mares. Evidence indicates that normally nonpathogenic bacteria in the gastrointestinal tract (e.g., alpha *Streptococcus* and *Actinobacillus* spp.) are transported by the setal fragments and can infect reproductive tissues, causing inflammation of the amnion, placenta, and umbilical cord, fetal pneumonia, and death. In the United States, clinical cases are known as **mare reproductive loss syndrome**, associated with species of Lasiocampidae (*Malacosoma*) and Notodontidae (*Datana*). In Australia, the syndrome is called **equine amnionitis and fetal loss** and involves certain caterpillars of the families Thaumetopoeidae (*Ochrogaster*) and Lymantriine (*Euproctis, Leptocneria*). The abortigenic factor that initiates the syndrome in each case remains uncertain but may be a toxin in the integument and setae of the caterpillars.

The largest epidemic of equine abortion in North America occurred in 2001 and 2002, when more than 1,500 cases were reported on horse farms in Kentucky (USA). Thoroughbreds were the most affected, with estimated economic losses of US$330-500 million during the 2-year period. The cause was attributed to ingestion of the eastern tent caterpillar (*Malacosoma americanum*) (Fig. 21.25) crawling on the ground after feeding on nearby, heavily infested trees. A similar syndrome occurred in 2005 in Florida (USA) in which cases of equine abortion occurred in mares following ingestion of the walnut caterpillar (*Datana integerrima*) (Fig. 21.26) after the severe defoliation of

FIGURE 21.25 Eastern tent caterpillar, *Malacosoma americanum* (Lasiocampidae); upon ingestion by mares can cause equine abortion and stillbirth. *Photograph by Lyle Buss.*

FIGURE 21.26 Walnut caterpillar, *Datana integerrima* (Notodontidae); can cause abortion in horses following ingestion by mares. *Photograph by James Castner.*

hickory trees (*Carya*, Juglandaceae) (John F. Roberts, personal communication)

Fetal losses have been reported in Thoroughbred and Quarter horse broodmares in Australia, involving caterpillars of the following three species: processionary caterpillar (*Ochrogaster lunifer*), white cedar moth (*Leptocneria reducta*), and mistletoe brown-tail moth (*Euproctis edwardsi*). Whereas *O. lunifer* feeds on acacia and eucalypt trees, the respective host plants of *L. reducta and E. edwardsi* are reflected by their common names.

PREVENTION AND CONTROL

The greatest problems are presented for individuals working under conditions in which urticating caterpillars commonly pose an occupational hazard. The best line of defense is to recognize, and thereby avoid contact with, those species that cause urticaria and other health-related problems. Protective clothing in the form of long-sleeved shirts, pants, gloves, and suitable headwear can greatly reduce the risks involved. In the case of workers exposed to airborne setae, the use of protective eyeglasses, masks, or respirators is recommended.

To reduce the risk of exposure to *Hylesia* and other moths, lights, burning candles, and fires should be extinguished in the evening to discourage the attraction of moths. Light fixtures, windowpanes, window and door frames, and other surfaces with which the moths may come in contact should be wiped clean with a damp cloth. Clothing and bed linen should be washed daily during periods of flight activity by the adult moths. This not only helps to remove urticating setae but also destroys the water-soluble toxins. Other approaches to reducing the problem include the application of insecticides to kill *Hylesia* adults. It is important to immediately remove the dead moths in order to eliminate them as a source of more setae. Desensitization of individuals with a series of injections of moth extracts is also a consideration under certain circumstances. The resultant immunity, however, is not permanent.

No practical preventive measures are recommended for protecting animals from eye-frequenting, wound-feeding, and skin-piercing moths.

REFERENCES AND FURTHER READING

Allard, R. F., & Allard, H. A. (1958). Venomous moths and butterflies. *Journal of the Washington Academy of Sciences, 48*, 20–21.

Amarant, T., Burkhart, W., LeVine, H., III, Arocha-Pinango, C. L., & Parikh, I. (1991). Isolation and complete amino acid sequence of two fibrinolytic proteinases from the toxic Saturnid caterpillar *Lonomia achelous*. *Biochimica et Biophysica Acta, 1079*, 214–221.

Arocha-Piñango, C. L., Marval, E., & Guerrero, B. (2000). *Lonomia* genus caterpillar toxins: Biochemical aspects. *Biochimie, 82*, 937–942.

Avilán, L., Guerrero, B., Alvarez, E., & Rodriguez-Acosta, A. (2010). Description of envenomation by the "gusano-pollo" caterpillar (*Megalopyge opercularis*) in Venezuela. *Investigacion Clinica, 51*, 127–132.

Baerg, W. J. (1924). On the life history and the poison apparatus of the white flannel moth, *Lagoa crispata* Packard. *Annals of the Entomological Society of America, 17*, 403–415.

Bänziger, H. (1968). Preliminary observations on a skin-piercing blood-sucking moth (*Calyptra eustrigata*) (Hmps.) (Lep., Noctuidae)) in Malaya. *Bulletin of Entomological Research, 58*, 159–163.

Bänziger, H. (1969). The extraordinary case of the blood-sucking moth. *Animals Magazine (London)*, 135–137.

Bänziger, H. (1971). Bloodsucking moths of Malaya. *Fauna, 1*, 3–16.

Bänziger, H. (1976). In search of the blood-sucker. *Wildlife Magazine (London)*, 366–369.

Bänziger, H. (1980). Skin-piercing blood-sucking moths. III: Feeding act and piercing mechanism of *Calyptra eustrigata* (Hmps.) (Lep., Noctuidae). *Mitteilungen der Schweizerischen Entomologischen Gesellschaft, 53*, 127–142.

Bänziger, H. (1986). Skin-piercing blood-sucking moths. IV: Biological studies on adults of 4 *Calyptra* species and 2 subspecies (Lep., Noctuidae). *Mitteilungen der Schweizerischen Entomologischen Gesellschaft, 59*, 111–138.

Bänziger, H. (1987). Description of new moths which settle on man and animals in S. E. Asia (genera *Thliptoceras*, *Hemiscopis*, *Toxobotys*, Pyarlaidae, Lepid.). *Revue Suisse de Zoologie, 94*, 671–681.

Bänziger, H. (1988a). Lachryphagous Lepidoptera recorded for the first time in Indonesia (Sumatra) and Papua New Guinea. *Hetero-cera Sumatrana, 2*, 133–144.

Bänziger, H. (1988b). The heaviest tear drinkers: Ecology and systematics of new and unusual notodontid moths. *Natural History Bulletin of the Siam Society, 36*, 17–53.

Bänziger, H. (1988c). Unsuspected tear drinking and anthropophily in Thyatirid moths, with similar notes on sphingids. *Natural History Bulletin of the Siam Society, 36*, 117–133.

Bänziger, H. (1989a). A persistent tear drinker: Notodontid moth *Poncetia lacrimisaddicta* sp. n., with notes on its significance to conservation. *Natural History Bulletin of the Siam Society, 37*, 31–46.

Bänziger, H. (1989b). Skin-piercing blood-sucking moths. V. Attacks on man by 5 *Calyptra* spp. (Lepidoptera, Noctuidae) in S and SE Asia. *Mitteilungen der Schweizerischen Entomologischen Gesellschaft, 62*, 215–233.

Bänziger, H. (1992). Remarkable new cases of moths drinking human tears in Thailand (Lepidoptera: Thyatiridae, Sphingidae, Notodontidae). *Natural History Bulletin of the Siam Society, 40*, 91–102.

Bänziger, H. (1995). *Microstega homoculorum* sp. n.—the most frequently observed lachryphagous moth of man (Lepidoptera, Pyralidae: Pyraustinae). *Revue Suisse de Zoologie, 102*, 265—276.

Bänziger, H., & Fletcher, D. S. (1988). Description of five new lachryphagous and zoophilous *Semiothisa* moths from SE Asia, with five new synonymies (Lepid., Geometridae). *Revue Suisse de Zoologie, 95*, 933—952.

Bänziger, H., & Büttiker, W. (1969). Records of eye-frequenting Lepidoptera from man. *Journal of Medical Entomology, 6*, 53—58.

Belding, D. L. (1964). Order Lepidoptera. In *Textbook of parasitology* (pp. 825—826). New York: Meredith Publishing.

Bettini, S. (Ed.). (1978). *Arthropod venoms*. Berlin: Springer-Verlag, 977 pp.

Bishopp, F. C. (1923). The puss caterpillar and the effects of its sting on man. In *U.S. Dept. Agric., Dept. Circular* (Vol. 288), 14 pp.

Bruchim, Y., Ranen, E., Saragusty, J., & Aroch, I. (2005). Severe tongue necrosis associated with the pine processionary moth (*Thaumetopoena wilkinsoni*) ingestion in three dogs. *Toxicon, 45*, 443—447.

Bucherl, W., Buckley, E. E., & Deulofeu, V. (Eds.). (1971), *Venomous insects: Vol. 3. Venomous animals and their venoms*. New York: Academic Press.

Büttiker, W. (1967). First records of eye-frequenting Lepidoptera from India. *Revue Suisse de Zoologie, 74*, 389—398.

Büttiker, W., & Bezuidenhout, J. D. (1974). First records of eye-frequenting Lepidoptera from south west Africa. *Journal of the Entomological Society of Southern Africa, 37*, 73—78.

Caffrey, D. J. (1918). Notes on the poisonous urticating spines of *Hemileuca oliviae* larvae. *Journal of Economic Entomology, 11*, 363—367.

Carrijo-Carvalho, L. C., & Chudzinski-Tavassi, A. M. (2007). The venom of the *Lonomia* caterpillar: An overview. *Toxicon, 49*, 741—757.

Castellani, A., & Chalmers, A. J. (1913). *Manual of tropical medicine*. London: Baillière, Tindall and Cox.

Chan, K., Lee, A., Onell, R., Etches, W., Nahirniak, S., Bagshaw, S. M., et al. (2008). Caterpillar-induced bleeding syndrome in a returning traveler. *Canadian Medical Association Journal, 179*, 158—161.

Cheverton, R. L. (1936). Irritation caused by contact with the processionary caterpillar (larva of *Thaumetopoea wilkinsoni* Tams and its nest). *Transactions of the Royal Society of Tropical Medicine & Hygiene, 29*, 555—557.

Cock, M. J. W., Godfray, H. C. J., & Holloway, J. D. (1987). *Slug and nettle caterpillars: The biology, taxonomy and control of the Limacodidae of economic importance on palms in south-east Asia*. C.A.B. International, 270 pp.

Davidson, F. F. (1967). Biology of laboratory-reared *Megalopyge opercularis* Sm. & Abb. Morphology and histology of the stinging mechanism of the larvae. *Texas Journal of Science, 19*, 258—274.

Delgado, A. (1978). Venoms of Lepidoptera. In S. Bettini (Ed.), *Arthropod venoms* (pp. 555—611). Berlin: Springer-Verlag.

Derraik, J. (2006). Erucism in New Zealand: Exposure to gum leaf skeletonizer (*Uraba lugens*) caterpillars in the differential diagnosis of contact dermatitis in the Auckland region. *The New Zealand Medical Journal, 119*(1241), 1242—1243.

Duarte, A. C., Crusius, P. S., Pires, C. A. L., Schilling, M. A., & Fan, H. W. (1996). Intracerebral haemorrhage after contact with *Lonomia* caterpillars. *Lancet, 348*, 1033.

Eagleman, D. M. (2008). Envenomation by the asp caterpillar (*Megalopyge opercularis*). *Clinical Toxicology, 46*, 201—205.

Epstein, M. E. (1996). Revision and phylogeny of the limacodid-group families, and evolutionary studies on slug caterpillars (Lepidoptera: Zygaenoidea). *Smithsonian Contributions to Zoology, 582*.

Foot, N. C. (1922). Pathology of the dermatitis caused by *Megalopyge opercularis*, a Texan caterpillar. *Journal of Experimental Medicine, 35*, 737—753.

French, R. N., & Brillhart, D. (2015). Images in clinical Medicine: Erucism due to Lepidoptera caterpillar envenomation. *New England Journal of Medicine, 373*(18), e21.

Gilmer, P. M. (1925). A comparative study of the poison apparatus of certain lepidopterous larvae. *Annals of the Entomological Society of America, 18*, 203—239.

Gilmer, P. M. (1928). The poison and poison apparatus of the whitemarked tussock moth *Hemerocampa leucostigma* Smith and Abbot. *The Journal of Parasitology, 10*, 80—86.

Guilbride, P. D. L., Barber, L., & Kalikwani, A. M. (1959). Bovine infectious keratitis suspected moth-borne outbreak in Uganda. *Bulletin of Epizootic Diseases of Africa, 7*, 149—154.

Gusmão, H. H., Forattini, O. P., & Rotberg, A. (1961). Dermatite provocada por lepidopteros do gênero *Hylesia*. *Revista do Instituto de Medicina Tropical de Sao Paulo, 3*, 114—120.

Haddad, V., Jr., Cardoso, J. L., Lupi, O., & Tyring, S. K. (2012). Tropical dermatology: Venomous arthropods and human skin. Part I. Insecta. *Journal of the American Academy of Dermatology, 67*, 331. e1—14.

Hilgartner, R., Raoilison, M., Büttiker, W., Lees, D. C., & Krenn, H. W. (2007). Malagasy birds as hosts for eye-frequenting moths. *Biology Letters, 3*, 117—120.

Hill, S. R., Zaspel, J. M., Weller, S. J., Hansson, B. S., & Ignell, R. (2010). To be or not to be… a vampire: A matter of sensilla numbers in *Calyptra thalictri? Arthropod Structure & Development, 39*, 322—333.

Hossler, E. W. (2010). Caterpillars and moths, Parts I and II. Dermatologic manifestations of encounters with Lepidoptera. *Journal of the American Academy of Dermatology, 62*, 1—10, 13—28.

Ishizaki, T., & Nagai, S. R. (1956). Clinical studies on dermatitis due to *Euproctis flava* (Report 1). *Japanese Journal of Sanitary Zoology, 7*, 113.

Jenkyns, M. S. (1886). Urtication by *Bombyx rubi*. *Entomologist, 19*, 42.

Jones, D. L., & Miller, J. H. (1959). Pathology of the dermatitis produced by the urticating caterpillar, *Automeris io*. *American Medical Association Archives of Dermatology, 79*, 81—85.

Kagan, S. L. (1990). Inhalant allergy to arthropods: Insects, arachnids, and crustaceans. *Clinical Reviews in Allergy, 8*, 99—125.

Kalender, Y., Kalender, S., Uzunhisarcikli, M., & Ogutcu, A. (2004). Effects of *Thaumetopoea pityocampa* (Lepidoptera: Thaumetopoeidae) larvae on the degranulation of dermal mast cells in mice; an electron microscopic study. *Folia Biologica* (Kraków). *Açikgoz, 53*, 13—17.

Kano, R. (1967). Venomous Lepidoptera. *Japanese Journal of Sanitary Zoology, 18*, 170—171.

Kano, R. (1979). Lepidoptera (butterflies and moths). In M. Sasa, H. Takahasi, R. Kano, & H. Tanaka (Eds.), *Animals of medical importance in the Nansei Islands in Japan* (pp. 117—119) (Shinjuku Shobo, Japan).

Katzenellengogen, I. (1955). Caterpillar dermatitis as an occupational disease. *Dermatologica, 111*, 99—106.

Kawamoto, F., & Kumada, N. (1984). Biology and venoms of Lepidoptera. In A. J. Tu (Ed.), *Handbook of natural toxins* (pp. 291—330). New York: Dekker.

Keegan, H. L. (1963). Caterpillars and moths as public health problems. In H. L. Keegan, & W. V. Macfarlane (Eds.), *Venomous and poisonous*

animals and plants of the Pacific region (pp. 165–170). Elmsford, NY: Pergamon Press.

Kristensen, N. P. (1984). Studies on the morphology and systematics of primitive Lepidoptera (Insecta). *Steenstrupia, 10*, 141–191.

Kuspis, D. A., Rawlins, J. E., & Krenzelok, E. P. (2001). Human exposures to stinging caterpillar: *Lophocampa caryae* exposures. *American Journal of Emergency Medicine, 19*, 396.

Lamy, M., Pastureaud, M. H., Novak, F., Ducombs, G., Vincendeau, P., Maleville, J., et al. (1986). Thaumetopoein: An urticating protein from the hairs and integument of the pine processionary caterpillar (*Thaumetopoea pityocampa* Schiff., Lepidoptera, Thaumetopoeaidae). *Toxicon, 24*, 347–356.

Lehnert, M. S., Bennett, A., Reiter, K. E., Gerard, P. D., Wei, Q.-H., Byler, M., et al. (2017). Mouthpart conduit sizes of fluid feeding insects determine the ability to feed from pores. *Proceedings of the Royal Society of London. Series B: Biological Sciences (London), 284*(1846). https://doi.org/10.1098/rspb.2016.2026.

Lian, Y., & Liu, Z. (2006). Advances in silkwork pupa allergy and their allergens. *Journal of Tropical Medicine (Guangzhou), 6*, 224–226.

Long, F. R. J. (1886). Urtication by larvae of *Bombyx rubi*. *Entomologist, 19*, 45.

Lucas, T. A. (1942). Poisoning by *Megalopyge opercularis* ("Puss caterpillar"). *The Journal of the American Medical Association, 119*, 877–880.

MacKinnon, J. A., Waterman, G., Piastro, K., Oakes, J., & Pauze, D. (2015). Oropharyngeal edema in an 8-month-old girl after wooly bear caterpillar exposure. *Journal of Emergency Medicine, 49*, 147–149.

Maggi, S., & Faulhaber, G. A. (2015). *Lonomia obliqua* Walker (Lepidoptera: Saturniidae): Hemostasis implications. [Review]. *Revista da Associação Médica Brasileira, 61*, 263–268.

Moneo, I., Vega, J. M., Caballero, M. L., Vega, J., & Alday, E. (2003). Isolation and characterization of Thap 1, a major allergen from the pine processionary caterpillar *Thaumetopoea pityocampa*. *Allergy, 58*, 34–37.

Marshall, G. A. K., Jack, R. W., & Neave, S. A. (1915). A noctuid feeding on the moisture from the eyes of mules. *Proceedings of the Entomological Society of London*, 117–119.

McGovern, J. P., Barkin, G. B., McElhenney, T. R., & Wende, R. (1961). *Megalopyge opercularis*. Observations of its life history, of its sting in man, and report of an epidemic. *The Journal of the American Medical Association, 175*, 1155.

McMillan, C. W., & Durcell, W. R. (1964). Health hazard from caterpillars. *New England Journal of Medicine, 271*, 147–149.

Mills, R. G. (1923). Observations on a series of cases of dermatitis caused by a liparid moth *Euproctis flava* Bremer. *The Chinese Medical Journal, 37*, 351–371.

Murphy, S., Lill, J. T., & Epstein, M. E. (2011). Natural history of Limacodidae of theWashington, D.C., region. *Journal of the Lepidopterist Society, 65*, 137–152.

Neuedorf, F. (2007). *Caterpillars are aborting our mares* (pp. 56–58). Horse.

Neustater, B. R., Stollman, N. H., & Manten, H. D. (1996). Sting of the puss caterpillar: An unusual cause of abdominal pain. *Southern Medical Journal, 89*, 826–827.

Nielsen, E. S., & Common, I. F. (1991). Lepidoptera (moths and butterflies). In I. D. Naumann (Ed.), *The insects of Australia* (2nd ed., Vol. 2, pp. 817–915). London: Melbourne University Press, Carlton, Victoria and University College of London Press.

Niza, M. E., Ferreira, R. L., Coimbra, I. V., Guerreiro, H. M., Félix, N. M., Matos, J. M., et al. (2012). Effects of pine processionary caterpillar *Thaumetopoea pityocampa* contact in dogs: 41 cases (2002–2006). *Zoonoses Public Health, 59*, 35–38.

Paniz-Mondolfi, A. E., Pérez-Alvarez, A. M., Lundberg, U., Fornés, L., Reyes-Jaimes, O., Hernádez-Pérez, M., et al. (2011). Cutaneous lepidopterism: Dermatitis from contact with moths of *Hylesia metabus* (Cramer 1775) (Lepidoptera: Saturniidae), the causative agent of caripito itch. *International Journal of Dermatology, 50*, 535–541.

Perlman, F., Press, E., Googins, J., Malley, A., & Poareo, H. (1976). Tussockosis: Reactions to Douglas fir tussock moth. *Annals of Allergy, 36*, 302–307.

Pesce, H., & Delgado, A. (1971). Poisoning from adult moths and caterpillars. In W. Brucherl, et al. (Eds.), *Venomous animals and their venoms* (Vol. 3, pp. 119–156). New York: Academic Press.

Picarelli, Z., & Valle, J. R. (1971). Pharmacological studies of caterpillar venoms. In W. Brucherl, et al. (Eds.), *Venomous arthropods and their venoms* (Vol. 3, pp. 103–118). New York: Academic Press.

Pinto, A., Berger, M., Reck, J., Jr., Terra, R., & Guimaraes, J. (2010). *Lonomia obliqua* venom: In vivo effects and molecular aspects associated with the hemorrhagic syndrome. *Toxicon, 56*, 1103–1112.

Plotkin, D., & Goddard, J. (2013). Blood, sweat, and tears: A review of the hematophagous, sudophagous, and lachryphagous Lepidoptera. *Journal of Vector Ecology, 38*, 289–294.

Pouzot-Nevoret, C., Cambournac, M., Violé, A., Goy-Thollot, I., Bourdoiseau, G., & Barthélemy, A. (2017). Pine processionary caterpillar *Thaumetopoea pityocampa* envenomation in 109 dogs: A retrospective study. *Toxicon, 132*, 1–5.

Rodriguez-Morales, A. J., Arria, M., Rojas-Mirabel, J., Borges, E., Benitez, J. A., Herrera, M., et al. (2005). Lepidopterism due to exposure to the moth *Hylesia metabus* in northeasern Venezuela. *The American Journal of Tropical Medicine and Hygiene, 73*, 991–993.

Rotberg, A. (1971). Lepidopterism in Brazil. In W. Brucherl, et al. (Eds.), *Venomous animals and their venoms* (Vol. 3, pp. 157–168). New York: Academic Press.

Rothschild, M., Reichstein, T., von Euw, J., Aplin, R., & Harman, R. R. M. (1970). Toxic Lepidoptera. *Toxicon, 8*, 293–299.

Scoble, M. J. (1992). *The Lepidoptera: Form, function and diversity*. London: Oxford University Press, 404 pp.

Sebastian, M. M., Bernard, W. V., & Fitzgerald, T. D. (2006). *Mare reproductive loss syndrome*. Compendium: Equine Edition (Spring), 6 pp.

Selman, C. L. (1972). Observation of an eye-frequenting geometrid in the United States. *Journal of Medical Entomology, 9*, 276.

Shama, S. K., Etkind, P. H., Odell, T. M., Canada, A. T., Finn, A. M., & Soter, N. A. (1982). Gypsy-moth-caterpillar dermatitis. *The New England Journal of Medicine, 30*, 1300–1301.

Shannon, R. C. (1928). Zoophilous moths. *Science, London, 68*, 461–462.

Sharp, H. (1885). Urtication by larvae of *Bombyx rubi*. *Entomologist, 18*, 324.

Southcott, R. V. (1978). Lepidopterism in the Australian region. *Records of the Adelaide Children's Hospital, 2*, 87–173.

Southcott, R. V. (1983). Lepidoptera and skin infestations. In Parish, et al. (Eds.), *Cutaneous infestations of man and animal* (pp. 304–343). New York: Praeger, 392 pp.

Sterling, P. H. (1993). Brown-tail: The invisible itch. *Antenna, 7*, 110–113.

Stipetic, M. E., Stipetic, M., Rosen, P. B., & Borys, D. J. (1999). A retrospective analysis of 96 "asp" (*Megalopyge opercularis*)

envenomation in central Texas during 1996. *Journal of Toxicology — Clinical Toxicology, 37*, 457–462.

Tu, A. T. (1984). In , *Handbook of natural toxins: Vol. 2. Insect poisons, allergens and other invertebrate venoms*. New York: Dekker, 732 pp.

Van Nieukerken, E., Kaila, L., Kitching, I., Kristensen, N. P., Lees, D., Minet, J., et al. (2011). Order Lepidoptera Linnaeus, 1758. In Zhang (Ed.), *Zootaxa: Vol. 3148. Animal biodiversity: An outline of higher-level classification and survey of taxonomic richness* (pp. 212–221).

Webb, B. A., Dahlman, W. E., DeBorde, D. L., Weer, S. N., Williams, C., Donahue, N. M., et al. (2004). Eastern tent caterpillars (*Malacosoma americanum*) cause mare reproductive loss syndrome. *Journal of Insect Physiology, 50*, 185–193.

Wen, C. M., Ye, S. T., Zhou, L. X., & Yu, Y. (1990). Silk-induced asthma in children: A report of 64 cases. *Annals of Allergy, 65*, 375–378.

Wills, P. J., Anjana, M., Nitin, M., Varun, R., Sachidanandan, P., Jacob, T. M., et al. (2016). Populations explosion of tiger moth lead to lepidopterism mimicking infectious fever outbreaks. *PLoS One, 11*(4), e0152787.

Wirtz, R. A. (1984). Allergic and toxic reactions to non-stinging arthropods. *Annual Review of Entomology, 29*, 47–69.

Zaias, N., Ioannides, G., & Taplin, D. (1969). Dermatitis from contact with moth (genus *Hylesia*). *Journal of the American Medical Association, 207*, 525.

Zaspel, J. M., Scott, C. H., Hill, S. R., et al. (2014). Geographic distribution, phylogeny, and genetic diversity of the fruit- and blood-feeding moth *Calyptra thalictri* Borkhausen (Insecta: Lepidoptera: Erebidae). *The Journal of Parasitology, 100*, 583–591.

Zaspel, J. M., Weller, S. J., & Branham, M. A. (2011). A comparative survey of proboscis morphology and associated structures in fruit-piercing, tear-feeding and blood-sucking moths in the subfamily Calpinae (Lepidoptera: Noctuidae). *Zoomorphology, 130*, 203–225.

Zaspel, J. M., Weller, S. J., & Epstein, M. E. (2016). Origin of the hungry caterpillar: Evolution of fasting in slug moths (Lepidoptera: Limacodidae). *Molecular Phylogenetics and Evolution, 94*, 827–832.

Zaspel, J. M., Zahiri, R., Janzen, D., Hoy, M. A., Weller, S. J., & Wahlberg, N. (2012). A molecular phylogenetic analysis of the vampire moths and their fruit-piercing relatives (Lepidoptera: Erebidae: Calpinae). *Molecular Phylogenetics and Evolution, 65*, 786–791.

Chapter 22

Ants, Wasps, and Bees (Hymenoptera)

Hal C. Reed[1] and Peter J. Landolt[2]

[1]*Department of Biology and Chemistry, Oral Roberts University, Tulsa, OK, United States;* [2]*Temperate Tree Fruit and Vegetable Research, USDA ARS, Wapato, WA, United States*

A number of species of ants, bees, and wasps are pestiferous and problematic as a stinging hazard to humans throughout much of the world. They are members of the order Hymenoptera which includes non-pest species and beneficial insects (e.g., pollinating bees and parasitoid wasps) that serve as natural control agents for many insect pests. Ants and wasps, in particular, are among the foremost predators in regulating insect populations in forest communities and agroecosystems.

Many Hymenoptera species are incapable of stinging (sawflies, for example). However, the hymenopterans of most concern to human health are those that use their sting apparatus as either an offensive or a defensive weapon. These include many ants, some bees, and certain types of wasps. Even so, most of these stinging species are solitary and nonaggressive and use their sting and venom primarily to subdue prey. When these solitary species that are capable of stinging do sting, they usually cause only moderate discomfort to humans, and the pain is of short duration. Of far greater concern are the serious stings that can be inflicted by ants, bees, and wasps that are social and live in colonies. Often they exhibit defensive or aggressive behavior, and their venoms contain chemicals that cause intense pain that serve as effective deterrents against vertebrate predators. For these reasons, this chapter focuses primarily on the biology and pest status of social species of ants, bees, and wasps. In addition, commonly encountered solitary species that may be confused with more problematic ones are discussed.

TAXONOMY

Worldwide, there are more than 120,000 described hymenopteran species (Van Emden, 2013). The order is divided into two major suborders: the **Symphyta**, or sawflies, which are plant feeders, and the **Apocrita,** which normally feed on other arthropods, including bees. In the Apocrita, the abdomen is narrowly joined to the thorax ("wasp waist"), whereas in the Symphyta, the abdomen and thorax are broadly joined. The Apocrita are further divided into the **Terebrantia** (= Parasitica), which use their ovipositor for egg laying, and the **Aculeata**, which have the ovipositor modified as a sting. The 50,000−60,000 species of aculeates in the world are classified into eight superfamilies. Goulet and Huber (1993) provide a guide for identifying all families of Hymenoptera. The three superfamilies that cause most health-related problems are members of the Formicoidea (ants), Apoidea (bees), and Vespoidea (wasps).

The **Formicoidea** are divided into 20 subfamilies (AntWeb, 2014; Franks, 2009) (Table 22.1). There are approximately 13,000 described species of ants, although it is estimated that there may be as many as 15,000 species worldwide (Franks, 2009). Most ants possess a stinger, the exceptions being members of the subfamilies Formicinae and Dolichoderinae. Many of those that lack stingers (e.g., *Formica* spp.) squirt various caustic chemicals, such as formic acid, at antagonists for defensive purposes. Excellent sources of information on the taxonomy and biology of this diverse group are Hölldobler and Wilson (1990), Bolton (1994), and Fisher and Cover (2007).

Some taxonomists place all the bees, more than 20,000 species, in the family Apidae. However, we have followed bee taxonomy as presented by Michener (1974, 2007) in which bees are regarded as members of the superfamily **Apoidea** with nine families. Most of these families consist primarily of solitary or communal species that rarely sting humans. Most of the stinging bees are social species in the family **Apidae**, such as the ubiquitous honey bees (*Apis* spp.) and bumble bees (*Bombus* spp.). Some species of sweat bees (**Halictidae**) and carpenter

Medical and Veterinary Entomology. https://doi.org/10.1016/B978-0-12-814043-7.00022-4

TABLE 22.1 Subfamilies, Common Names, and Distribution of Ants (Formicidae) Based on Hölldobler and Wilson (1990)

Subfamily	Common Name(s)	Distribution	Sting
Ponerinae	Ponerine ants	Mostly tropical, worldwide	Present
Myrmeciinae	Bull-dog ants	Mostly Australia	Present
Pseudomyrmecinae	Acacia ants	Tropical Asia, Africa, America	Present
Myrmicinae	Harvester ants, leaf cutting ants, fire ants, pavement ants, pharaoh ants, others	Worldwide	Present in about half of species
Dorylinae (Old World)	Driver ants, safari ants	Asia, Africa	Present
Ecitoninae (New World)	Army ants, legionary ants	Mostly tropical America	Present
Dolichoderinae	Argentine ant and others	Worldwide	Absent
Formicinae	Thatching ants, carpenter ants, weaver ants, crazy ants, others	Worldwide	Absent

Omitted are the rare subfamilies Nothomyrmeciinae, Leptanillinae, and Aneuretinae.

bees (**Anthophoridae**) represent minor stinging threats. For further information on the phylogeny and classification of bees, see Alexander and Michener (1995), Michener et al. (1994), and Michener (2007).

Social wasps of the family **Vespidae**, represented by about 900 species worldwide (Schmidt, 2009), are the most important stinging wasps. A few species in other wasp families, including some sphecid wasps (**Sphecidae**), velvet ants (**Mutillidae**), and spider wasps (**Pompilidae**), occasionally cause stinging problems. A general reference of Hymenoptera (e.g., Gauld and Bolton, 1988; Goulet and Huber, 1993), supplemented with general textbooks on insects, should suffice for most readers wishing to identify solitary wasps to family or lower taxa. For identifying vespids in the northeastern Nearctic region, Buck et al. (2008) should be consulted. Most social vespids in North America are members of one of two subfamilies: **Vespinae**, the hornets and yellowjackets, and **Polistinae**, the paper wasps (Table 22.2). The Vespinae include the hornets—*Vespa* spp. found principally in Europe, Asia, and North Africa and *Provespa* spp. found in Southeast Asia—and the yellowjackets—*Dolichovespula* spp. and *Vespula* spp. found in temperate regions around the world. The Polistinae are further divided into four tribes: the Ropalidiini, mostly in the tropics of Africa and Asia; the Epiponini, mostly in tropical Asia, Africa, and South America; the Mischocyttarini in North, Central, and South America; and the Polistini, including the cosmopolitan paper wasps, *Polistes* spp. (Carpenter, 1999). Representatives of a third subfamily of social wasps, the Stenogastrinae, are found in much of Southeast Asia.

MORPHOLOGY

Members of the Hymenoptera range in body length from 0.1 mm (parasitic wasps) to greater than 50 mm for some of the predaceous wasps. The integument of hymenopterans is usually heavily sclerotized, with pleural sclerites of the thorax highly modified and fused for strength. Wings are usually well developed in most bees and wasps and in the reproductive (sexual) forms of ants. However, wings are lacking in the nonreproductive worker caste in ants.

In wasps, the first abdominal segment is fused with the thorax as a propodeum. The second abdominal segment forms the petiole, a narrow constriction between the thorax and the more enlarged remaining part of the abdomen called the gaster. The prominent petiole of many wasps has given rise to the term "wasp waist" and "wasp-waisted."

Although bees resemble wasps in many features, they generally have more hairs (setae) on the body. Most bees have branched hairs on the legs and gaster, and the broad hindlegs are modified for gathering and transporting pollen to their nests. Honey bees and other bees in the family Apidae possess dense rows of specialized, branched hairs on the hindlegs called corbicula, or pollen baskets, for carrying pollen.

Ants have evolved from primitive wasplike ancestors, and all exhibit social behavior. They are readily identified by a narrow petiole that consists of one or two segments and bears a dorsal lobe. Worker ants have geniculate (elbowed) antennae, with the first segment being very long.

Many ant species, like other social insects, have distinct **castes** of queens, sterile female workers, and males. The

TABLE 22.2 Subfamilies, Species, Common Names, and Principal Nest Sites of Selected Vespid Wasps in North America

Species	Common Name	Nest Site
Subfamily Polistinae		
Polistes		
P. apachus	None	Aerial
P. aurifer	Golden paper wasp	Aerial
P. annularis	Spanish Jack	Aerial, river and lake shores
P. carolina complex	Red wasp	Aerial, concealed
P. exclamans	Guinea wasp	Aerial, especially around man-made structures
P. dominula	European paper wasp	Aerial, concealed especially around man-made structures
P. metricus	None	Aerial, concealed
P. rubiginosus	Red wasp	Aerial
P. fuscatus	None	Aerial
Subfamily Vespinae		
Vespa		
V. crabro	European hornet	Hollow trees, attics
Dolichovespula		
D. arenaria	Aerial yellowjacket	Aerial, trees, structures
D. maculata	Baldfaced hornet	Aerial, trees
Vespula		
V. flavopilosa	Hybrid yellowjacket	Subterranean
V. germanica	German yellowjacket	Subterranean, voids in structures
V. maculifrons	Eastern yellowjacket	Subterranean
V. pensylvanica	Western yellowjacket	Subterranean
V. squamosa	Southern yellowjacket	Subterranean
V. sulphurea	California yellowjacket	Subterranean
V. alascensis (*formerly vulgaris*)	None	Subterranean

Based on Buck et al., 2008, 2012; Kimsey and Carpenter, 2012.

size variation among ant species is extremely great, ranging from the little black ant *Monomorium minimum* (workers 1.5−2.0 mm long) to the large hunting ponerine ant, *Dinoponera gigantea* (up to 34 mm). Some ants, such as the primitive, tropical bullet ant *Paraponera clavata*, exhibit few, if any, morphological or size differences between the queen and the workers; it is monomorphic (i.e., represented by only one form). However, most ants are polymorphic and are easily separated into castes. Some species of ants exhibit marked worker polymorphism with extremely large individuals called **majors**, usually with large heads. They may also have intermediate-size individuals, and **minors** or small workers. The minors usually constitute most of the **nurse workers** that take care of the brood, while the majors, or **soldiers**, respond quickly to disturbances to defend the colony. Soldiers of army ants (*Eciton* species) have disproportionately large heads that house large adductor muscles that operate the equally large, ice-tong−shaped mandibles. The great differences in size and morphology among the various castes within some species can cause problems in identification, unless the castes are associated with each other and recognized as all belonging to the same species.

The sting apparatus is an important morphological trait of the aculeate hymenopterans (Fig. 22.1). This complex structure in wasps, bees, and ants is a modification of the female egg-laying structure, or ovipositor. Males lack this structure and cannot sting. In the minority of ants that do not possess a sting, the components of the ovipositor have been reduced or lost. These ants, such as *Formica* spp.,

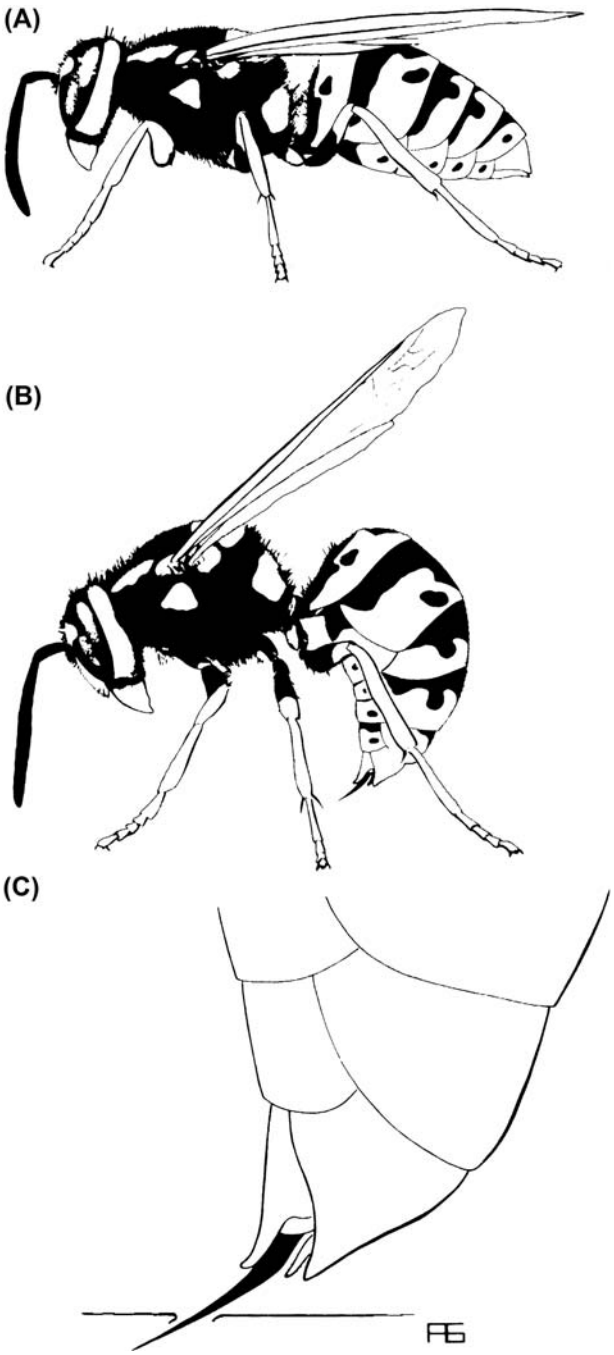


FIGURE 22.1 Sting action of a yellowjacket (Vespidae) worker. (A) Resting position. (B) Sting position with abdomen flexed and lancets of sting extruded. (C) Sting at tip of abdomen piercing skin. *From Akre et al., 1981.*

defend themselves by spraying secretions (e.g., formic acid) from the tip of the gaster into wounds inflicted by the mandibles (Fig. 22.2).

The external portion of a typical ovipositor consists of three pairs of elongate structures, or valves, which are used to insert the eggs into a substrate such as plant tissue or soil.

FIGURE 22.2 *Formica* species ant biting finger. *Photograph by Hal C. Reed.*

One pair of valves often serves as a sheath and is not a piercing structure; the other two pairs form a hollow shaft, which pierces the substrate by a back-and-forth sawing motion, with one pair held in position by the other (Fig. 22.3). The eggs pass down through the shaft, except in the stinging species (Aculeata). During oviposition by aculeate hymenopterans, the sting apparatus is flexed up

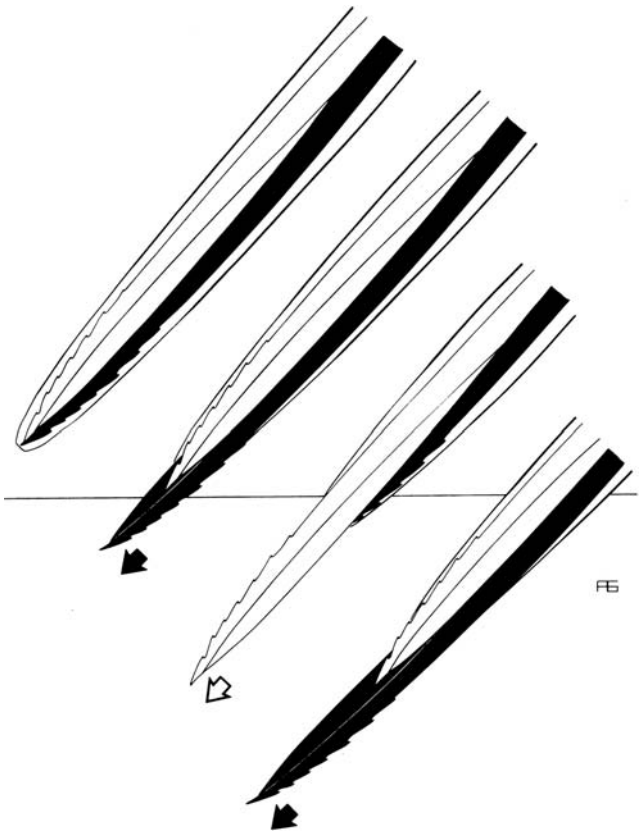


FIGURE 22.3 Mechanism by which the pair of lancets of hymenopterans penetrate the skin when a victim is stung. The lancets slide alternately back and forth as their serrated tips quickly work their way into the tissue. The opposing surfaces of the lancets are concave, forming a tubular channel through which venom is injected into the wound. *From Akre et al., 1981.*

and out of the way as the eggs are passed from the genital opening at the base of the ovipositor.

There are usually two accessory glands in the female that secrete substances to form egg coverings or glue the eggs to the substrate.

Although cartoonists delight in portraying the stinger of wasps and bees as a constantly protruding spike, the sting is a complex apparatus typically retracted within a cavity at the end of the abdomen (Fig. 22.4). The morphological differences in the sting apparatus among the various aculeate groups are relatively minor. The principal components are a pair of long, slender **lancets** that are encompassed by a single **stylet**. The ventral edges of the stylet function as a guide rail along which each lancet can slide freely. These structures converge at the base to form a channel through which the venom flows. In some species the distal tip of the lancets is armed with barbs to aid their penetration and resist retraction. When in the retracted position, the sting apparatus is covered by two membranous sting sheaths. Penetration by the lancets and stylet into a victim is accomplished by contraction of muscles connected to large sclerotized plates articulating with the base of the stylet and lancets.

The accessory glands of the female reproductive tract have become modified for specialized purposes in stinging hymenopterans (Fig. 3.2). One has become a **poison gland**, which produces venom. Another, called **Dufour's gland**, produces lubricants and coatings for the eggs, linings for brood chambers, and pheromones in some species. The venom is actually produced in the poison gland, which consists of two slender, elongate tubules that empty their products into a prominent, sometimes muscular, reservoir, or poison sac. This reservoir stores the venom until the insect stings, at which time the venom is ejected through a narrow tubule at the base of the sting apparatus. The morphology of the glands associated with the venom apparatus in stinging hymenopterans is reviewed by Van Marle and Piek (1986).

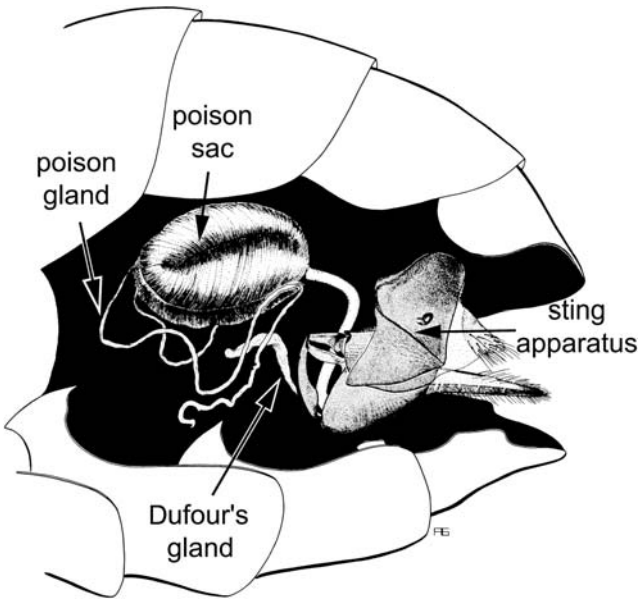


FIGURE 22.4 Poison gland and associated structures of the sting apparatus of a yellowjacket (Vespidae) worker. *From Akre et al., 1981.*

LIFE HISTORY

Hymenopterans undergo holometabolous development with egg, larval, pupal, and adult stages. They exhibit **facultative arrhenotoky**, in which males are produced parthenogenetically, enabling these insects to control the sex of their offspring. Males are haploid and develop from unfertilized eggs, whereas the diploid females develop from fertilized eggs. Colonies of most social hymenopterans consist of sterile female workers with a single queen (i.e., monogyny) or multiple queens (i.e., polygyny).

In temperate climates, reproductive males and females of social species of ants, bees, and wasps are produced in late summer and early fall. These reproductives usually leave the colony to find and select mates. Males typically die soon after the mating season and do not contribute to colony activities, such as foraging, brood care, nest maintenance, or defense. An inseminated queen usually will overwinter outside of a colony and start a new colony in the spring, as in the case of yellowjackets and hornets, paper wasps, many ants, and bumble bees. However, great variation in colony founding modes is seen among the social species. Honey bee colonies, for example, reproduce when a mated queen leaves the parent colony with a group of workers to begin a new hive, a process known as **swarming**. Colonies of honey bees and many ant species exist for longer than 1 year as perennial colonies, whereas most temperate social wasps, some ants, and bumble bees typically have annual colonies. Development of the queen among social insects is the result of controlled nutrition and available space (e.g., cell size), for a larva during development. These activities are mediated by pheromones that influence the behavior of the workers toward the developing brood. For example, honey bee larvae destined to become queens are reared in large queen cells and fed a special diet of **royal jelly**, secretions from head glands of nurse bees, throughout their development. Rearing of a new queen usually occurs when the queen becomes old, her pheromone secretions are waning, or she has died. New queens also are produced in large, healthy colonies of the honey bee and other species of *Apis*.

The life histories of social species of Hymenoptera are more diverse in tropical latitudes. Colonies may be started by a single queen, multiple queens, or a swarm of one or more queens, along with a retinue of worker females. Colony reproduction may be by individual mated female foundresses, by multiple mated females, by swarms of

queens with workers, or by queen replacement. Colony formation, and the later production of new queens and males, can be somewhat synchronized with a wet/dry seasonal cycle or may appear to be asynchronous. Colony duration might be a fraction of a year, multiple years, or potentially unlimited as occurs with army ants (*Eciton*).

BEHAVIOR AND ECOLOGY

Behavior among hymenopteran species is extremely diverse. It varies from individuals that are **solitary**, with little or no interaction among nesting females, to species that are highly **social**. Many species have intermediate levels of social behavior in which the parent cares for its offspring and may even share a common nesting area. Eusocial species exhibit a reproductive division of labor, a worker caste caring for the young, and overlapping generations in which the offspring assist the parent(s) in rearing the brood. Ants, honey bees, bumble bees, and vespid wasps are eusocial species and typically are found in colonies, often within nest structures.

Stinging behavior is one of the intriguing aspects of social hymenopterans. Visual cues, vibrations, movement, chemicals, and colony status (e.g., nutrition, reproductive presence) all play important roles in eliciting a stinging response. Stinging can occur with the disturbance of a nest, in response to swatting, touching, or squeezing of a worker or queen, and in response to alarm chemicals released by workers. Dark, moving objects are readily attacked by honey bees and vespid wasps when defending their colonies. Vibrations of the ground or other substrate, such as a branch that contacts the nest, can stimulate alarm and attack in most species.

Alarm pheromones that alert and recruit nest mates to defend the colony are a common strategy among many ants, bees, and wasps, particularly those with large colonies. For example, the honey bee produces several volatile compounds associated with the sting apparatus and one from the mandibular glands. Together they alert the colony and help to focus the attack of hive mates, such that numerous bees can quickly deter a potential predator or other threat. Disturbed workers may also attack when they sense certain chemicals on the attacked individual. Honey bee workers, for example, may sting people when they are applying enamel paints containing isoamyl acetate (= isopentyl acetate), a natural component of the honey bee alarm pheromone.

Foraging wasps and bees are less prone to sting than are individuals near or on the nest. However, foragers may approach and inspect humans, especially if one emits sweet-smelling odors, and, if irritated, will sting to defend themselves. Typical stinging behavior of social hymenopterans is illustrated by an attacking yellowjacket (Fig. 22.1). The insect grips the skin firmly with its legs and sometimes with its mandibles, exposes the sting apparatus, then plunges the tip of the interlocked lancets and stylet into the skin with a downward thrust of the abdomen. Simultaneously, the contraction of the muscles surrounding the poison sac forces the venom into the sting bulb and through the channel formed by lancets and shaft, much like a hypodermic needle. In order to penetrate, the lancets are moved forward in alternate strokes, each sliding on its track against the stylet shaft (Fig. 22.3). The tips of the lancets are equipped with barbs in bees, wasps, and a few ants to facilitate penetration; they literally saw through the victim's flesh as each lancet in turn is thrust forward and anchored in place by the barbs.

Sting autotomy is behavior in which the anchored sting is left embedded in the skin when the insect pulls away (Fig. 3.2). It is best known in honey bees, and it also occurs in some tropical epiponine social wasps and in harvester ants (*Pogonomyrmex* spp.). However, bumble bees do not lose their stingers and may sting repeatedly. Although sting autotomy results in evisceration and eventual death of the insect, the sting with the attached poison gland reservoir continues to pump venom into the wound. Stinger loss is due to the presence of large, recurved barbs on the lancets and to lines of weakness in the structure of the sting apparatus, causing it to readily detach as the insect pulls away after stinging. The barbs on a yellowjacket or paper wasp sting are much smaller and less recurved than those of the honey bee and do not normally become anchored in a person's flesh. The wasp usually can quickly withdraw with an upward pull of its abdomen and sting multiple times. However, the stingers of a few species (especially *Vespula maculifrons*) can become embedded when they attack human skin and cannot pull out, and workers of some species of yellowjackets leave their stingers behind when they sting thick leather gloves (see Greene et al., 2012).

Aculeate hymenopterans feed on a variety of prey and plant sources. Most food foraging is for larval food. This consists of nectar and pollen among bees and masarine wasps, seeds in harvester ants, plant material in some ants, or various arthropod prey in most wasps and many ants. Some yellowjackets will also scavenge for dead arthropods and vertebrate carrion with which to feed the larvae. Adult food includes similar materials but in a liquefied state. Adults of many species of wasps, ants, and bees also actively forage for sweet materials that they can ingest or, in the case of social species, carry back to the colony. Such foraging may include nectar from flowers (Fig. 22.5) or extrafloral nectaries of plants, fruits, tree saps, and insect honeydews. In eusocial species, liquids are mutually exchanged among workers and between larvae and workers in a process called **trophallaxis**.

The use of chemical communication is very important in maintaining cohesion and activities in most Hymenoptera colonies. Alarm pheromones are often used to recruit

FIGURE 22.5 *Polistes fuscatus* wasp feeding on floral nectar. *Photograph by Hal C. Reed.*

workers to defend the nest and to focus attacks. Many species of ants lay down trail pheromones to food sites to recruit additional foragers, thereby increasing the feeding efficiency. Ants, bees, and wasps produce queen pheromones that limit reproduction by workers and stimulate colony activities such as foraging, construction, and brood care. In some species, queen pheromones can convey information about the status of the colony through trophallactic exchanges of chemical substances among colony members. Additional information on pheromones of social insects can be found in Vander Meer et al. (1998).

HYMENOPTERA VENOMS

Venoms of social hymenopterans are complex biochemical mixtures that paralyze prey, induce pain in vertebrate predators, or act as toxicants (Schmidt, 2009, 2016). Probably less than 100 of these venom compounds have been identified, while many more remain to be discovered and characterized. There are three general categories of venom compounds:

1. Small, nonproteinaceous molecules with molecular weights less than 300 Da
2. Peptides with molecular weights of 1,500−4,000 Da
3. Larger proteins and enzymes with molecular weights greater than 10,000 Da

Compounds of the first category include histamines, serotonin, and various catecholamines that induce itching, immediate pain, redness, and changes in capillary permeability. The second category is peptides such as hemolysins that destroy red blood cells and cause pain, neurotoxins, and other pain-inducing compounds such as kinins in social wasps. The third category of larger proteins and enzymes generally do not cause pain but aid in the spread and activity of other venom components; some act as allergens. An example is hyaluronidase, which facilitates the spread of toxic components through tissues. Less commonly encountered are phospholipases. They are toxic, disrupt cell membranes and cause the release of pain-inducing agents (Schmidt, 1986a, 1986c, 1992, 2009).

Venoms of solitary hymenopterans such as sphecid and pompilid wasps are designed to cause paralysis in insects and other arthropods on which they prey. These venoms directly affect the nervous system and cause a general decline in the rate of metabolic processes. The purpose of these venoms is not to cause the death of the prey but to incapacitate it as food for the larvae. Common components of the venoms of various solitary wasps are histamines, polyamines, and substances such as bradykinins that cause smooth muscles to contract. Some of their venoms also contain large amounts of the neurotransmitter acetylcholine, as in the case of the solitary sphecid wasp *Philanthus triangulum* and tarantula hawks (Pompilidae: *Pepsis* spp.) (Schmidt, 2016). The venoms of solitary wasps generally produce only momentary, slight pain in humans. However, the venoms of large tarantula hawks contain a powerful neurotoxin and, thus, it is considered to cause one of the most painful stings (Schmidt, 2016).

Ant Venoms

Ant venoms serve a variety of functions including defense, prey capture, aggregation, trail marking, alarm, repelling intruders, and antimicrobial activity (Aili et al., 2014). Only the components of ant venoms that are toxic to vertebrate animals are discussed here. The toxins normally are injected via the sting; however, some ants lack a sting and spray **formic acid** at their attackers (e.g., many formicine ants). Formic acid is a very effective deterrent, especially if sprayed into the eyes or applied directly to wounds made with the ant's mandibles (Fig. 22.2). Schmidt (2009) and Aili et al. (2014) provide excellent overviews of the functions and chemistry of ant venoms.

Venoms of the majority of stinging ants are predominately composed of proteinaceous mixtures. Fire ant venoms, however, largely consist of alkaloids (95%) with only a small proteinaceous component (0.1%−1%) (Touchard et al., 2014). The alkaloids cause most of the local sting reactions, whereas the proteins contain active allergenic antigens. The alkaloids are methyl-n-alkylpiperidines called solenopsins and a piperidine. The alkaloids are cytotoxic, hemolytic, fungicidal, insecticidal, and bactericidal. The characteristic dermal necrosis that becomes evident at the sting site is due to these alkaloids.

Protein-rich ant venoms are found in most subfamilies of ants, including the Ponerinae, Myrmeciinae, Pseudomyrmecinae, Ecitoninae, and some of the Myrmicinae (Table 22.1). These venoms have not been well

investigated because of difficulty in obtaining sufficient quantities of pure venom for analysis. However, advances in analytical techniques have made it possible to investigate minute quantities of venom. Most studies available are those of primitive ants in the genus *Myrmecia* (Myrmeciinae) and the highly evolved *Myrmica* and *Pogonomyrmex* (Myrmicinae). Harvester ants have a proteinaceous venom with high amounts of phospholipases A1 and B, hyaluronidase, a potent hemolysin called barbatolysin, and histamines (Schmidt, 1986b). Their venom is also considered the most toxic known insect venom (in terms of LD_{50}) (Schmidt, 2009, 2016). Most ant venoms contain only small amounts of these compounds. Other enzymes that have been identified in ant venoms include acid phosphatase, alkaline phosphatase, phosphodiesterase, lipase, esterase, and a nonspecific protease. The primary function of these compounds is to cause pain, either directly or through tissue destruction.

Vespid Venoms

Vespid venoms are biochemically complex and are designed to cause pain (Nakajima, 1984, 1986). Their venom typically produces immediate pain, local swelling, and erythema caused by an increase in the permeability of blood vessels at the sting site. The pain often continues for several hours, whereas itching at the sting site may persist for several days. Vespid venoms also cause the contraction of smooth muscles, reduced blood pressure, and the release of histamine and other biogenic amines. Hemolysis induced by lytic peptides and phospholipases may cause kidney damage. There is usually additional damage to surrounding tissues from the products of histolysis. Vespid venoms contain biologically active amines such as serotonin, histamines, tyramine, and catecholamines, all of which tend to produce pain. Acetylcholine has been reported to occur in the venoms of some *Vespa* spp. However, the primary pain-causing substances are kinins. In addition, the venoms contain mast cell—degranulating peptides called mastoparans that cause the release of histamines and hyaluronidase acting as a venom-spreading agent. Venoms also contain enzymes that can act as specific allergens and, in some species, neurotoxic compounds. The immediate pain caused by a vespid sting is principally due to serotonin and kinins. Venoms of some vespids, like honey bees, contain alarm pheromones that function to alert nestmates to an intruder and focus stinging attacks.

Honey Bee Venom

The venom of honey bees is the most studied of all insect venoms (Schmidt, 2016). It is a complex mixture of proteins, peptides, and small organic molecules (Banks and Shipolini, 1986; Schmidt, 1992). The most dangerous components for humans are phospholipases and hyaluronidase. Individuals can become sensitized to these materials and subsequently even die from a serious allergic reaction. Bee venom contains large quantities of a potent membrane-disrupting material called melittin, which makes membranes extremely susceptible to attack by phospholipases. Melittin is also a cardiotoxin, causes pain, increases capillary blood flow and cell permeability, triggers lysis of red blood cells, and enhances the spread of toxins. The effects of melittin, phospholipase, and a mast cell-degranulating peptide cause the release of histamine and serotonin from red blood cells and mast cells. While the components of honey bee venom that cause pain are very different from those in vespid venom, the end results are very similar. Some components of honey bee venom regulate and/or decrease inflammatory responses in some individuals. This perhaps explains why bee-venom therapy has been useful in the treatment of certain forms of arthritis. Another component of honey bee venom, a neurotoxin called apamin, seems to cause more effects in insects than in humans.

ANTS

Ants are ubiquitous, occurring throughout much of the world, including oceanic islands. Most major taxa of ants have species that occur worldwide. However, some groups and subgroups are restricted to specific areas. The acacia ants (pseudomyrmecines) and ponerines, for example, occur primarily in the tropics, and bull-dog ants are found only in Australia. The primarily tropical ecitonine army ants are neotropical, with a limited distribution in the United States and two species occurring as far north as Iowa.

The ants of significant medical-veterinary concern in the United States are the fire ants (*Solenopsis* and *Wasmannia* spp.) and harvester ants (*Pogonomyrmex* spp.). Most other North American ants rarely sting people, or they are so small that they are incapable of piercing human skin. The carpenter ants (*Camponotus* spp.), which are commonly destructive pests of wooden structures, lack a sting, as do members of the subfamilies Formicinae and Dolichoderinae. Other stinging ants such as ponerines (Ponerinae) and army ants (Ecitoninae) are a concern in tropical regions. However, there is one ponerine species occurring in the southeastern United States, *Odontomachus haematodus*, which can cause painful stings to humans. *Odontomachus haematodus* is peculiar in that it possesses elongated mandibles that are held open at 180 degrees and snap shut quickly to impale prey on their sharp teeth. It also uses its mandibles to snip and jump away when threatened. The **Asian needle ant** (*Pachycondyla chinensis*), also called the **Chinese needle ant**, can cause painful stings. This species was introduced to the southeastern United States sometime before 1932 and has been reported as a particularly

pestiferous, stinging ant (Nelder et al., 2006). Other pestiferous ants that may on occasion be a hazard are the ponerine *Hypoponera punctatissim, Pseudomyrmex ejectus*, the pavement ant, and pharaoh's ant.

The highly invasive **Rasberry (tawny) crazy ant** (*Nylanderia fulva*) has become pestiferous in southeastern Texas (USA), where it has reached enormous population sizes (Gotzek et al., 2012). This species lacks a sting but does release a high dose of formic acid, which can counteract fire ant venom (LeBrun et al., 2014). The high worker densities, along with this chemical defense, has given this crazy ant a competitive advantage over the imported fire ant, resulting in their displacing fire ants in some areas of southeastern United States. Although this species can bite humans, it causes only momentary minor pain. Like fire ants, crazy ants have a propensity for inhabiting electrical equipment, which may bring them into frequent contact with humans.

The life cycle of ants is highly varied. In some species, such as the army ants whose queens are wingless throughout their life, colony initiation occurs by budding, a process whereby a colony divides into two colonies. In contrast, many ant species have winged male and female reproductives and colony initiation is typically by a single winged queen. At certain times of the year mature colonies produce an abundance of winged males and queens that leave the nest en masse on a nuptial flight. After mating, the males die and the inseminated queens lose their wings before searching for a suitable nest site. The queen lays eggs in the new nest site and feeds the developing larvae from her food reserves stored as fat and flight muscle. The emerging brood becomes the workers, and they take over nest maintenance, foraging, and nursing activities. The small colony grows slowly at first and may take a few years to become mature and produce its own reproductives. A single queen (monogyny) is the rule in some ant species, whereas multiple queens (polygyny) occur in others. A few species, like the imported fire ants, have both monogynous and polygynous colonies.

Ants nest in a variety of situations. In the case of army ants, there is no physical nest but only a bivouac, formed from the ants holding on to one another by their legs to form a large mass. Carpenter ants excavate wood to form cavities for their nest, while other ants make aerial nests of carton, a material formed from soil or plant fibers and the ant's saliva. Some *Formica* spp. build large nests up to 1 m high consisting of a mound of small stems and twigs—hence the name "thatching ants." Most ants establish their nests in soil where they excavate extensive galleries and tunnels. Soil is an ideal nesting material: it moderates temperature extremes, holds moisture, and can be easily shaped by the ants into brood or food-holding chambers. Ants often have preferences for specific types of soil.

The size of ant colonies varies tremendously. Colonies of primitive ants tend to have only a few hundred workers (e.g., some ponerines), whereas other ants may have up to 10,000 workers (e.g., harvester ants). The enormous colonies of 1−2 million individuals for New World army ants and 22 million for Old World driver ants (Dorylinae) are impressive. However, colonies of these species are small compared with the supposed megacolonies of some *Formica* spp., which may contain 300 million workers and nearly 1 million queens occupying an area of a few square kilometers! Such polygynous colonies are usually very tolerant of non-nestmate ants, and it is very difficult to determine whether such colonies are separate units or indeed one giant ant colony.

Fire Ants (*Solenopsis* spp.)

Most ant stinging problems in North America are due to the two species of imported fire ants in the southern United States: *Solenopsis invicta* (Fig. 22.6) and *S. richteri* (Fig. 22.7). Less frequent stinging problems are caused by

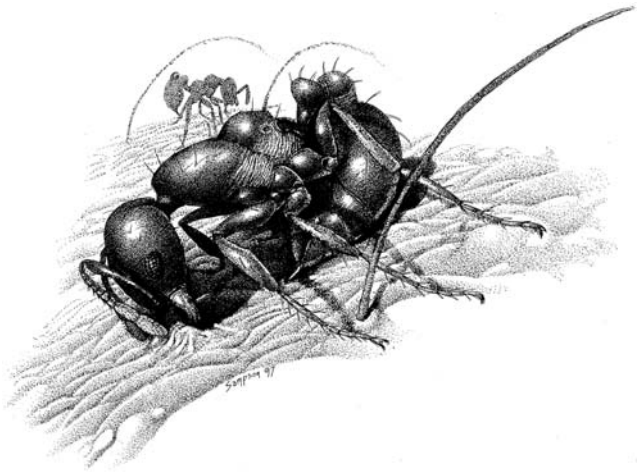


FIGURE 22.6 Red imported fire ant (*Solenopsis invicta*) worker, stinging skin of human. The ant seizes the skin between its mandibles to provide leverage as it flexes the tip of its abdomen forward to penetrate the skin with its sting. *Original by Blair Sampson.*

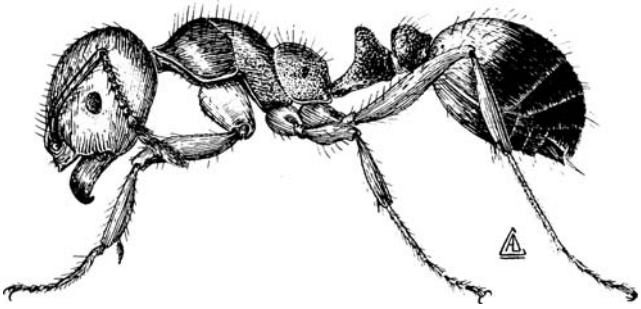


FIGURE 22.7 Black imported fire ant (*Solenopsis richteri*), worker. *Courtesy of U.S. Department of Agriculture.*

the two native fire ants in the southern United States, *Solenopsis geminata* and *S. xyloni*, and the non-native, little fire ant *Wasmannia auropunctata*.

The **black imported fire ant** (*S. richteri*) was introduced from South America to the United States at Mobile, Alabama, in the early 1900s, followed by the introduction of the **red imported fire ant** (*S. invicta*) at the same port in the late 1930s. Since that time, few other stinging insects have created more controversy, generated more research, or received more publicity than these two species of ants. They now inhabit 15 contiguous U.S. states, Puerto Rico, several Caribbean islands, Hawaii, Australia, and even parts of Asia, especially China, with the potential of further global expansion (Morrison et al., 2004) (Fig. 22.8). The black imported fire ant (*S. richteri*) occurs only in parts of Arkansas, Mississippi, Alabama, Tennessee, and Georgia. Although *S. invicta* and *S. richteri* are reproductively isolated in their native South America, they sometimes hybridize in the southeastern United States, which can complicate identification and surveillance for these two species (Gardner et al., 2008; Menzel and Nebeker, 2008). The distributions of these ants probably have reached their northernmost limits where they can survive in the central and eastern states. However, climatic models predict further range expansion in the United States and with the potential of invasion in similar areas in Europe and Asia (Morrison et al., 2004; Antmaps, 2017).

Fire ants are omnivores and opportunistic feeders. They feed mostly on insects and other arthropods. Fire ants also feed on the seeds of some plants and can affect local plant assemblages by transporting viable seeds of other plant species. In addition, they feed on germinating plants, as well as on fruits and roots, and can cause damage by girdling tree seedlings.

Fire ants are soil nesters, with most colonies being initiated by a single inseminated queen after a nuptial flight during April−August. A queen makes a burrow 3−12 cm deep and within 24 h lays her first eggs in a chamber at the end of the burrow. As many as 2,500 colonies can be initiated per hectare, but few of these incipient colonies survive the next winter. Colony growth is rapid and often produces more than 10,000 workers within a year. Some colonies contain tens of thousands to hundreds of thousands of workers within a few years. Polygynous colonies are fairly common. Brood can be produced year-round in the southernmost United States, but brood production ceases during the winter months north of 30°N latitude. The size and texture of the above-ground mound vary depending on soil type, moisture, and vegetation (Fig. 22.9). Mounds in sandy areas are generally low, whereas those occurring in clay soil may be up to 1 m tall × 1 m in

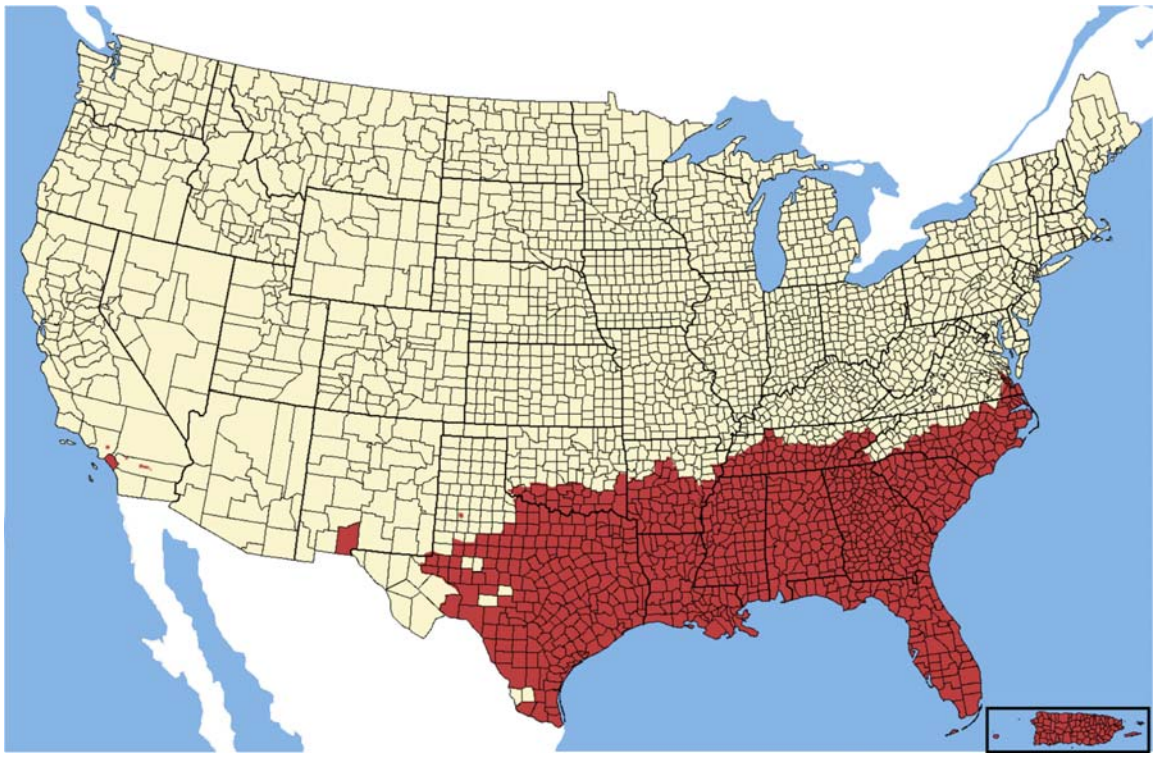


FIGURE 22.8 Quarantine map of the imported fire ant (*Solenopsis invicta*) in the continental United States and Puerto Rico (insert at lower right), 2016. *Modified from U.S. Department of Agriculture/Animal and Plant Health Service map.*

FIGURE 22.9 Typical fire ant mound in Piedmont region of southeastern United States. *Photograph by Gary R. Mullen.*

diameter. Large colonies often construct several interconnected mounds. Colonies can even survive flood events by forming a large floating mass of ants, as witnessed in the aftermath of Hurricane Harvey in southeastern Texas.

Fire ants quickly respond to disturbances of their nests and attack intruders in force. When a colony is disturbed, the ants swarm over the intruder until the first worker stings and alarm pheromones are released. This triggers stinging behavior in other workers. The workers grasp the skin with their mandibles (Fig. 22.6) and sometimes turn in a circle from this attachment, stinging the entire time: the result is often a semicircle of sting pustules. The common name "fire ants" reflects the burning sensation caused by their stings.

Fire ants occur in rural, urban, and suburban areas where they constitute a serious stinging problem. In addition to damaging crops, they often sting farm workers as they harvest crops by hand. Large sun-hardened mounds can damage farm equipment or make it nearly impossible to harvest the crops by machine. High densities of colonies in agricultural areas can result in land devaluation. The impact of the red imported fire ant on recreation is decidedly negative as tourists tend to avoid areas with heavy infestations. Colonies are common in urban yards and parks and can be a serious stinging problem to small children and pets. Colonies are even known to nest in traffic lights, air conditioners, and other electrical equipment. Foraging ants will enter houses such that stinging incidents also can occur inside homes.

These introduced pests are out-competing and displacing many native ant species and can adversely affect ecological communities. At the same time these ants have had some beneficial effects. They are general predators of a wide variety of crop-damaging insects and of ticks and flies detrimental to livestock and game animals.

The **southern fire ant** (*Solenopsis xyloni*) ranges from California to South Carolina and Florida (USA). This ant usually nests in soil, although it also will nest under stones, in the woodwork of houses, and in masonry. Outdoor nests are marked by excavated soil deposited in irregular piles around the entrances. These ants can girdle nursery stock and other plants and will burrow into plant buds, potato tubers, and strawberries.

The **tropical fire ant** (*Solenopsis geminata*) ranges from South America to Texas and Virginia to Florida as well as parts of Africa, southern Asia, Australasia, and the Pacific region. Until the imported fire ants were introduced, this ant was the most common and serious ant pest in Florida. Its nests are built in the soil, producing mounds up to 20 cm high. The biology of this species is similar to that of *S. invicta*. Like *S. xyloni*, it represents only a minor problem and has been largely displaced by *S. invicta* in the United States as *S. invicta* has extended its range.

The **little fire ant** (*Wasmannia auropunctata*) is only 1.5 mm long. It nests in soil, in decayed wood, under stones, in cavities in plants, at the bases of trees, and in houses. The limits of a nest are hard to determine, suggesting that nests have satellites and that colonies are polygynous. This ant occurs in Florida (USA) and has spread to other subtropical and tropical areas and cannot survive in cooler areas of the United States (Wetterer, 2013). Unlike other fire ants, workers of this ant are not aggressive and sting only when trapped in clothing or similar situations. Unfortunately, these ants will nest in houses and infest clothing, beds, and food. Laborers sometimes refuse to work in cropland where these ants are abundant. Perhaps this avoidance is due to the fact that their stings are associated with corneal lesions in humans and animals (Rosselli and Wetterer, 2017). Additional information on the biology of *Wasmannia* can be found in Williams (1994). For reviews of fire ants in the United States, see VanderMeer et al. (1990), Taber (2000), and Tschinkel (2006).

Harvester Ants (*Pogonomyrmex* spp.)

As their common name implies, these ants regularly include seeds as part of their diet. In addition, *Pogonomyrmex* workers scavenge for dead arthropods. Although there are a number of genera in the subfamilies Ponerinae, Myrmicinae, and Formicinae that compose the harvester ants, only *Pogonomyrmex* spp. are of concern as a stinging threat in North America as well as in Central and South America. Seven species of *Pogonomyrmex* in North America might constitute a stinging hazard (Cole, 1968; Taber, 1998). The more common ones are the **western harvester ant** (*P. occidentalis*), the **red harvester ant** (*P. barbatus*), the **California harvester ant** (*P. californicus*), and the **Florida harvester ant** (*P. badius*).

Pogonomyrmex workers are large, up to 10 mm in length. Most are light red or brown, although the gaster of some species may be dark brown to black. These ants are identified by the presence of a psammophore, a fringe of

hairs on the underside of the head. These "beards" are used in excavating nests, pushing material from the nest much like the blade of a bulldozer. Harvester ants usually move slowly unlike the fire ants. Dense concentrations of colonies are common in the western United States, where most North American species occur.

Harvester ants construct their nests in dry, sandy to hard soils. The entrance to the nest is often marked by a crater or a cone in the center of a slight mound, usually surrounded by a pile of small stones (Fig. 22.10). Some species in hot deserts lack a mound. The nest can be 1−10 m in diameter with tunnels extending down to 5 m or more. The area around the nest is usually completely devoid of vegetation. Colonies of some species have up to 10,000 workers. Individual colonies often survive for 14−50 years, reaching maximum densities of 80 or more nests per hectare. Foraging trails from individual nests may extend out 60 m. Where nest densities are high, large expanses of ground may have little vegetation. Because of the habit of harvesting seeds and reducing vegetation, they can damage rangeland used for cattle grazing and sometimes become significant pests locally (MacKay, 1990). At the same time they are beneficial in aerating the soil, providing enrichment, and promoting new plants sprouting from discarded seeds. Nests invariably occur in sunny locations, and if nests become shaded by vegetation or human activity, the ants generally move.

Harvester ants sting readily and can inflict intense pain. The incidence of stings is low, however, because their relatively large size and conspicuous nests cause most people to avoid them. Also, their colony numbers are relatively small compared with some other ant species.

Pavement Ant (*Tetramorium caespitum*)

This ant (Fig. 22.11) was introduced to North America likely from Europe as it has a widespread distribution across Europe and Asia. At one time it was especially common only along the Atlantic seacoast, but now it is very common throughout North America. Although nests are usually located in soil, they also commonly occur in houses. The pavement ant is omnivorous, being particularly fond of meats and fatty substances. Although it can cause damage to cultivated plants, it is more of a nuisance than a stinging problem for homeowners. This small ant (3−3.5 mm long) usually is not capable of penetrating human skin with its sting. Nevertheless, stings have been reported to cause a skin rash in children, but only rarely have they stimulated serious allergic reactions.

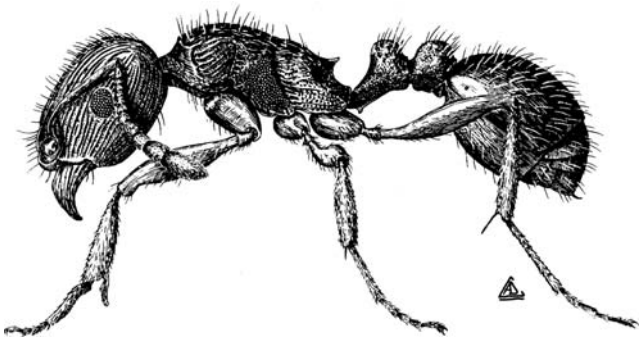


FIGURE 22.11 Pavement ant (*Tetramorium caespitum*), worker. *Courtesy of U.S. Department of Agriculture.*

FIGURE 22.10 Harvester ant (*Pogonomyrmex* sp.) nest showing characteristic soil pellets surrounding the entrance. *Photograph by Hal C. Reed.*

Pharaoh's Ant (*Monomorium pharaonis*)

This tiny ant (Fig. 22.12) is only about 2 mm long and nests in every conceivable habitat: in soil, in houses, between sheets of paper or linen, and in trunks of clothing to mention only a few. The pharoah's ant probably is native to Africa and has been widely disseminated by commerce so that it is now found throughout most of the world. It occurs in nearly all cities in the United States. It forms huge, polygynous colonies of more than 1 million workers and produces broods year-round. This ant is omnivorous, feeding on sugary materials, dead insects, breads, and many other food stuffs; like the pavement ant, it prefers meats and fats. It is one of the most difficult ants to control.

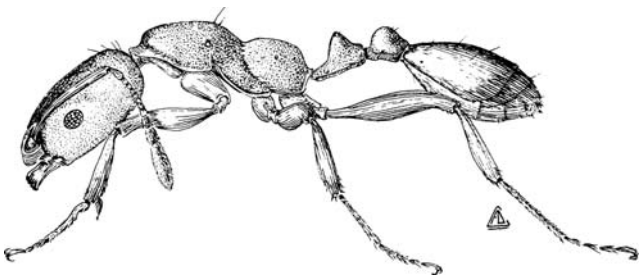


FIGURE 22.12 Pharaoh's ant (*Monomorium pharaonis*), worker. *Courtesy of U.S. Department of Agriculture.*

In addition to being a concern to human health because it stings, the pharoah's ant has been known to infest surgical dressings and intravenous units in hospitals and will attack the delicate tissues of newborn babies, especially the eyelids and navel. Because this ant has been found in bedpans, drains, and wash basins, it can come into contact with disease organisms and has been implicated in the transmission of pathogenic organisms in some hospitals. In tropical regions other ant species also have been implicated as vectors of pathogens in hospitals (Fowler et al., 1993; Pantoja et al., 2009). Edwards (1986) summarizes information on the biology and pest status of pharoah's ant.

WASPS

The term "wasp" encompasses a diverse assemblage of hymenopterous groups including solitary-nesting mud daubers, digger wasps, parasitoid wasps, and social wasps such as yellowjackets and paper wasps. Parasitoid wasps generally sting and oviposit on or into arthropod hosts that remain viable for the resulting wasp larvae to feed upon and then develop into adults. The host is much larger than the wasp, with the potential for many wasps to develop from an individual host. These are not a significant stinging problem for people. Solitary-nesting wasps, called here "solitary wasps," and social wasps, each have a well-developed sting and both use it defensively.

Solitary Wasps

Most solitary wasps hunt various arthropods as prey to feed their larvae. Their venom is designed primarily to paralyze their prey and not to cause pain in vertebrates. The female wasp may lay an egg on the immobilized prey, whereas others provision a cell of a nest with multiple prey items. Because the prey is still alive, it is essentially preserved until it can be consumed by the developing larva. Only a few solitary wasps are a serious stinging threat to people.

Mutillidae

Among the nonsocial wasps, perhaps the best known for their stings are the brightly colored **velvet ants**, or mutillids (Fig. 22.13). The sting of large species produces a painful and intense burning sensation and may cause substantial swelling and redness at the sting site. Commonly mistaken for ants, the wingless females may be seen walking over the ground during mid-summer, especially in open sandy areas, searching soil nests of bees and wasps to invade and parasitize. One large red and black mutillid, *Dasymutilla occidentalis* (Fig. 22.13), is called the **cow killer** because of its particularly painful sting. For further information on this poorly known group of solitary wasps, including keys to species in the southeastern United States, see Manley (1991).

FIGURE 22.13 A brightly colored velvet ant, *Dasymutilla occidentalis* (Mutillidae), commonly known as a cow killer because of the intense pain its sting causes. © *Photograph by Jena Johnson, Jena Johnson Photography.*

Pompilidae

The metallic blue or black **spider wasps** in this family may be seen flying low over the ground in search of spiders, which they attack, sting, and carry to a burrow in the soil where they oviposit on their prey. Some spider wasps (e.g., *Pepsis* spp.) (Fig. 22.14) use their sting not only offensively but also defensively as it contains powerful pain-inducing compounds (Schmidt, 2016).

Sphecidae

This family is a large, diverse group of solitary wasps, most of which do not commonly pose a stinging threat (Bohart and Menke, 1976; O'Neill, 2001). Sphecid wasps like mud daubers (*Sceliphron* and *Chalybion* spp.) and cicada killers (*Sphecius* spp.) typically nest around human dwellings and in disturbed soil sites. Although they appear threatening, stings to humans are relatively infrequent. **Mud daubers**, also called dirt daubers, such as the black and yellow mud daubers (*Sceliphron* spp.) (Fig. 22.15), commonly build their mud nests in attics, carports, and porches, where they provision them with spiders. These wasps are often evident at water near their nesting sites, making frequent back-and-

FIGURE 22.14 Spider wasp, *Pepsis* sp. (Pompilidae). *Photograph by Roger D. Akre.*

FIGURE 22.15 Mud dauber, *Sceliphron* sp. (Sphecidae), constructing nest. *Courtesy of Roger D. Akre.*

forth trips carrying mud to build their nests. They will rarely sting during these flights; in fact, it is difficult to induce mud daubers and other solitary wasps to sting humans or animals even by disturbing them. When stinging does occur, it usually involves the wasp being trapped inside clothing, being stepped on, or otherwise being pressed against one's skin or clothing. Occasionally they sting careless collectors.

The **cicada killers** (*Sphecius speciosus* and related species) are large wasps marked with yellow patches on the gaster (Fig. 22.16). They capture and sting cicadas and carry their prey to burrows that they have excavated in soil. The female digs a fairly large burrow with a distinct soil mound surrounding the entrance. They often nest in bare-soil areas of lawns, gardens, or flower beds around human dwellings. The female will not sting unless handled, but can inflict a mildly painful sting to people walking barefoot near their nest sites. The males frequently fly around the nests patrolling their territories. During their flights they will approach other insects, birds, and even people that encroach on their space. Although they can be intimidating, male cicada killers, like all hymenopteran males, cannot sting.

Social Wasps (Vespidae)

In contrast to the relatively innocuous solitary wasps, social wasps generally are defensive and sting readily. This usually occurs near their nests but also at foraging sites. Most vespid wasps are unique and easily recognized, in that they fold their wings longitudinally when at rest. In North America the most common social wasps are the yellowjackets (*Vespula* and *Dolichovespula*), a hornet species (*Vespa crabro*), and the paper wasps (*Polistes* spp.). The yellowjackets are stinging hazards worldwide, especially in the cooler temperate areas with hornets being a major problem in Asia. *Polistes* is a cosmopolitan genus in both temperate and tropical regions, so it is a stinging threat wherever it occurs. The large colonies of the epiponine wasps are serious stinging hazards primarily in the Neotropics.

Yellowjacket and hornet nests consist of horizontal, rounded paper combs of hexagonal cells attached one below the other, with a multilayered paper envelope. Some species build nests in exposed aerial locations (Fig. 22.17), whereas others build them in subterranean or enclosed and protected sites (Fig. 22.18). A few species construct their nests in either situation. Colony size ranges from fewer than 100 to several thousand individuals. Colonies are typically annual and are initiated by a single inseminated queen. Queens are produced late in the season and survive the winter in protected locations. During early spring, the queen begins to construct a nest, lays eggs, and then forages for arthropods to feed the developing larvae. After the

FIGURE 22.16 Cicada killer, *Sphecius speciosus* (Sphecidae). *Photograph by Hal C. Reed.*

FIGURE 22.17 Aerial paper nest of baldfaced hornet, *Dolichovespula maculata* (Vespidae) in tree. *Courtesy of Washington State University.*

FIGURE 22.18 Exposed underground nest of eastern yellowjacket, *Vespula maculifrons* (Vespidae). *Photograph by Hal C. Reed.*

first group of workers emerge, they assume most colony functions of foraging for food, fiber, and water and taking care of the larvae. The queen no longer leaves the nest after this time but assumes her primary function of laying eggs.

Perennial yellowjacket colonies sometimes occur when the new queens that emerge in the fall mate and then rejoin an active colony. This is most likely to occur in warmer climates and when the foundress queen has died or is losing her influence over the colony. These polygynous colonies can become perennial and contain tens of thousands of workers. Large perennial colonies of *Vespula alascensis* (formerly *V. vulgaris*), *V. pensylvanica*, *V. germanica*, *V. squamosa*, and *V. maculifrons* have been reported in the United States, as well as in other countries. Perennial yellowjackets have been reported in subtropical areas such as south Florida and Hawaii or in moderate temperate climates like southwestern California and the southeastern United States. Such nests are dangerous to destroy even by well-trained people.

Paper-wasp nests are single paper combs with no enclosing envelope (Fig. 22.19). They are constructed in a variety of locations, from open situations such as under eaves, to enclosed spaces such as wall voids and roof eaves. Paper-wasp nests are generally smaller than vespine nests, commonly 4–20 cm in diameter, and numbers of wasps per colony range from several to 100 or more. These nests are founded in the spring by one or more females that were produced the previous summer and have overwintered. The offspring of these nests are female workers early in the season, with new queens and males produced in late summer.

Yellowjackets (*Dolichovespula* and *Vespula*)

"Yellowjacket" is an American term that is used for all species of wasps in the genera *Dolichovespula* and *Vespula*. The name refers to the yellow-and-black color patterns of most species. However, several species are black and white, like the **baldfaced hornet**, *Dolichovespula maculata*. There are 19 species of yellowjackets in North America (6 *Dolichovespula* spp., 13 *Vespula* spp.) (Kimsey and Carpenter, 2012). Yellowjackets occur throughout the United States, including Alaska and Hawaii. Hawaii is home to an introduced population of *V. pensylvanica*. The greatest diversity of yellowjacket species is found in the northern areas of the United States and southern Canada (Akre et al., 1981; Buck et al., 2008).

Two species of *Dolichovespula* are considered hazardous in North America: the **aerial yellowjacket** (*D. arenaria*) and the baldfaced hornet (*D. maculata*). Both species occur primarily in forested areas (including riparian and urban forest) and are distributed throughout much of North America; however, the aerial yellowjacket does not occur as far south as the baldfaced hornet (Akre et al., 1981). Their large aerial, spherical or egg-shaped nests are familiar sights in trees and shrubs (Fig. 22.17) and are particularly obvious following leaf fall in autumn. The aerial yellowjacket occasionally builds nests at or just below ground level. In urban areas nests may be built under the eaves of houses or on nearby bushes and trees. When nests are constructed above a doorway, the vibrations from opening and closing the door can induce alarm and stinging by the wasps. Both species readily attack and sting when the nest itself is disturbed. They also are capable of squirting venom into the face and eyes. Fortunately, the

FIGURE 22.19 Paper wasps, *Polistes* sp. (Vespidae), with queen and daughter guarding nest. Note eggs and developing larvae in unsealed cells. *Photograph by Gary R. Mullen.*

average colony size is relatively small by vespine standards, consisting of fewer than 400 workers.

Vespula spp. are typically subterranean nesters (Fig. 22.18). Often the queen initiates the nest in a vacant rodent burrow or in other cavities in soil, logs, or trees. Some species, however, commonly establish colonies in wall voids of buildings and other enclosed spaces. The paper nests of most yellowjackets are made of gray carton, but those of *V. flavopilosa*, *V. maculifrons*, and *V. alascensis* consist of tan, fragile carton. Colony cycles of some species, particularly those related to *V. alascensis*, extend later into the fall than those of other species, particularly those related to *V. rufa*. Colonies of yellowjackets that are active into the fall construct nests that become quite large and may possess up to 5,000 workers (Table 22.3). Yellowjackets appear to be more aggressive and are likely to sting late in the season. This may be related to defense of the colony when queens and males are produced. Additionally, yellowjackets tend to be more persistent in seeking and scavenging for food late in the season, bringing them into more frequent contact with people and leading to more stingings.

Workers of several *Vespula* spp. (e.g., *V. maculifrons*, *V. pensylvanica*, *V. germanica*) commonly scavenge at garbage or picnic sites (Table 22.3). Scavenging yellowjackets feed on a wide variety of protein-rich foods (e.g., insects, vertebrate flesh, processed meats) and on carbohydrates (e.g., fruits, fruit juices, tree sap, soft drinks, beer, and sweets). This behavior brings these species into frequent contact with people. These yellowjackets can be problematic for fishermen and hunters with freshly caught fish or killed game. They are a problem at outdoor events where food is present and consumed, such as fairs, campgrounds, and outdoor eateries. These same species typically have longer colony durations and larger colony and nest sizes and are most abundant late in the season.

Other yellowjacket species, such as *V. atropilosa* and *V. vidua*, lack this scavenging habit. Instead they prey on insects for much of their food, thus posing less of a stinging nuisance. These species typically have shorter-duration colonies, and smaller colony and nest sizes, and do not become so abundant. When their nests are disturbed they will sting, but because their colonies are small the result is not as serious as attacks from more populous colonies.

TABLE 22.3 Comparison of Colony Parameters and Foraging Behavior of Yellowjackets in North America

Yellowjacket	Distribution	Foraging Behavior	Colony Size	Colony Decline
Vespula				
germanica	Transcontinental	Predators and scavengers	500–5,000 workers	Late September to early December
maculifrons	Eastern		500–15,000 cells	
pensylvanica	Western		Perennial colonies	
alascensis	Transcontinental		100,000+ workers	
flavopilosa	Eastern		1 million cells	
Vespula				
atropilosa	Continental	Strictly predators	75–400 workers	Late August to September
acadica	Continental		500–2,500 cells	
consobrina	Continental		No perennial colonies	
Dolichovespula				
arenaria	Transcontinental	Strictly predators	100–700 workers	Early July to September
			500–4,500 cells	
maculata	Transcontinental		No perennial colonies	
Vespula				
squamosa	Southern USA	Predators and some scavengers	500–4,000 workers	Late August to November
			500–10,000 cells	
sulphurea	California		Some perennial colonies	

Colony decline is defined as the period when reproductives emerge.

The **eastern yellowjacket** (*V. maculifrons*) and the **hybrid yellowjacket** (*V. flavopilosa*) are major picnic and campsite pests in late summer and fall in the eastern United States. Workers of both species will feed on fresh and decaying fruits. These two species are responsible for many stings to humans and their pets during outdoor recreational activities, especially during years of very high yellowjacket population densities.

The **western yellowjacket** (*V. pensylvanica*) is the dominant native yellowjacket in the seasonally dry forests of the western United States. It attains high population densities in some years. This commonly results in increased encounters with humans around homes, picnic areas, and camp grounds. During peak populations of *V. pensylvanica*, recreational areas such as resorts, hunting camps, and parks have been closed. These "wasp years" may coincide with a high incidence of forest fires, posing a serious stinging problem for fire fighters and smoke jumpers, including significant loss of worker time. Very large perennial colonies of this species occur in Hawaii (USA), where they have disrupted both agriculture and tourism.

The **German yellowjacket** (*V. germanica*), also referred to in the literature as the German wasp, is a species native to Europe that has shown a remarkable propensity for becoming established in temperate areas of the world. It now occurs in many other countries including New Zealand, Australia, South Africa, Argentina, Chile, Canada, and the United States. Colonies tend to be large, especially in warmer areas, where perennial polygynous colonies may contain thousands of workers. The German yellowjacket became firmly established in the United States by the late 1960s. The biotype in North America may nest inside structures, unlike the European biotype that typically nests underground. Colonies in buildings benefit from the associated heat and protection and tend to persist very late into the fall and winter months, resulting in larger colonies. At that time of year, workers may chew through walls and emerge inside buildings. It is now well established across much of eastern and western North America, and it is the dominant yellowjacket in some areas.

Early in the colony cycle the workers scavenge for foods rich in protein and carbohydrates. This early season scavenging can give these wasps an advantage over other yellowjacket competitors that exclusively spend time capturing insect prey. German yellowjackets are a significant pest species due to their scavenging habits, selection of nesting sites in structures, long colony duration, large colony size, and high population densities.

The **common yellowjacket** (*V. vulgaris*), a native of Europe and temperate areas of Asia, was long considered to be conspecific with the North American species that is now referred to as *V. alascensis* (Kimsey and Carpenter, 2012). The European species has been introduced into New Zealand and Australia. Workers of both species are a significant stinging problem, and their pestiferous nature is no doubt related to their scavenging behavior and great numbers where they are prevalent. In North America, they occur principally in moist forest habitats. Perennial colonies consisting of 50,000−100,000 workers have been found in southern California (*V. alascensis*) as well as in New Zealand and Australia (*V. vulgaris*). In more temperate climates, colony sizes are much smaller, typically fewer than 1,000 workers.

The **southern yellowjacket** (*V. squamosa*) is widely distributed from the mid-Atlantic states to the Midwest (USA) and south into Central America. In the northern part of its U.S. range, such as in north Georgia, it occurs in colonies with a single queen and an annual cycle. Farther south, it produces perennial colonies with a few to several dozen, egg-laying queens. Some colonies consist of more than 100,000 workers and can be deadly hazardous to humans and other animals. One such nest reported from Parrish, Florida, was 3.6 m high by 1.8 m in diameter, built on a broken tree stump. Workers of some *V. squamosa* colonies are scavengers and are common pests at picnics. The **California yellowjacket** (*V. sulphurea*), a species closely related to *V. squamosa*, also scavenges and has been reported as a nuisance for picnickers. It occurs from southern Oregon through California (USA) into northwestern Mexico.

Hornets (*Vespa* spp.)

Large-sized social wasps in the genus *Vespa* are considered the true hornets. There is only one true hornet that occurs in North America: the **European hornet** (*Vespa crabro*). This species was introduced from Europe into the New York area during the mid 1800s. Today it can be found throughout much of the eastern United States and west to Missouri. This is the largest social wasp in North America (body length >20 mm), with contrasting brown and deep yellow bands on the gaster. Although *Vespa* hornets are important stinging hazards in Asia, *V. crabro* is seldom responsible for stinging incidents in North America. This is primarily because *V. crabro* is relatively uncommon and usually nests in the hollows of trees in areas away from human activities. Brown envelope and carton distinguish their nests from the more common gray nests of the bald-faced hornet (*Dolichovespula maculata*). Despite the large size of the workers and the nest, this species is less aggressive than most other North American vespines. A typical mature colony consists of 200−400 workers, although larger colonies can reach 1,000. Colonies have a long seasonal cycle lasting from early spring into late autumn. *Vespa* species are a major predator of honey bee colonies in some areas.

Paper Wasps (*Polistes* spp.)

Paper wasps are the most common stinging wasps encountered by humans in the southern United States, especially during the summer months. Some paper wasps, such as *P. exclamans* and the invasive *P. dominula*, are improperly called "yellowjackets" due to their alternating bands of yellow and dark-brown or black markings on their gaster. Paper wasps are usually longer and have more slender bodies than yellowjackets. They can be most certainly distinguished from yellowjackets by the shape of the anterior-most segment of the gaster, which slopes gradually to the wasp waist in *Polistes*, whereas it is truncated abruptly in the vespines. *Polistes* wasps construct paper nests consisting of a single comb with no envelope (Fig. 22.19). Several species tend to nest near or on buildings and as such come in frequent contact with people. Their nests can be found under roof eaves and window sills; around door frames; and inside garages, storage buildings, clothesline poles, bird houses, wall voids, and attics. More natural nesting sites include trees, shrubs, and cliff overhangs. Some species such as *P. exclamans* prefer nesting in exposed sites on structures, while others like the **red wasp** (*P. rubiginosus*) usually nest in concealed sites such as wall voids, inside roof eaves, and in attics.

Most colonies are small, with fewer than 100 adults and 100–200 cells. Larger nests consist of about 400 cells. The annual colonies are initiated by a single foundress or a group of foundresses that compete for reproductive dominance. After the workers emerge, one foundress usually becomes the primary reproductive or queen. Unlike vespine wasps, no queen-worker size dimorphism occurs in paper wasps. Paper wasps forage for caterpillars and other insects and thus are excellent natural control agents of many crop pests. Colonies have been propagated and transplanted into fields to decrease pest populations.

In the fall, queens and males of some species of paper wasp may swarm in large numbers at the top of tall towers and buildings where they may create concerns (Reed and Landolt, 1991). However, many of these wasps are males, which cannot sting, whereas the females are primarily involved in mating and aggregation and only rarely sting at these sites. Later in the fall and winter the females aggregate in large numbers at hibernation sites such as the attics of houses, apartment buildings, and barns. Contact with humans is common at this time, resulting in stings. People are also stung by paper wasps in houses in the spring when the aggregating wasps come out of hibernation. However, most stingings occur when nests are disturbed.

Polistes annularis is known as **Spanish jack** to fishermen, boaters, and river dwellers in the Gulf Coast states (USA) because of the occurrence of its nests in shrubs, trees, and other structures along and often overhanging streams and lakes. People are commonly stung when they inadvertently disturb a nest. This large wasp produces the more populous colonies of paper wasps in North America and often inflicts multiple stings. Several other species, such as *Polistes metricus*, *P. fuscatus*, *P. aurifer*, *P. exclamans*, *P. apachus*, and the introduced and now invasive European species *P. dominula*, are commonly encountered by people, resulting in stings (Table 22.2). This latter species has become the dominant and ubiquitous species of paper wasp in some parts of the United States (e.g., Ohio, Washington State).

BEES

Solitary Bees

Most bees are solitary or, at most, communal or semisocial. Some species of the family Halictidae, which include the sweat bees, are in fact true social bees. A few of the solitary communal and social species form dense nesting aggregations, usually in soil, where they excavate cells in which they rear their larvae. Larvae of all bee species feed on pollen and nectar provisions in these cells. Nesting activity is quite variable, ranging from a single nest to thousands of individual nests concentrated in a given area. Some bees make nests in wood cavities such as the hollow stems of trees and shrubs, burrows of wood–boring beetles, and artificial cavities such as the hollow pipes of wind chimes and keyholes in doors. Solitary bees generally are not stinging hazards to humans.

Halictidae

Some of these are known as **sweat bees** (Fig. 22.20) because of a propensity to land on people to imbibe perspiration. A person may then be stung when swatting the bee. The stings usually are considered minor irritations and simply a nuisance.

FIGURE 22.20 Sweat bee, *Augochloropsis metallica* (Halictidae). © *Photograph by Jena Johnson, Jena Johnson Photography.*

Anthophoridae

The **carpenter bees** are similar in size and general appearance to bumble bees. However, they lack the fuzzy appearance and yellow coloration typical of bumble bees, and the dorsum of the gaster is mostly shiny black (Fig. 22.21). These bees often nest in wood around human dwellings, where they bore round holes in window sills, eaves, railings, fence posts, and other wooden structures (Fig. 22.22). People are rarely stung by female carpenter bees, and when this occurs, the pain is relatively mild. Males often are seen flying around nesting sites, may make a loud buzzing noise, and may appear threatening; however, like all male hymenopterans, they cannot sting. The common carpenter bee in eastern North America is *Xylocopa virginica*; the common species in western North America are *X. californica* and *X. varipuncta*.

FIGURE 22.21 Carpenter bee, *Xylocopa virginica* (Anthophoridae), male. Note that the abdomen is mostly black. *Photograph by Gary R. Mullen.*

FIGURE 22.22 Circular entrance to nest of carpenter bee, *Xylocopa virginica* (Anthophoridae), in cedar wood of house eave. *Photograph by Gary R. Mullen.*

SOCIAL BEES

Apidae

Most bee-sting cases are due to social bees in the family Apidae, which in North America are bumble bees (subfamily Bombinae) and honey bees (subfamily Apinae). The **stingless bees** (subfamily Meliponinae) are exclusively tropical and are found in the Old and New World. It is often assumed that these bees pose no hazard to humans because they are stingless. However, they do swarm out of the nest to attack intruders by biting with their mandibles, especially around the eyes, nose, and ears. One species is known as the **fire bee** (*Oxytrigona tataira* because of the caustic defensive secretions produced by its mandibular glands that cause intense burning pain when applied to the skin.

Bumble Bees (*Bombus* spp.)

These large, hairy, yellow-and-black bees (Fig. 22.23) pose little stinging hazard for most people, despite their abundance in gardens at flowers. Most of the 400 species are nonaggressive, even when the nest is disturbed, although a few species can be very aggressive and persistent in their stinging attacks when their nests are threatened. Bumble bee workers can sting an intruder repeatedly because, like wasps, they have a stinger that is weakly barbed.

Bumble bees are abundant particularly in the more temperate areas of North America where their colony cycle is similar to that of yellowjackets. Colonies are initiated by an inseminated queen early in the spring, followed by the production of a few hundred workers during the summer. Most nests are established in abandoned rodent burrows or old rodent nests under debris or objects on the ground. They also are known to build nests in attics and wall voids of houses. Nests consist of wax pouches or empty cocoons for storing pollen and honey and of wax chambers for

FIGURE 22.23 Bumble bee, *Bombus* sp. (Apidae), foraging worker. Note conspicuous yellow hairs on abdominal segments. *Photograph by Takumasa Kondo.*

FIGURE 22.24 Exposed subterranean nest of bumble bee, *Bombus* sp. (Apidae), showing both open and sealed cells, and surrounding nesting material. *Photograph by Roger D. Akre.*

rearing brood (Fig. 22.24). Workers forage for pollen and nectar to feed the larvae. These bees are important both as natural pollinators and pollinators of many agricultural crops, such as cranberries and raspberries. A helpful identification guide to bumble bees species is provided by Williams et al. (2014).

Honey Bees (*Apis* spp.)

Honey bees (Figs. 22.25 and 22.26) are native to Europe, western Asia, and Africa. Four of them are common species that occur from Southeast Asia to Africa and in Europe: the **honey bee** (*A. mellifera*), **giant honey bee** (*A. dorsata*), **Asian honey bee** (*A. cerana*), and **dwarf honey bee** (*A. florea*). Only the honey bee occurs in North America. Several subspecies and races of *A. mellifera* were domesticated by people in Europe and have been introduced around the world. This species is an invaluable pollinator of native and commercial plants, especially fruits and vegetables. Populations of *A. mellifera* have experienced sharp declines in Europe, Asia, North America, and elsewhere in recent years, due to multiple factors: parasitic mites, pathogens, pesticides, changing farming practices, and a phenomenon called **colony collapse disorder** (CCD). Although many theories abound, currently no definitive cause (or causes) for CCD has been identified.

Honey bees build large nests, called hives, consisting of wax combs of hexagonal cells (Fig. 22.26) in which they rear the brood and store pollen and nectar. Feral, or "wild," honey bee colonies construct nests in cavities of hollow trees and in attics and wall voids of human dwellings. These colonies typically have 15,000−30,000 bees, whereas commercial hives are usually larger, with 30,000−50,000 bees. Despite the fact that commercial breeding of honey bees has dampened the defensive tendencies of many races, honey bee colonies are yet capable of attacking and stinging intruders in large numbers.

The perennial colonies of honey bees usually survive the winter, following which, in late spring through early summer, they reproduce by swarming. Swarms may be seen resting in exposed sites (e.g., trees, shrubs, and under eaves of buildings) while they are seeking a suitable cavity in which to establish a new hive. Although these swarms are less defensive than an established colony because they lack brood, and stored pollen and honey, it is best not to approach or disturb them. Professional beekeepers can be contacted to collect them.

Of particular concern in the United States is the continued northward movement of the **Africanized honey bee** (*Apis mellifera scutellata*), an aggressive subspecies of the honey bee that has spread through tropical regions of South and Central America. It is most abundant in tropical humid areas of Africa but extends into arid regions of South Africa. This honey bee was introduced into Brazil in 1956 in an effort to improve the beekeeping industry in Latin

FIGURE 22.25 A honey bee worker, *Apis mellifera* (Apidae) with pollen attached to its hindleg, foraging on a water melon flower. *From U.S. Department of Agriculture Image Gallery; photograph by Stephen Ausmus.*

FIGURE 22.26 Honey bees (*Apis mellifera*) on surface of comb in hive. European honey bee (center), surrounded by Africanized honey bee workers. *Courtesy of Entomological Society of America.*

America. Captive bees escaped in 1957 when queen excluders were removed from some of the hives. They spread south to Argentina and north through South America, reaching Panama in 1982. The first Africanized honey bee colony trapped in the United States was in Texas in 1990. Colonies of Africanized honey bees have now been found across the southwestern tier of states: California, Nevada, Arizona, Utah, Texas, Oklahoma, New Mexico, Louisiana, and Florida. They also occur in Puerto Rico; however, they exhibit less aggressiveness on the island than they do in the continental United States.

This bee quickly establishes itself in new areas. When foraging or nesting conditions become restrictive, the bees leave their hives in a process called absconding and relocate to new nesting sites. Other subspecies of the common honey bee become established more slowly in new areas and are less likely to abscond when conditions change.

The Africanized honey bee has an increased propensity for mass stinging attacks of both people and animals (Fig. 22.27). Detailed, quantitative studies have shown that these bees are much more responsive than other subspecies to movement, vibration, and their own pheromones that mediate alarm and attack. The alarm compounds are not released in greater quantity by Africanized bees; rather the threshold of response of the bees to a perceived threat is much lower. Nor is their venom any more potent than that of other *A. mellifera* subspecies; in fact, the composition is nearly identical. Aside from their behavorial differences, individual Africanized honey bee workers are visually indistinguishable from workers of the European subspecies of *A. mellifera*. It requires a DNA characterization or an examination and measurements of a set of morphological structures (e.g., wings) and biochemical features of several bees to determine their identity. About 350 human deaths in Venezuela between 1975 and 1988 were attributed to Africanized honey bees (Winston, 1992). Since first recorded in Mexico in 1986, the bees have reportedly killed several dozen people. In Brazil, Africanized honey bee envenomation increased 10-fold from 2000 with 1,440 cases and three deaths to 13,597 cases with 39 deaths in 2015 (Barbosa et al., 2017). However, annual fatalities due to bee stings in Texas (USA) have not increased since this bee arrived there in 1990.

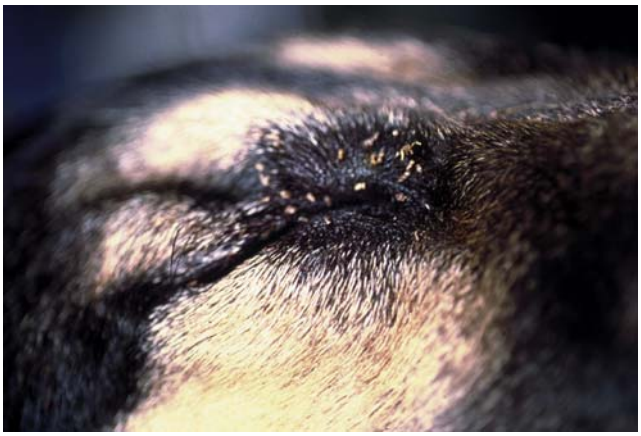


FIGURE 22.27 Head of German shepherd dog in fatal case of attack by Africanized honey bees (*Apis mellifera*). Note the concentration of bee stings about the eye. *Photograph by Justin Schmidt.*

Stinging problems due to the Africanized honey bee are actually less of a concern than the potential disruption to beekeeping and agriculture. The bees are more difficult to manage, and they store less honey than their European counterparts. These traits, coupled with their extremely aggressive nature, pose concerns for beekeeping in North America, which relies on organized transport of many managed hives for pollination services. However, the impact can be lessened by modifying hive management practices (e.g., re-queening with docile European queens), thereby altering their genetic makeup by interbreeding with European strains. Indeed, in parts of the Americas, particularly Brazil, beekeepers have successfully adapted to managing Africanized honey bees.

PUBLIC HEALTH IMPORTANCE

Most encounters with venomous arthropods involve stings from ants, wasps, and bees, most of which do not require professional medical treatment. Hymenoptera stings represent about a quarter (22%) of the annual 1 million emergency department visits due to noncanine bite and sting injuries in the United States in 2001−2010 (Langley et al., 2014). Honey bee stings were recorded 4 times as frequently as vespid stings. However, the offending insect was often either incorrectly unidentified or unknown and assumed to be honey bee. Limited data are available for sting frequency by fire ants. Studies in the southeastern United States indicate that 30%−60% of people are stung by fire ants each year, but only 1%−5% of these cases require medical treatment (Lofgren, 1986; Schmidt, 1986b; deShazo et al., 1999). This rate is higher than that of all other hymenopterans combined. Fire ant stings in South Carolina in 1998 resulted in 3.3 million people receiving hospital treatment, including 660 individuals with anaphylactic shock and two deaths (Xu et al., 2012). Stinging hymenopterans also pose a hazard during natural catastrophes such as forest fires and hurricanes. Insect stings, primarily due to yellowjackets, were the single most common cause of nonfatal injuries following Hurricane Hugo, which hit the southeastern coast of the United States in 1993 (Brewer et al., 1994). The peak of sting incidence occurred on the day of the storm, decreased rapidly on days 1−3, and continued at a low rate for the next 12 days.

A total of 40−50 deaths due to Hymenoptera stings are usually reported in the United States each year (Akre and MacDonald, 1986; Casale and Burks, 2014; Schmidt,

1986c). Mortality and morbidity data due solely to stings are not readily available, but some information has been documented in U.S. occupational injury reports (Pegula and Kato, 2014) and for U.S. Air Force personnel (Voss et al., 2016). Honey bees are believed to cause about half of the annually reported human deaths due to hymenopteran stings in the United States (Schmidt, 1992). This figure may be somewhat misleading because the general public and medical community often do not reliably differentiate between honey bees and other types of bees and wasps. Also noteworthy, more people die each year as a result of allergic reactions to penicillin or from lightning strikes than from hymenopteran stings (Camazine, 1988).

The stings of most social hymenopterans cause intense pain to humans, with reactions to various species differing primarily in intensity and/or duration. Comparative scales for ranking the severity of pain caused by aculeate hymenopterans and other insects have been proposed by Starr (1985) and Schmidt (l986c, 1990, 2016). The intensity of pain caused by most social bees and wasps is similar, and only the responses to stings of a few species such as the fire and harvester ants are clearly diagnostic. The location of the sting site can influence individual reactions. For example, stings to the lips and nose are typically more painful than stings to the upper arms and head (Smith, 2014). Also, a person's sensitivity to pain and the amount of venom injected significantly influence the severity of the sting reaction (Schmidt, 2016).

Although most ant stings are painful, those of *Paraponera* and *Pogonomyrmex* spp. are especially noteworthy. The large bullet ant *Paraponera clavata* of Central and South America injects venom that produces intense, debilitating pain lasting several hours. This sting is considered to be the most painful of all Hymenoptera (Schmidt, 2016) and has been likened to being hit by a bullet—hence the origin of their common name. The affected area can enlarge to 20−30 cm in diameter within 1 h of the sting. Schmidt's pain scale of 1−4 rates the intense pain of the stings of the bullet ant and tarantula hawk at the maximum pain level (4) and the common sting of the honey bee as the midpoint reference (2) of pain intensity. The pain caused by stings from harvester ants (*Pogonomyrmex* spp.) also is very intense (level 3) and can last 4−12 h. It has been likened to "turning a screw" into the flesh around the sting site, also causing a sensation that has been described as "chilling." The sting is unique in that it induces piloerection (elevation of the hairs), sweating at the sting site, and localized pain in lymph nodes (Schmidt, 2016). The venoms of some harvester ants are more toxic to mice (i.e., LD_{50}) than any other tested insect venoms and 8−10 times more toxic than honey bee venom. They also are more toxic per unit volume than most venomous snakes (Schmidt and Blum, 1978; Schmidt, 2016).

Victims of fire ant stings usually experience a temporary burning sensation and discomfort, with some swelling

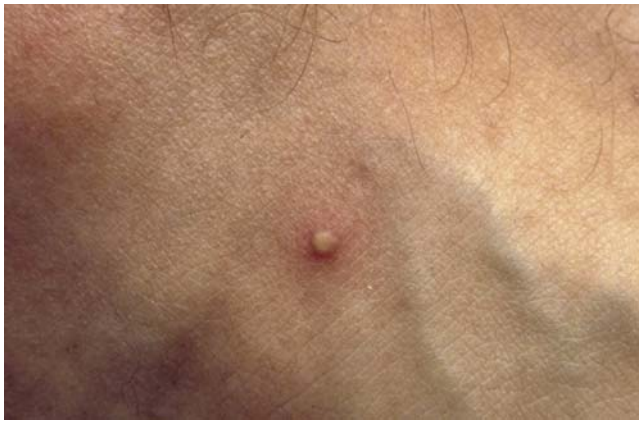


FIGURE 22.28 Fire ant sting by *Solenopsis invicta* on human ankle, showing characteristic sterile pustule formed at sting site, 24 h after sting. *Courtesy of M. Horton.*

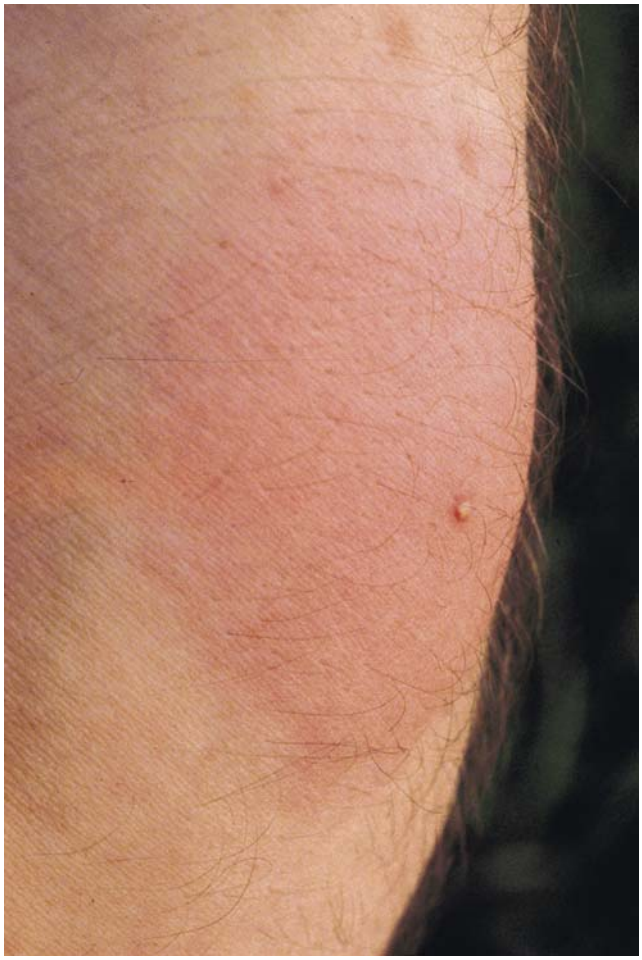


FIGURE 22.29 Fire ant sting by *Solenopsis invicta* on human forearm, with localized swelling and inflammation, 15 h following sting. *Photograph by Gary R. Mullen.*

TABLE 22.4 Descriptions of the Four General Types of Reactions to Hymenopteran Stings

Type	Duration	Response
Local	~2 h	Redness, itching, swelling, pain, wheal forms at sting site
Large local	2–5 d	Painful swelling (<10 cm) of sting site or entire extremity; general malaise or ill feeling, palpitation of heart, elevated blood pressure
Systemic	10 min–3 weeks	Reactions to areas beyond the sting site; normal symptoms plus hives, respiratory distress, laryngeal swelling, gastrointestinal distress, hypotension, cutaneous uticaria, widespread edema, anaphylactic shock, sometimes leading to death
Toxic	Hours–months	Destruction of muscle and red blood cells, blood pressure drop, kidney failure

around the sting site. In most cases a characteristic vesicle, 3–5 mm in diameter and containing a clear fluid, develops within 6–24 h at each sting site. Often fire ants can sting repeatedly while pivoting at the bite site, thus producing a semicircle of pustules. The fluid becomes cloudy, forming a white pustule (Figs. 22.28 and 22.29). The pustules usually disappear within a few days. They are replaced by a discolored lesion, resulting from tissue necrosis caused by alkaloids in the venom. These lesions can be intensely pruritic, can persist for weeks or months, and are subject to secondary bacterial infections when they are scratched or otherwise broken.

Reactions to insect stings have been variously classified (Schmidt, 1992; Reisman, 1994a) but can be divided into four categories: local, large local, systemic, and toxic reactions (Table 22.4). Local reactions are normal for most people and include immediate pain and/or burning at the sting site, development of a flare and wheal, redness (Fig. 22.30), and swelling that is limited to the sting site (<10 cm diameter). Later, itching at the sting site usually occurs. A large local reaction involves painful swelling at the site (>10 cm diameter) and even an associated extremity (Fig. 22.31); this reaction is not considered serious. These are common responses that are not life-threatening. They may be accompanied by systemic, cutaneous reactions (e.g., hives) on parts of the body other than where the sting occurs. These two types of reactions are the more common, occurring perhaps in a quarter of the general population. Systemic reactions, the most serious, tend to occur only in 1%–3% of the population (Pucci et al., 2015).

Systemic reactions are more generalized responses that induce reactions away from the sting site. The most serious of these are allergic reactions, which can be fatal. Allergic reactions typically occur after a second or subsequent sting by the same or closely related species. The first sting causes the body to produce venom specific IgE antibodies to specific proteins in the venoms (Casale and Burks, 2014). Later, when a hypersensitized individual is stung again, the immune system overreacts to the presence of the same venom proteins. These stinging episodes can result in **anaphylaxis**, a sudden drop of blood pressure and respiratory distress

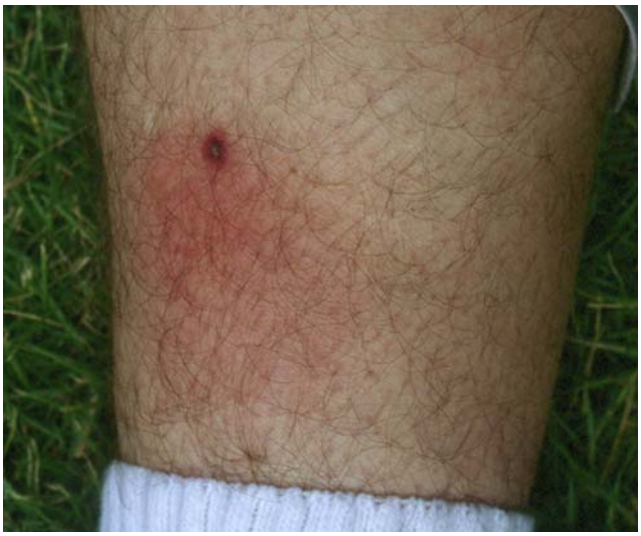


FIGURE 22.30 Reaction to sting of the southern yellowjacket (*Vespula squamosa*) on lower leg, with reddening and localized hemorrhaging at sting site. *Photograph by Gary R. Mullen.*

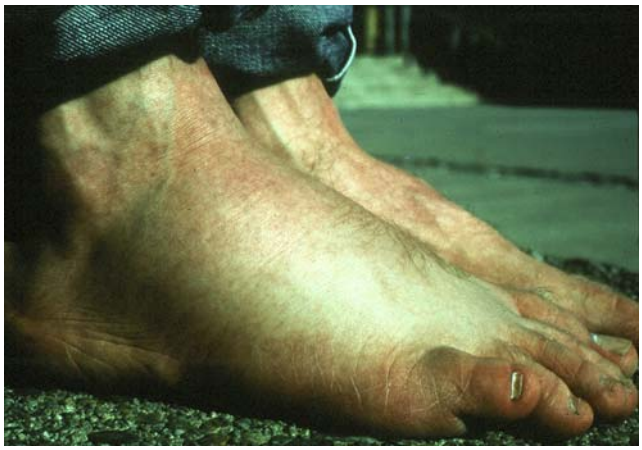


FIGURE 22.31 Swelling of human foot as a result of yellowjacket (*Vespula* sp.) sting. *Courtesy of Roger D. Akre.*

triggered by the immunological response. In some cases this can result in **anaphylactic shock** and death. Such lethal reactions have been reported for stings of vespines, paper wasps, honey bees, and fire ants. It is difficult to predict who may be at risk of a severe systematic reaction. However, an individual who has had a previous systematic reaction is at a high risk of another systemic reaction.

Deaths from hymenopterous stings are usually due to respiratory failure (70%). Other reported causes of mortality are anaphylaxis (15%), cardiovascular collapse (9%), and neurological complications (6%) (Schmidt, 1986b). Death from stings, when it occurs, can be very rapid, within 20 min of the sting, with 60% of deaths occurring in less than 1 h. Therefore it is imperative in cases of severe allergic responses that treatment (e.g., injection of epinephrine) be administered as soon as possible after the sting occurs. In about 20% of these systemic episodes, the initial reaction is followed by a recurrence of symptoms up to 8 h or even days later. Thus, patients should be closely monitored in order to respond to such delayed or biphasic episodes (Casale and Burks, 2014). A few deaths attributed to toxic reactions have been reported in people and animals receiving hundreds to thousands of stings from the Africanized honey bee or yellowjackets. The victims in these cases usually are either young children or people who are incapacitated or restrained in some way in close proximity to a disturbed colony.

Although relatively few people are killed by hymenopteran stings, many people suffer some degree of sting hypersensitivity. Estimates of the incidence of hypersensitivity reactions, from mild to severe systemic responses, vary from 0.15% to 5% in the United States (Schmidt, 1992). An incidence of 2% in this case represents about 6 million people. Fewer than 1% of fire ant sting victims experience anaphylactic shock when stung. However, the frequency of serious reactions is not well documented for other stinging hymenoptera, and the importance of the species involved differs with the region. In the United States, for example, yellowjackets cause more serious reactions than do honey bees in the Pacific Northwest and Washington, D.C., areas. Paper wasps are most important in Texas and the southwestern states, whereas fire ants cause more allergic reactions and deaths than do honey bees in the southeastern states (Camazine, 1988).

A unique type of allergy has been associated with honey bees. Family members of beekeepers can develop an intense allergy to constituents of honey bee venom while laundering venom-impregnated garments. Most of these people do not realize they have become sensitized to the venom because they have had little or no direct contact with the bees themselves.

Several approaches can be taken in the treatment of sting victims. Embedded stings should be removed as soon as possible to stop any further venom injection. The sting apparatus should be scraped off as close as possible to the skin surface, not pinched off, as the latter tends to squeeze more venom into the sting site. Immediate removal of the embedded sting apparatus reduces the severity of the sting reaction, including pain and local swelling.

The sting site should be washed with soap and water to minimize the possibility of secondary infection. Ice packs or cool compresses, topical lidocaine, corticosteroid lotions, and antihistamines are recommended treatments for local sting reactions. The use of meat tenderizers, which generally contain a proteolytic enzyme (e.g., papain), is questionable, with no published data from controlled experiments to support their effectiveness. Other home remedies such as baking soda, wet salt, moistened tobacco, 10% ammonia solution, and commercial sting relievers have been suggested to help to reduce swelling and pain when applied to the sting sites (Weathersby, 1984); however, most have not been tested for their effectiveness. Wet salt was tested and was shown not to be beneficial (Weathersby, 1984). A topical aspirin treatment did not reduce the duration of pain or swelling in bee and wasp stings, and even increased the duration of redness (Balit et al., 2003).

People with suspected or known venom hypersensitivity should be evaluated by an allergist-immunologist using skin testing or in vitro testing for specific venom IgE antibodies in the blood serum (Golden et al., 2017). A skin test involves subcutaneous injection of diluted venom extracts (e.g., honey bee, yellowjackets, wasps) or whole-body extract for fire ants. Some individuals who have had a severe reaction should be evaluated for mast cell disorders and levels of basal serum tryptase. All tests should be done in an allergy clinic where immediate medical treatment is available should adverse reactions occur.

Most physicians recommend that persons with demonstrated hypersensitivity wear an identification tag and carry a small emergency sting kit containing antihistamines and a syringe of epinephrine. Such kits are available with a physician's prescription. Hypersensitive people at high risk of a fatal reaction should consider immunotherapy. These individuals can be desensitized by a series of injections using attenuated doses of the appropriate venom. Such treatment gradually builds up the individual's tolerance to the venom and helps to prevent subsequent systemic reactions. Desensitization programs can be expensive and sometimes require 3−5 years.

Additional information about hymenopteran venoms, clinical aspects of stings, allergic reactions, and treatment of sting victims is provided by Piek (1986b), Schmidt (1992), Charpin et al. (1994), Levine and Lockey (1995), Meier (1995), Mosbech (1995), Fitzgerald and Flood (2007), and Golden et al. (2017).

VETERINARY IMPORTANCE

Comparatively little is known about the effects of hymenopteran stings on farm and game animals, except for stings by fire ants. Imported fire ants (*S. invicta* and *S. richteri*) commonly attack and kill newborn wild animals such as rabbits, deer, quail and other ground-nesting birds, and even hatchling tortoises (Lofgren, 1986). The native fire ant in the United States, *S. xyloni*, is known to attack and kill newly hatched poultry. However, deaths due to *S. xyloni* have been rarely reported for other newborn farm animals. A few accounts of animal deaths have been attributed to stings of Africanized honey bee, usually involving dogs (Oliveria et al., 2007) (Fig. 22.27) or livestock, while restrained or enclosed near a hive. A massive stinging attack by eastern yellowjackets on a flamingo resulted in its death several hours later (Suedmeyer and Trupkiewicz, 2014). The German yellowjacket can injure the teats of milking cows by biting (Braverman et al., 1991), and the resulting lesions have been associated with outbreaks of mastitis in Israel (Shwimmer et al., 1995). Yellowjackets also have been observed cutting flesh from wounds on horses.

There is little information available on allergic sting reactions in nonhuman animals. Severe systemic reactions to stings of bees and wasps have been reported in dogs and cats (Boord, 2013). Fire ant stings to dogs do not develop pustules characteristic of those on humans, and there have been no reports of anaphylaxis in dogs (Rakich et al., 1993). Some dogs, when stung on the face by bees or wasps, develop enormous swelling of the head that may persist for a few days to a week.

Some ant species are intermediate hosts for parasitic helminths of vertebrates. *Formica* spp. are intermediate hosts for the lancet fluke (*Dicrocoelium dendriticum*), which infests the bile ducts of cattle, sheep, pigs, goats, horses, dogs, and occasionally humans. Pavement ant workers and *Pheidole* ants serve as intermediate hosts of the poultry tapeworms *Raillietina tetragona* and *R. echinobothrida* (Harwood and James, 1979).

PREVENTION AND CONTROL

Avoidance of stinging insects is the best approach to preventing envenomation. People with hypersensitivity should avoid areas with high concentrations of ants, bees, or yellowjackets and be very observant and alert when in areas where they are active or colonies are known to occur. People should be aware that in temperate climates, wasps are most abundant in late summer and early autumn. Knowledge of the nesting habits of species in one's environment is important to reducing encounters, such as the likely locations of paper wasp or yellowjacket nests, fire ant mounds, etc. Colonies of harvester ants can be easily avoided because the ants are slow moving and their nest mounds are very noticeable in the center of areas devoid of vegetation.

Certain colors of clothing should be avoided during seasons of high populations and activity of stinging insects. Foraging yellowjackets and bees are attracted to yellow. Dark-colored objects may be more attractive to attacking wasps. It is advisable to wear long pants, preferably with the legs tucked into boots, and to wear a long-sleeved shirt to protect the arms. Individuals at risk should avoid using perfumes, hair sprays, sweet-smelling lotions, aftershave lotions, hand and body lotions, and even certain suntan lotions. Some of the odors emanating from these materials attract foraging wasps and bees, and if disturbed they may sting. Paints containing isoamyl acetate, a component of the honey bee alarm pheromone, should not be used around honey bees. These materials should not be applied outdoors when and where stinging bees and wasps are likely to be attracted to them.

Repellents commonly used to deter mosquitoes and other biting insects are not effective against attacking ants, bees, and wasps. For example, the common insect repellent DEET has little deterrence to honey bees (Schmidt et al., 2003). However, studies have suggested that lotions containing plant essentials oils and some natural products may repel foraging yellowjackets from human food sources (Boeve et al., 2014, 2016).

Care should be taken not to provoke colonies by approaching too closely or creating substrate vibrations that might disturb the nest. Similarly, one should avoid using machines such as power tools and lawnmowers near nests, or slamming doors and windows in the vicinity of a nest. Once a colony is aroused, it is best to slowly back out of the area rather than to run. Wasps and bees defending their nests tend to attack nearby moving objects. However, once a person is stung, it is best to leave the area as quickly as possible before other nestmates are recruited to attack in response to a released alarm pheromone.

In cases of potential mass stinging attacks (e.g., Africanized honey bee or large colonies of yellowjackets), it is best to run away from the nest. The nose and eyes should be covered without blocking vision to avoid stings directed to one's head. Running through brushy vegetation or near and around objects is advised if possible. Running for some distance, such as hundreds of meters, may be required to gradually leave the attacking wasps or bees behind. If you remain in the area, the potential exists to receive many dozens to hundreds of stings.

Insecticides, especially aerosol sprays containing a quick knock-down agent and long-lasting pesticides, can be used to kill colonies of yellowjackets and paper wasps. While these aerosols can propel toxicants up to 6–7 m and are designed primarily for treating aerial nests, they work equally well on colonies nesting underground. Nest destruction should be conducted at night when wasps are

least active and most, if not all, the individuals are in the nest. If insecticides are applied during the daytime, the nest entrance should not be blocked after treatment so that returning wasps can readily enter the nest and contact the toxicant. Long-lasting residual insecticides will kill workers emerging from capped cells after the initial treatment. Some aerosol-type sprays do not contain a long-lasting toxicant and are not effective on later-emerging adults. Controlling any nest, especially large colonies of paper wasps and yellowjackets, can be difficult and dangerous, so it is often best left to experienced pest control operators. Protective clothing, such as a bee suit with veil and appropriate footwear, should be worn when destroying colonies.

Only under certain circumstances is there good reason to kill honey bee colonies. When necessary, local beekeepers may be willing to collect swarms near houses or other sites of human activity. Colonies of honey bees nesting in the wall voids of houses probably should be destroyed. They can be killed with the combination of a quick-knockdown insecticide directed into the nest entrance, followed by an application of insecticidal dust into the voids through openings such as electrical plate outlets or holes drilled into the wall. Destruction of well-established honey bee colonies in walls of buildings can result in the stored honey dripping through the walls or ceiling. In such cases, the colony and honey combs should be physically removed after the bees are killed or otherwise removed.

Control of some social insects is best accomplished by the use of **baits** that incorporate a slow-acting insecticide. Foragers are attracted to the bait, return to the colony with the toxic bait, and spread the material throughout the colony by trophallaxis. Several toxicants that show promise for control are classified as insect growth regulators, but several other slow-acting toxicants with direct poisoning action are also effective. Although ants in heavily used areas such as picnic grounds can be killed by direct application of insecticides to the nest, it is best to use some type of carbohydrate (sweet) or fatty baits that contains a slow-acting poison. Baits are the control of choice for pharaoh's ants and pavement ants because treatments with insecticidal perimeter sprays and barriers in and around buildings tend to cause the ants to disperse or become trapped inside a structure, making them more difficult to control (Rust and Su, 2012). Traps baited with pheromones are used to attract honey bee swarms to monitor the movement of the Africanized honey bee.

Control measures for fire ants include direct application of insecticides to the nest or small areas, toxic baits and insect growth regulators, and biological control agents such as pathogens and parasitoids. Also, botanicals and other natural compounds are being investigated as fire ant repellents and/or biopesticides and in modification of bait delivery systems.

Control of scavenging yellowjackets may be accomplished by trapping systems using appropriate attractive baits containing a persistent toxicant (e.g., fipronil). Scavenging *Vespula* spp. are attracted to meat baits. A simple meat trap with fish or ham suspended over a pan of detergent water can be used to drown thousands of attracted wasps. Workers cut off pieces of meat that are too heavy for them to carry and drop into the water as they try to fly away. Each day, drowned wasps should be removed, and the detergent water and the meat should be replaced. Toxic meat baits have been successfully used to reduce pestiferous yellowjacket populations in several countries, including the United States, Argentina, and New Zealand.

Because meat baits spoil and lose their attractiveness quickly and the impregnated insecticides may harm nontarget species, potential synthetic attractants have been investigated. The western yellowjacket, as well as the California and southern yellowjackets, can be lured into traps containing the synthetic attractants hexyl butyrate, heptyl butyrate, or octyl butyrate. Two co-attractants, isobutanol and acetic acid, have been shown to be effective baits for nearly all scavenging yellowjacket species in North America, as well as the baldfaced hornet, the European hornet, and some species of paper wasps. Intensive trapping with heptyl butyrate has reduced local populations of some species (e.g., *V. pensylvanica*) but does not effectively provide area-wide control.

For further information on avoidance and control measures for various stinging hymenopterans, see Akre and MacDonald (1986), Edwards et al. (2017), and Liang and Pietri (2017) for yellowjackets; Drees and Vinson (1993) and Lofgren (1986) for fire ants; and Rust and Su (2012) for social insects in general.

REFERENCES AND FURTHER READING

Addesso, K. M., Oliver, J. B., O'Neal, P. A., & Youssef, N. (2017). Efficacy of Nootka oil as a biopesticide for management of imported fire ants (Hymenoptera: Formicidae). *Journal of Economic Entomology, 110*, 1547−1555.

Agostinucci, W., Cardoni, A. A., & Rosenberg, R. (1981). Effect of papain on bee venom toxicity. *Toxicon, 19*, 851−855.

Aili, S. R., Touchard, A., Escoubas, P., Padula, M. P., Orivel, J., Dejean, A., et al. (2014). Diversity of peptide toxins from stinging ant venoms. *Toxicon, 92*, 166−174.

Akre, R. D. (1982). Social wasps. In H. R. Hermann (Ed.), *Social insects* (Vol. 4, pp. 1−105). New York: Academic, 385 p.

Akre, R. D., Grain, A., MacDonald, J. F., Landolt, P. J., & Davis, H. G. (1981). The yellowjackets of America north of Mexico. In *U.S. Department of Agricultural Handbook No. 552*, 102 p.

Akre, R. D., & MacDonald, J. F. (1986). Biology, economic importance, and control of yellowjackets. In S. B. Vinson (Ed.), *Economic impact and control of social insects* (pp. 353−412). New York: Praeger, 432 p.

Akre, R. D., & Reed, H. C. (1984). Biology and distribution of social Hymenoptera. In A. T. Tu (Ed.), *Insect poisons, allergens, and other invertebrate venoms: handbook of natural toxins* (Vol. 2, pp. 3–47). New York: Marcel Dekker, 732 p.

Alexander, B., & Michener, C. D. (1995). Phylogenetic studies of the families of short-tongued bees (Hymenoptera: Apoidea). *University of Kansas Science Bulletin, 55*, 377–424.

AntWeb. Available from: https://www.antweb.org/. Accessed 14 May 2017

Antmaps. http://antmaps.org/?mode=species&species=Solenopsis.invicta. Accessed 11-30-2017.

Balit, C. R., Isbister, G. K., & Buckley, N. A. (2003). Randomized controlled trial of topical aspirin in the treatment of bee and wasp stings. *Journal of Toxicology - Clinical Toxicology, 41*(6), 801–808.

Banks, B. E. C., & Shipolini, R. A. (1986). Chemistry and pharmacology of honey-bee venom. In T. Piek (Ed.), *Venoms of the Hymenoptera: biochemical, pharmacological and behavioural aspects* (pp. 329–416). San Diego, CA: Academic, 570 p.

Barbosa, A. N., Boyer, L., Chippaux, J. P., Medolago, N. B., Caramori, C. A., Paixão, A. G., et al. (2017). A clinical trial protocol to treat massive Africanized honeybee (*Apis mellifera*) attack with a new apilic antivenom. *Journal of Venomous Animals and Toxins Including Tropical Diseases, 23*, 14–24.

Boevé, J. L., Eertmans, F., Adriaens, E., & Rossel, B. (2016). Field method for testing repellency of an Icaridin-containing skin lotion against vespid wasps. *Insects, 7*(2), 22.

Boevé, J. L., Honraet, K., & Rossel, B. (2014). Screening of repellents against vespid wasps. *Insects, 5*(1), 272–286.

Bohart, R. M., & Menke, S. (1976). *Sphecid wasps of the world: A generic revision*. Berkeley: Univ. California Press, 695 p.

Bolton, B. (1994). *Identification guide to the ant genera of the world*. Harvard Univ. Press, 224 p.

Boord, M. J. (2013). Venomous insect hypersensitivity. In C. Noli, A. Foster, & W. Rosenkrantz (Eds.), *Veterinary allergy*. Oxford, UK: John Wiley and Sons, Ltd, 907 p.

Braverman, Y., Marcusfeld, O., Adler, H., & Yakobson, B. (1991). Yellowjacket wasps can damage cow's teats by biting. *Medical and Veterinary Entomology, 5*, 129–130.

Brewer, R. D., Morris, P. D., & Cole, T. B. (1994). Hurricane-related emergency department visits in an inland area: An analysis of the public health impact of Hurricane Hugo in North Carolina. *Annals of Emergency Medicine, 23*, 731–736.

Buck, M., Cobb, T. P., Stahlhut, J. K., & Hanner, R. H. (2012). Unravelling cryptic species diversity in eastern Nearctic paper wasps, *Polistes (Fuscopolistes)*, using male genitalia, morphometrics and DNA barcoding, with descriptions of two new species (Hymenoptera: Vespidae). *Zootaxa, 3502*, 1–48.

Buck, M. C., Marshall, S. A., & Cheung, D. K. B. (2008). Identification atlas of the Vespidae (Hymenoptera, Aculeata) of the northeastern Nearctic region. *Canadian Journal of Arthropod Identification, 5*, 1–138.

Camazine, S. (1988). Hymenoptera stings: Reactions, mechanisms, and medical treatment. *Bulletin of the Entomological Soceity of America, 34*, 17–21.

Carpenter, J. M. (1999). Taxonomic notes on paper wasps (Hymenoptera, Vespidae, Polistinae). *American Museum Novitates, 3259*.

Casale, T. B., & Burks, A. W. (2014). Hymenoptera-sting hypersensitivity. *New England Journal of Medicine, 370*(15), 1432–1439.

Charpin, D., Birnbaum, J., & Vervloet, D. (1994). Epidemiology of Hymenoptera allergy. *Clinical and Experimental Allergy, 24*, 1010–1015.

Cole, A. C. (1968). *Pogonomyrmex harvester ants: A study of the genus in North America*. Knoxville: University of Tennessee Press, 222 p.

Drees, B. M., & Vinson, S. B. (1993). Fire ants and their management. *Texas Agricultural Extension Service B, 1536*, 18 p.

Edwards, E., Toft, R., Joice, N., & Westbrooke, I. (2017). The efficacy of Vespex® wasp bait to control *Vespula* species (Hymenoptera: Vespidae) in New Zealand. *International Journal of Pest Management*, 1–7.

Edwards, J. P. (1986). The biology, economic importance and control of the Pharaoh's ant, *Monomorium pharaonis* (L.). In S. B. Vinson (Ed.), *Economic impact and control of social insects*. New York: Praeger, 432 p.

Fisher, B. L., & Cover, S. P. (2007). *Ants of North America: A guide to genera*. Berkeley: University of California Press, 194 p.

Fitzgerald, K. T., & Flood, A. A. (2006). Hymenoptera stings. *Clinical Techniques in Small Animal Practice, 21*, 194–204.

Fowler, H. G., Bueno, O. C., Sadatsune, T., & Montelli, A. C. (1993). Ants as potential vectors of pathogens in hospitals in the state of São Paulo, Brazil. *Insect Science and Its Application, 14*, 367–370.

Franks, N. R. (2009). Ants. In V. H. Resh, & R. T. Carde (Eds.), *Encyclopedia of insects* (2nd ed.). Oxford: Elsevier Science & Technology [Online]. Available from: http://search.credoreference.com/content/entry/estinsects/ants/0?institutionId=5550. Accessed on 21 March 2017.

Gardner, W. A., Diffie, S., Vander Meer, R. K., & Brinkman, M. A. (2008). Distribution of the fire ant (Hymenoptera: Formicidae) hybrid in Georgia. *Journal of Entomological Science, 43*(1), 133–137.

Gauld, I. D., & Bolton, B. (1988). *The Hymenoptera*. Oxford University Press, 332 p.

Gillaspy, J. E. (1986). *Polistes* wasps: Biology and impact on man. In S. B. Vinson (Ed.), *Economic impact and control of social insects* (pp. 332–352). New York: Praeger, 432 p.

Goddard, J. (2012). *Physician's guide to arthropods of medical importance* (6th ed.). Boca Raton: CRC, 480 p.

Golden, D. B., Demain, J., Freeman, T., Graft, D., Tankersley, M., Tracy, J., et al. (2017). Stinging insect hypersensitivity. *Annals of Allergy, Asthma, & Immunology, 118*(1), 28–54.

Gotzek, D., Brady, S. G., Kallal, R. J., & LaPolla, J. S. (2012). The importance of using multiple approaches for identifying emerging invasive species: The case of the Rasberry crazy ant in the United States. *PLoS One, 7*(9), e45314.

Goulet, H., & Huber, J. T. (1993). *Hymenoptera of the world: An identification guide to families*. Ottawa: Agriculture Canada, Research Branch, 668 p.

Greene, A., Breisch, N. L., Golden, D. B. K., Kelly, D., & Douglass, L. W. (2012). Sting embedment and avulsion in yellowjackets (Hymenoptera: Vespidae): A functional equivalent to autotomy. *American Entomologist, 58*(1), 50–57.

Harwood, R. F., & James, M. T. (1979). *Entomology in human and animal health* (7th ed.). New York: MacMillan Co., 548 p.

Hoffman, D. R. (1984). Insect venom allergy, immunology, and immunotherapy. In A. T. Tu (Ed.), *Insect poisons, allergens, and other invertebrate venoms. Handbook of natural toxins* (Vol. 1, pp. 187–223). New York: Marcel Dekker, 732 p.

Hölldobler, B., & Wilson, E. O. (1990). *The ants*. Cambridge: Belknap/Harvard Univ. Press, 732 p.

Hölldobler, B., & Wilson, E. O. (2008). *The superorganism: The beauty, strangeness, and elegance of insect societies*. W.H. Norton, Co, 544 p.

Kimsey, L., & Carpenter, J. (2012). The Vespinae of North America (Vespidae, Hymenoptera). *Journal of Hymenoptera Research, 28*, 37.

Klotz, J., Hansen, L., Pospischil, R., & Rust, M. (2008). *Urban ants of North America and Europe: Identification, biology, and management*. Cornell University Press, 224 p.

Landolt, P. J. (1998). Chemical attractants for trapping yellowjackets *Vespula germanica* and *Vespula pensylvanica* (Hymenoptera: Vespidae). *Environmental Entomology, 27*, 1229−1234.

Landolt, P. J., Reed, H. C., Aldrich, J. R., Antonelli, A. L., & Dickey, C. (1999). Social wasps (Hymenoptera: Vespidae) trapped with acetic acid and isobutanol. *Florida Entomologist, 82*, 609−614.

Langley, R., Mack, K., Haileyesus, T., Proescholdbell, S., & Annest, J. L. (2014). National estimates of noncanine bite and sting injuries treated in US hospital emergency departments, 2001−2010. *Wilderness and Environmental Medicine, 25*(1), 14−23.

LeBrun, E. G., Jones, N. T., & Gilbert, L. E. (2014). Chemical warfare among invaders: A detoxification interaction facilitates an ant invasion. *Science, 343*(6174), 1014−1017.

Levine, M. I., & Lockey, R. F. (Eds.). (1995). *Monograph on insect allergy*. Pittsburgh: Dave Lambert Assoc.

Liang, D., & Pietri, J. E. (2017). Enhanced trapping of yellowjacket wasps (Hymenoptera: Vespidae) via spatial partitioning of attractants. *Insects, 8*(1), 17.

Lofgren, C. S. (1986). The economic importance and control of imported fire ants in the United States. In S. B. Vinson (Ed.), *Economic impact and control of social insects* (pp. 227−256). New York: Praeger, 432 p.

MacKay, W. P. (1990). The biology and economic impact of *Pogonomyrmex* harvester ants. In R. K. Vander Meer, K. Jaffe, & A. Cedeno (Eds.), *Applied myrmecology: A world perspective* (pp. 533−543). Boulder, Colorado: Westview, 715 p.

Manley, D. G. (1991). The velvet ants (Hymenoptera: Mutillidae) of South Carolina. *South Carolina Agricultural Experiment Station, Technical Bulletin, 1100*, 55 p.

van Marle, J., & Piek, T. (1986). Morphology of the venom apparatus. In T. Piek (Ed.), *Venoms of the Hymenoptera: Biochemical, pharmacological and behavioural aspects* (pp. l7−44). San Diego: Academic, 583 p.

Meier, J. (1995). Biology and distribution of hymenopterans of medical importance, their venom apparatus and venom composition. In J. Meier, & J. White (Eds.), *Handbook of clinical toxicology of animal venoms and poisons* (pp. 331−348). Boca Rotan, FL: CRC Press, 768 p.

Menzel, T. O., & Nebeker, T. E. (2008). Distribution of hybrid imported fire ants (Hymenoptera: Formicidae) and some native ant species in relation to local environmental conditions and interspecific competition in Mississippi forests. *Annals of the Entomological Society of America, 101*(1), 119−127.

Michener, C. D. (1974). *The social behavior of the bees*. Cambridge, Massachusetts: Belknap/Harvard University Press, 404 p.

Michener, C. D. (2007). *The bees of the world*. Cambridge, Massachusetts: Belknap Press, 963 p.

Michener, C. D., McGinley, R. J., & Danforth, B. N. (1994). *The bee genera of North and Central America (Hymenoptera Apoidea)*. Washington, DC: Smithsonian Institution Press, 209 p.

Morrison, L. W., Porter, S. D., Daniels, E., & Korzuhkin, M. D. (2004). Potential global range expansion of the invasive fire ant, *Solenopsis invicta*. *Biological Invasions, 6*, 183−191.

Mosbech, H. (1995). Clinical toxicology of hymenopteran stings. In J. Meier, & J. White (Eds.), *Handbook of clinical toxicology of animal venoms and poisons* (pp. 349−359). Boca Rotan, FL: CRC Press, 768 p.

Nakajima, T. (1984). Biochemistry of vespid venoms. In A. T. Tu (Ed.), *Insect poisons, allergens, and other invertebrate venoms. Handbook of natural toxins* (Vol. 2, pp. 109−133). New York: Marcel Dekker, 732 p.

Nakajima, T. (1986). Pharmacological biochemistry of vespid venoms. In T. Piek (Ed.), *Venoms of the Hymenoptera: Biochemical, pharmacological and behavioural aspects* (pp. 309−327). San Diego: Academic, 583 p.

Nelder, M. P., Paysen, E. S., Zungoli, P. A., & Benson, E. P. (2006). Emergence of the introduced ant *Pachycondyla chinensis* (Formicidae: Ponerinae) as a public health threat in the Southeastern United States. *Journal of Medical Entomology, 43*, 1094−1098.

Oi, D. H. (2008). Pharoah ants and fire ants. In X. Bonnefoy, H. Kampen, & K. Sweeney (Eds.), *Public health significance of urban pests* (pp. 175−207). Geneva: World Health Organization, 569 p.

Oliveira, E. C., Pedroso, R. M. O., Meirelles, A. E. W. B., Pescador, C. A., Gouvea, A. S., & Driemeier, D. (2007). Pathological findings in dogs after multiple Africanized bee stings. *Toxicon, 49*, 1214−1218.

O'Neill, K. M. (2001). *Solitary wasps: Behavior and natural history*. Geneva: Cornell University, 406 p.

O'Neill, M. E., Mack, K. A., & Gilchrist, J. (2007). Epidemiology of noncanine bite and sting injuries treated in U.S. emergency departments, 2001-2004. *Public Health Reports, 122*, 764−775.

Pantoja, L. D. M., Moreira Filho, R. E., Brito, E. H. S., Aragão, T. B., Brilhante, R. S. N., Cordeiro, R. D. A., et al. (2009). Ants (Hymenoptera: Formicidae) as carriers of fungi in hospital environments: An emphasis on the genera *Tapinoma* and *Pheidole*. *Journal of Medical Entomology, 46*(4), 895−899.

Pegula, S., & Kato, A. (2014). Fatal injuries and nonfatal occupational injuries and illnesses involving insects, arachnids, and mites. In *Beyond the numbers* (Vol. 3, No. 17). Washington, DC: Bureau of Labor Statistics.

Piek, T. (1984). Pharmacology of Hymenoptera venoms. In A. T. Tu (Ed.), *Insect poisons, allergens, and other invertebrate venoms: Handbook of natural toxins* (Vol. 2, pp. 135−185). New York: Marcel Dekker, 732 p.

Piek, T. (1986a). Venoms of bumble-bees and carpenter-bees. In T. Piek (Ed.), *Venoms of the Hymenoptera: Biochemical, pharmacological and behavioural aspects* (pp. 417−424). San Diego: Academic, 583 p.

Piek, T. (1986b). In *Venoms of the Hymenoptera. Biochemical, pharmacological and behavioural aspects*. San Diego: Academic, 583 p.

Pucci, S., D'Alò, S., De Pasquale, T., Illuminati, I., Makri, E., & Incorvaia, C. (2015). Risk of anaphylaxis in patients with large local reactions to Hymenoptera stings: A retrospective and prospective study. *Clinical and Molecular Allergy, 13*, 21−23.

Rakich, P. M., Latimer, K. S., Mispagel, M. E., & Steffens, W. L. (1993). Clinical and histological characterization of cutaneous reactions to stings of the imported fire ant (*Solenopsis invicta*) in dogs. *Veterinary Pathology, 30*, 555−559.

Reed, H. C., & Landolt, P. J. (1991). Swarming of paper wasp (Hymenoptera: Vespidae) sexuals at towers in Florida. *Annals of the Entomological Society of America, 84*, 628–635.

Reisman, R. E. (1994a). Insect stings. *New England Journal of Medicine, 331*, 523–527.

Reisman, R. E. (1994b). Venom hypersensitivity. *The Journal of Allergy and Clinical Immunology, 94*, 651–658.

Rhoades, R. B., Stafford, C. T., & James, F. K., Jr. (1989). Survey of fatal anaphylactic reactions to imported fire ant stings. *The Journal of Allergy and Clinical Immunology, 84*, 159–162.

Ross, K. G., & Matthews, R. W. (Eds.). (1991). *The social biology of wasps*. Ithaca, New York: Comstock Publication Association, 678 p.

Rosselli, D., & Wetterer, J. K. (2017). Stings of the ant *Wasmannia auropunctata* (Hymenoptera: Formicidae) as cause of punctate corneal lesions in humans and other animals. *Journal of Medical Entomology, 20*, 1–3.

Rust, M. K., & Su, N. Y. (2012). Managing social insects of urban importance. *Annual Review of Entomology, 57*, 355–375.

Schmidt, J. O. (1986a). Allergy to Hymenoptera venoms. In T. Piek (Ed.), *Venoms of the Hymenoptera: Biochemical, pharmacological and behavioural aspects* (pp. 509–546). San Diego: Academic Press, 583 p.

Schmidt, J. O. (1986b). Chemistry, pharmacology, and chemical ecology of ant venoms. In T. Piek (Ed.), *Venoms of the Hymenoptera. Biochemical, pharmacological and behavioural aspects* (pp. 425–508). San Diego: Academic Press, 583 p.

Schmidt, J. O. (1986c). Hymenoptera envenomation. In G. W. Frankie, & C. S. Koehler (Eds.), *Urban entomology: Interdisciplinary perspectives* (pp. l87–220). New York: Praeger, 493 p.

Schmidt, J. O. (1990a). Africanized and European honey bee venoms: Implications for beekeepers and the public. *American Bee Journal, 130*, 810–811.

Schmidt, J. O. (1990b). Hymenoptera venoms: Striving toward the ultimate defense against vertebrates. In D. L. Evans, & J. O. Schmidt (Eds.), *Insect defenses: Adaptive mechanisms and strategies of prey and predator* (pp. 390–395). Albany, New York: State University of New York Press, 482 p.

Schmidt, J. O. (1992). Allergy to venomous insects. In J. Graham (Ed.), *The hive and the honey bee*. Hamilton, Illinois: Dadant and Sons, 1324 p.

Schmidt, J. O. (2009). Venom. In V. H. Resh, & R. T. Carde (Eds.), *Encyclopedia of insects* (2nd ed.). Oxford: Elsevier Science & Technology [Online] Available from: http://search.credoreference.com/content/entry/estinsects/venom/0?institutionId=5550. Accessed on 21 March 2017.

Schmidt, J. O. (2016). *The sting of the wild*. John Hopkins University Press, 280 p.

Schmidt, J. O., & Blum, M. (1978). A harvester ant venom: Chemistry and pharmacology. *Science, 200*, 164–166.

Schmidt, J. O., Johnston, A. N., Ginter, D. L., & Spangler, H. G. (2003). Olfactory stimulation of Africanized honey bee (Hymenoptera: Apidae) attacks by insect repellents. *Journal of Medical Entomology, 40,*(3), 275–278.

Schmidt, J. O., Menke, G. C., Chen, T. M., & Pinnas, J. L. (1984). Demonstration of cross-allergenicity among harvester ant venoms using RAST and RAST inhibition. *The Journal of Allergy and Clinical Immunology, 73*(1, part 2), 158.

Schumacher, M. J., & Egen, N. B. (1995). Significance of Africanized bees for public health. *Archives of Internal Medicine, 155*, 2038–2043.

deShazo, R. D., Williams, D. F., & Moak, E. S. (1999). Fire ant attacks on residents in health care facilities: A report of two cases. *Annals of Internal Medicine, 131*, 424–429.

Shipolini, R. A. (1984). Biochemistry of bee venom. In A. T. Tu (Ed.), *Insect poisons, allergens, and other invertebrate venoms. Handbook of natural toxins* (Vol. 2, pp. 49–85). New York: Marcel Dekker, 732 p.

Shwimmer, A., Shpigel, N. Y., Yeruham, I., & Saren, A. (1995). Epidemiological and bacteriological aspects of mastitis associated with yellowjacket wasp teat lesions in Israeli dairy cows. *Proceedings of the Third International Dairy Federation International Mastitis Seminar*, 100–102.

Smith, M. L. (2014). Honey bee sting pain index by body location. *Peer J, 2*, e338.

Spivak, M., Fletcher, D. J. C., & Breed, M. D. (1991). In *The 'African' honey bee*. Boulder, Colorado: Westview, 435 p.

Starr, C. K. (1985). A simple pain scale for field comparison of Hymenoptera stings. *Journal of Entomological Science, 20*, 225–232.

Suedmeyer, W. K., & Trupkiewicz, J. G. (2014). Fatal envenomation of a Chilean Flamingo (*Phoenicopterus chilensis*) from eastern yellow jacket wasps (*Vespula maculifrons*). *Journal of Avian Medicine and Surgery, 28*(4), 330–335.

Taber, S. W. (1998). *The world of harvester ants*. College Station, Texas: Texas A&M University Press. xvii + 213 pp.

Taber, S. W. (2000). *Fire ants*. College Station, TX: Texas A&M University Press, 368 p.

Touchard, A., Aili, S. R., Fox, E. G. P., Escoubas, P., Orivel, J., Nicholson, G. M., et al. (2014). The biochemical toxin arsenal from ant venoms. *Toxins, 8*, 1–30.

Tschinkel, W. R. (2006). *The fire ants*. Cambridge, MA: Harvard University Press, 723 p.

Tu, A. T. (1984). Insect poisons, allergens, and other invertebrate venoms. In A. T. Tu (Ed.), *Handbook of natural toxins* (Vol. 2, pp. 49–85). New York: Marcel Dekker, 732 p.

USDA. (2016). *Imported fire ant quarantine detail*. https://www.aphis.usda.gov/plant_health/plant_pest_info/fireants/downloads/fireant.pdf. Accessed on 6 November 2017.

Vander Meer, R. K., Breed, M. K., Winston, M. L., & Espelie, K. E. (1998). *Pheromone communication in social insects*. Boulder, Colorado: Westview, 368 p.

Vander Meer, R. K., Jaffe, K., & Cedeno, A. (Eds.). (1990). *Applied myrmecology: A world perspective*. Boulder, Colorado: Westview, 741 p.

Vander Meer, R. K., & Milne, D. E. (2017). Enhanced pest ant control with hydrophobic bait. *Journal of Economic Entomology, 110*, 567–574.

Van Emden, H. F. (2013). *Subclass Pterygota, division Endopterygota, order Hymenoptera (sawflies, ants, bees and wasps)—c. 120,000 described species. Handbook of agricultural entomology* (pp. 193–220).

Vinson, S. B. (1994). Impact of the invasion of *Solenopsis invicta* (Buren) on native food webs. In D. F. Williams (Ed.), *Exotic ants: Biology, impact, and control of introduced species* (pp. 240–258). San Francisco: Westview, 332 p.

Visscher, P. K., Vetter, R. S., & Camazine, S. (1996). Removing bee stings. *Lancet, 348*, 301–302.

Voss, J. D., Kugblenu, R., Salter, K., Johnson, L., & Reeves, W.K. (2016). Case series of 23 deaths from Hymenoptera stings among

United States Air Force populations. *Journal of Hymenoptera Research, 48*, 95.

Weathersby, A. B. (1984). Wet salt for envenomization. *Journal of the Georgia Entomological Society, 19*, 1–6.

Wetterer, J. K. (2013). Worldwide spread of the little fire ant, *Wasmannia auropunctata* (Hymenoptera: Formicidae). *Terrestrial Arthropod Reviews, 6*(3), 173–184.

Williams, D. F. (Ed.). (1994). *Exotic ants. Biology, impact, and control of introduced species*. Boulder: Westview Press, 332 p.

Williams, P. H., Thorp, R. W., Richardson, L. L., & Colla, S. R. (2014). *Bumble bees of North America: An identification guide*. Princeton University Press, 208 p.

Winston, M. L. (1992). *Killer bees: The Africanized honey bee in the Americas*. Cambridge: Harvard Univ. Press, 162 p.

Xu, Y., Huang, J., Zhou, A., & Zeng, L. (2012). Prevalence of *Solenopsis invicta* (Hymenoptera: Formicidae) venom allergic reactions in mainland China. *Florida Entomologist, 95*(4), 961–965.

Chapter 23

Scorpions (Scorpiones)

Gary R. Mullen[1] and W. David Sissom[2]

[1]*Department of Entomology and Plant Pathology, Auburn University, Auburn, AL, United States;* [2]*Department of Life, Earth and Environmental Sciences, West Texas A&M University, Canyon, TX, United States*

Scorpions represent an ancient group of arachnids that first appeared in the Silurian Period in the fossil record. They are believed by many arachnologists to be related to the now-extinct eurypterids, large chelicerates that lived in estuaries and coastal lagoons from the Ordovician Period through the Permian Period. Except for their smaller size, scorpions are notably similar in appearance to fossilized marine scorpions of the Silurian and Devonian. They probably first crawled onto land as air-breathing arachnids during the late Devonian and early Carboniferous 325−350 million years ago. Throughout recorded history scorpions have intrigued human cultures, being revered and attributed special powers by some and feared as sinister and ominous by others. Scorpion images appear as religious symbols; on seals, magical tablets, amulets, and boundary stones; and in the origin stories of ancient civilizations such as the Chaldeans and Egyptians. They also figured prominently in Greek mythology and as one of the 12 constellations or signs of the Zodiac.

Despite the many superstitions and misconceptions about scorpions that persist to this day, their reputation as dangerously venomous arthropods is generally overstated. Most scorpions are not aggressive and inflict only minor, transient pain and discomfort when they do sting, typically to defend themselves when threatened. There are, however, 40−50 species worldwide that pose significant health problems. About 25 species are considered to be capable of causing human deaths. Most of them occur in the Tropics and Subtropics or in arid regions of temperate zones.

TAXONOMY

Scorpion taxonomy has been unstable since the turn of this century. In his review of scorpion classification, Sissom (1990) listed 1,077 species, 117 genera, and nine families. During the 1990s, many new families were proposed that helped to create a clearer picture of phylogenetic relationships among the genera of scorpions. In their catalog of the scorpions of the world, Fet et al. (2000) listed 16 families, 154 genera, and 1,252 species of extant scorpions.

A phylogenetic study by Soleglad and Fet (2003) resulted in a widely debated revision of the higher classification of scorpions, which seemed to increase taxonomic instability rather than improve it. This arrangement was questioned by Prendini and Wheeler (2005), who challenged most of the changes made by Soleglad and Fet (2003) and essentially returned scorpion taxonomy to its pre-2003 state. The classification provided here follows that of Prendini and Wheeler (2005), with modifications of the included genera. For a listing of most of the valid genera, see the *Conspectus Genericus Scorpionorum* of Dupré (2007). The most recent estimate of scorpion taxonomic diversity is 19 families, 213 genera, and 2,363 species (modified from Rein, 2017, to follow Prendini and Wheeler, 2005); it should be noted that in the past 20 years more than 1,000 scorpion species have been described.

At least 106 species belonging to 21 genera and five families are known to occur in the continental United States; eight of these species are polytypic. A single species (*Paruroctonus boreus*) occurs in the extreme southern portions of British Columbia, Alberta, and Saskatchewan, Canada. Mexico, with the highest scorpion diversity of any country in the world, is home to 289 species spread among 37 genera and seven families.

The catalog by Fet et al. (2000), although now outdated, remains an invaluable resource. The production of a new catalog is in progress, with two parts already published (Kovařík, 2009; Kovařík and Onjanguran Affilastro, 2013). A partial key to the North American families is provided by Ponce et al. (2016). Generic identifications of North American scorpions are more problematic, as there is no single reference that can be used; some sources that may be

Medical and Veterinary Entomology. https://doi.org/10.1016/B978-0-12-814043-7.00023-6

consulted include Soleglad and Fet (2006, 2008), Gonzalez and Prendini (2013), and Ponce et al. (2016). For species identification of the North American fauna, see the following regional works: Nevada (Gertsch and Allred, 1965), Utah (Johnson and Allred, 1972), Idaho (Anderson, 1975), California (Hjelle, 1972; Williams, 1976); Baja California, Mexico (Williams, 1980), Florida (Muma, 1967), and mainland Mexico (Ponce et al., 2016).

Buthidae

This is the largest and most widespread scorpion family, with 90 currently recognized genera and more than 1,127 valid species. Buthids are found throughout the world with their greatest diversity in the Old World, especially the Afrotropical Region and the southern Palaearctic Region. Most of the scorpions that are dangerously venomous to humans and other animals belong to this family (Table 23.1). The important genera in this respect are *Androctonus, Buthus,* and *Leiurus* in northern Africa and western Asia, *Hottentotta* in Asia and India, *Parabuthus* in southern Africa, *Centruroides* in North America, and *Tityus* in South America. Numerous other genera may contain members of minor medical importance. Members of this scorpion family are commonly encountered by people and pets, making their status as venomous pests all the more important. The only buthid genus that naturally occurs in North America is *Centruroides*. Members of this genus are crevice dwellers that commonly enter homes. Five species occur in the United States. *Centruroides hentzi* is found throughout Florida and adjoining portions of Alabama and Georgia. *Centruroides guanensis* is common on the islands of the Bahamas and Cuba but is also found in the southernmost part of the Florida Peninsula and the Florida Keys. *Centruroides gracilis* is native to Central America and Mexico but has been introduced to many other tropical areas including Florida. *Centruroides vittatus* (Fig. 23.1) is the most widespread species of scorpion in the United States, with a range that extends from the Rio Grande River in the west to the Mississippi River in the east. This species has been collected as far north as southernmost Nebraska. It is found throughout Texas and Oklahoma and in adjoining parts of New Mexico, Colorado, Kansas, Illinois, Missouri, Arkansas, and Louisiana. *Centruroides sculpturatus* is found in most of Arizona, as well as adjoining parts of New Mexico, Utah, Nevada, and California. All species of *Centruroides* can deliver extremely painful stings that can be accompanied by systemic symptoms. However, only *C. sculpturatus* is considered dangerous, but only to small children and the elderly. The only other buthid occurring in the United States is the cosmotropical species *Isometrus maculatus*, an Asian species that has been introduced to tropical port cities around the world. This species is common on the island of Oahu in Hawaii.

TABLE 23.1 Dangerous Species of Scorpions Based on the Toxicity of Their Venoms

Species	Lethal Dose (LD_{50})	Geographic Occurrence
Leiurus quinquestriatus	0.25	Turkey, Israel, Egypt, Algeria, Libya, Sudan
Androctonus mauretanicus	0.31	Morocco
Androctonus australis	0.32	Morocco, Algeria, Libya, Tunisia, Egypt
Androctonus crassicauda	0.40	Turkey, Israel, Iraq, Arabian Peninsula
Tityus serrulatus	0.43	Brazil
Centruroides limpidus	0.69	Mexico
Androctonus amoreuxi	0.75	Middle East
Buthus occitanus	0.90	Morocco, Algeria, Jordan, southern Europe
Centruroides sculpuratus	1.12	United States, northern Mexico
Parabuthus transvaalicus	4.25	Southern Africa

The lethal dose is expressed as mg/kg of venom required to kill 50% of mice (LD_{50}) following subcutaneous injection. The lower the LD_{50}, the more potent is the venom. All of the scorpions listed are members of the family Buthidae. (Compiled from multiple sources.)

FIGURE 23.1 *Centruroides vittatus* (Buthidae). A common scorpion in the south central United States and northern Mexico. *Photograph by W. David Sissom.*

Pseudochactidae

This recently described family contains one genus with two species from Uzbeckistan, Tajikstan, and Afghanistan and two genera with four species from Laos and Vietnam. Members exhibit an unusual trichobothrial pattern similar to that of the Buthidae.

Chaerilidae

This family is represented by the single genus, *Chaerilus*, with 42 described species, none of which are dangerously venomous. These scorpions are unique in many ways, but they share some characteristics with the Buthidae. They occur in the Old World in India, Sri Lanka, Nepal, Bangladesh, Myanmar, Malaysia, Singapore, and many islands of the Philippines and Indonesia.

Chactidae

This family contains 14 genera and approximately 199 species. None are known to be dangerous. Most of the chactid species are found in South America (Colombia, Venezuela, Trinidad and Tobago, Guyana, French Guiana, Suriname, Ecuador, Peru, and Brazil). A few species are found as far north as Panama and Costa Rica. One peculiar species, *Nullibrotheas alleni*, is endemic to Baja California Sur (Mexico). *Anuroctonus*, a genus of enigmatic phylogenetic position from California, Nevada, and Utah in the United States and Baja California del Norte in Mexico, is considered by some researchers to be a chactid. *Anuroctonus* constructs permanent burrows in a variety of habitats throughout its range and is associated with canyons, ravines, and hillsides.

Euscorpiidae

This small family shares many characteristics with the Chactidae, of which it was once considered a subfamily. Recent work on this family has resulted in a large number of new species. Additionally, the members of the family Scorpiopidae were transferred to this family (Soleglad and Sissom, 2001), along with the genus *Chactopsis*, which was formerly placed in the family Chactidae. There are currently 12 genera and 137 species. One genus, *Euscorpius*, with 59 species, is found throughout southern (Mediterranean) Europe and has been variously divided into four subgenera and numerous subspecies. Seven genera of the former Scorpiopidae (now Scorpiopinae) may be found at relatively high altitudes throughout their range. They are native to parts of Afghanistan, Pakistan, India, Sikkim, Nepal, China, Bangladesh, Myanmar, Thailand, Laos, Vietnam, Malaysia, Indonesia, and Bhutan. None of them are regarded as dangerous. *Chactopsis* is native to South America (Peru, Venezuela, and Brazil). The other genera in this family are found in eastern Mexico and Guatemala. *Megacormus* and *Plesiochactas* are closely related ground-dwelling (epigean) forms. The two species of *Troglocormus* (Scorpiopinae) are troglobitic, having lost the median eyes, and are found only in caves.

Superstitioniidae

This monotypic family was previously regarded as a subfamily in the Chactidae. It shares few characters with the Chactidae but instead is closely related to the Typhlochactidae. Superstitioniidae contains the single species of the genus *Superstitionia*. *Superstitionia donensis* is found throughout much of the southwestern United States, including Arizona, Nevada, California, and southwestern New Mexico, as well as adjacent parts of northern Mexico. It is a small, uncommon scorpion and is not known to be medically important.

Typhlochactidae

The Typhlochactidae includes four genera and 11 species known mostly from caves in Mexico; two species are associated with leaf-litter habitats. All species lack both median and lateral eyes and have reduced or no pigmentation; some have attenuated appendages. They are generally small in size. At 9 mm total length, *Typhlochactas mitchelli* is one of the smallest species of scorpion in the world. The one exception in the family is *Alacran tartarus*, which measures up to 70 mm in length.

Troglotayosicidae

The two genera in this family were formerly placed in the Superstitioniidae. Both are poorly known, and their relationships remain uncertain. These species burrow underground (endogean) or live just beneath the ground surface (hypogean), and they possess lateral eyes but no median eyes. The four species of *Troglotayosicus* are known from Ecuador and Colombia. *Belisarius xambeui*, from caves in the eastern Pyrenees of Spain and France, was traditionally grouped with *Euscorpius*, with which it bears a superficial resemblance; however, this species, and a second one recently described from southern Spain, share a number of significant features with *Troglotayosicus*.

Akravidae

This family is based on a single troglobitic species described in 2007. The family is of dubious validity, and

phenetic comparisons place it near the Superstitioniidae and Typhlochactidae (Soleglad and Fet, 2011).

Iuridae

This interesting but small group of scorpions is restricted to south central Eurasia. None of them are considered dangerous. There are four genera and approximately 14 species. The genera *Iurus*, *Calchas*, *Neocalchas*, and *Protoiurus* are closely related, relatively large, and found in Turkey, Greece (including Samos, Crete, and other islands), Iraq, and possibly Syria.

Caraboctonidae

The Caraboctonidae is a New World family with four genera and 32 species. The genera *Caraboctonus* (one species) and *Hadruroides* (22 species) are found in western South America. *Caraboctonus* is found in Chile and Peru; *Hadruroides* is distributed through Ecuador (including the Galapagos Islands), Bolivia, Peru, and Chile. There are seven species in the genus *Hadrurus* that occur in desert areas of Oregon, Idaho, California, Nevada, Utah, Colorado, and Arizona (USA), as well as Sonora, Baja California Norte, and Baja California Sur (Mexico). Additionally, two species from south central Mexico formally contained in *Hadrurus* were recently placed in their own genus, *Hoffmannihadrurus* (Fet et al., 2004).

Vaejovidae

This family is composed of 26 described genera and approximately 214 species that are restricted mostly to North America. Species of Vaejovidae are found in every conceivable habitat in nearly every state of Mexico and much of the United States, especially the west (Washington, Oregon, Idaho, Montana, Wyoming, California, Nevada, Utah, Colorado, North Dakota, South Dakota, Nebraska, Arizona, New Mexico, and Texas). One species ranges south into Guatemala, while another species can be found in Canada. *Vaejovis carolinianus* occurs in wooded, mountainous areas of the eastern United States (parts of Kentucky, Tennessee, Virginia, North Carolina, South Carolina, Georgia, Alabama, Mississippi, and Louisiana). It is a small (about 2.4 cm), dark scorpion that readily enters homes throughout its range. Other species also may occur indoors, such as members of the genera *Pseudouroctonus* and *Uroctonus* that commonly enter homes in California. No members of this family pose any appreciable health threat, but stings from these species are more likely to cause minor localized discoloration, swelling, and necrosis than are the more painful stings of buthid scorpions.

Bothriuridae

This family of 17 genera and 154 species exhibits a Gondwanan distribution. One genus, *Cercophonius*, with seven described species, is distributed throughout Australia but is also found on New Caledonia and in northern India; the latter species is considered by most researchers to be of dubious validity. Two genera, *Lisposoma,* with two species, and *Brandbergia,* with one species, are endemic to Namibia. The remaining genera are distributed throughout western and southern South America (Ecuador, Peru, Brazil, Bolivia, Paraguay, Chile, Argentina, Uruguay). None of the bothriurids are considered medically important.

Hormuridae

Formerly known as Ischnuridae and Liochelidae, or considered a subfamily of the Scorpionidae, the hormurids are now regarded as a separate family. They range in size from small to very large and typically have a flattened body shape. The claws are massive in comparison to the body, but the metasoma is unusually thin and feeble-looking. Sometimes the metasoma is so short that it cannot reach to the front of the animal. Though capable of burrowing, most of these species are associated with crevice habitats in rocky areas, on trees, under debris, on man-made structures such as stone walls and wooden bridges, etc. The most impressive of the family is *Hadogenes troglodytes* from South Africa, the males of which can attain a body length up to 21 cm.

The family contains 11 genera and 88 species. Representatives are widely distributed throughout the tropics. In the Caribbean they are found in Haiti and Dominican Republic; in Central America: Panama and Cocos Island (Costa Rica); in South America: Peru, Colombia, Venezuela, French Guiana, Brazil; in central and southern Africa: Cameroon, Democratic Republic of Congo, Gabon, Congo, Malawi, Uganda, Ethiopia, Kenya, Tanzania, Angola, Namibia, Botswana, Zimbabwe, Mozambique, South Africa, Lesotho, Swaziland, Mauritius, Round Island, Seychelles, Zanzibar, Madagascar; in Asia: China, Korea, Japan, India, Aru Islands, Bangladesh, Myanmar, Thailand, Cambodia, Laos, Vietnam, Malaysia, Indonesia, Philippines, Papua New Guinea; in Oceania: Federated States of Micronesia, Fiji, French Polynesia, Key Islands, Kiribati, Mariana Islands, Marshal Islands, New Caledonia, Palau, Ponape, Tuvalu, Samoa, Solomon Islands, Tonga, Vanuatu; and, of course, Australia. As a group, they are considered relatively harmless. In the case of stings by the giant *Hadogenes* scorpions, the effect may be so slight as to be barely felt. Other species, such as *Opisthacanthus lepturus* in Panama, can deliver a sting that causes soreness in joints, as well as mild, localized discoloration, swelling, and necrosis.

Heteroscorpionidae

This is a tiny family with one genus (*Heteroscorpion*) and six species that are endemic to Madagascar. This genus was formerly placed among the Ischnuridae, with which they share many characteristics. Their medical importance is not known.

Hemiscorpiidae

Members of this family superficially resemble members of the Hormuridae, being flattened and possessing a thin, delicate postabdomen. They are crevice dwellers like many of the hormurids. In the past, they have been variously placed in the Hormuridae or the Scorpionidae. There is a total of 16 species in this group. All are considered to belong to a single genus, *Hemiscorpius*, although some authors still recognize the validity of the monotypic genus *Habibiella*. These species are found in Somalia, Eritrea, Saudi Arabia, Yemen, Oman, United Arab Emirates, Iraq, Iran, and Pakistan. *Hemiscorpius lepturus* in Iran is the only nonbuthid species of scorpion reported to cause significant mortality in humans. The venom contains a potent cytotoxin that causes severe tissue damage and necrosis near the sting site, as well as severe systemic symptoms. The medical importance of the other species of *Hemiscorpius* is not known; however, it is safe to assume that they have venom that, at the very least, causes soreness in joints, as well as mild, localized discoloration, swelling, and necrosis, as in their close relatives, the Hormuridae and Scorpionidae.

Urodacidae

This family, often considered a subfamily of Scorpionidae, contains two genera, *Urodacus* and *Aops*, which are endemic to Australia. There are 21 described species of *Urodacus* and a single species of *Aops*. *Aops oncodactylus* is troglobitic. None are known to be dangerous.

Diplocentridae

This is another comparatively small family with 10 genera and 130 described species that occur primarily in the New World. Exceptions are the genera *Nebo,* found in Syria, Jordan, Lebanon, Israel, Egypt (Sinai), Saudi Arabia, Yemen, and Oman, and two species of *Heteronebo*, both known from the island of Abd-el-Kuri (Yemen). Oddly, the other 17 species of *Heteronebo* are found on various islands in the Caribbean, along with the genera *Oiclus*, *Cazierius*, *Cryptoiclus*, and most of the species in the genus *Didymocentrus*. The latter genus is also represented in Honduras, El Salvador, Nicaragua, and Costa Rica. Another genus, *Tarsoporosus*, is found in Venezuela and Colombia. The genus *Bioculus* is endemic to Baja California Sur (Mexico) and its associated islands. Widespread through Honduras, Guatemala, Belize, and Mexico, *Diplocentrus*, with 64 described species, is the largest genus in the family (Fig. 23.2). Five species occur in the southern parts of Arizona, New Mexico, and Texas (USA). Two species of *Kolotl* occur in southern Mexico. Diplocentrids are not generally considered dangerous. However, stings from the Middle Eastern species *Nebo hierichonticus* may cause mild, local hemorrhages and slight necrosis.

FIGURE 23.2 *Diplocentrus diablo* (Diplocentridae). Lower Rio Grande Valley in Texas (USA) and adjacent Mexico. *Photograph by W. David Sissom.*

Scorpionidae

As the oldest recognized scorpion family, this group once included all known scorpions. Over the years, its scope has been reduced. Currently, the family is compact and homogeneous, with nine genera and 157 species, and includes some of the world's largest and most formidable-looking scorpions. All are heavy-bodied with large, powerful pedipalps. Some members of the Asian genus *Heterometrus* reach lengths of 16 cm or more. *Pandinus imperator* from West Africa is often cited as one of the largest scorpions, occasionally attaining a body length of 18 cm and weighing up to 32 g as nongravid females (Fig. 23.3). This large, black scorpion is commonly sold in pet stores. The 17 species of *Scorpio* are distributed across northern Africa (Senegal, Mauritania, Morocco, Algeria, Tunisia, Libya, Egypt) and the Middle East (Turkey, Lebanon, Syria, Iraq, Kuwait, Iran, Israel, Jordan, Saudi Arabia, Qatar, Yemen). The former genus *Pandinus*, containing the large African emperor scorpions, is now subdivided into four genera: *Pandinus, Pandinurus,*

FIGURE 23.3 Emperor scorpion, *Pandinus imperator* (Scorpionidae). A relatively harmless West African scorpion, despite its large size and intimidating appearance; commonly sold as pets. *Photograph by João O, Burini.*

Pandinopsis, and *Pandinoides*. These genera range across central Africa, (Senegal, Gambia, Guinea-Bissau, Guinea, Ivory Coast, Burkina Faso, Ghana, Togo, Nigeria, Cameroon, Equatorial Guinea, Gabon, Congo, Democratic Republic of Congo, Sudan, Eritrea, Ethiopia, Somalia, Kenya, Tanzania, Malawi, Zimbabwe, Mozambique). One species of *Pandinurus* is also represented on the nearby coasts of Saudi Arabia and Yemen. *Opistophthalmus*, with its 59 species, is found throughout southern Africa (Tanzania, Angola, Zambia, Zimbabwe, Mozambique, Namibia, Botswana, Lesotho, South Africa). The genus *Heterometrus* (37 species) is distributed through India, Sri Lanka, Bangladesh, Myanmar, Thailand, Cambodia, Laos, Vietnam, Malaysia, Singapore, Indonesia, Brunei, and the Philippines. Though normally not considered dangerous, most species can deliver stings that can cause mild to severe localized discoloration, swelling, and tissue damage. The stings of many species of *Heterometrus* can cause serious localized hemorrhaging and blistering.

MORPHOLOGY

The scorpion body (Fig. 23.4) is divided into two major parts: the **prosoma** (cephalothorax) and the **opisthosoma** (abdomen). The opisthosoma is segmented and further divided into the **mesosoma** (preabdomen) and the more slender, tail-like **metasoma** (postabdomen). The metasoma

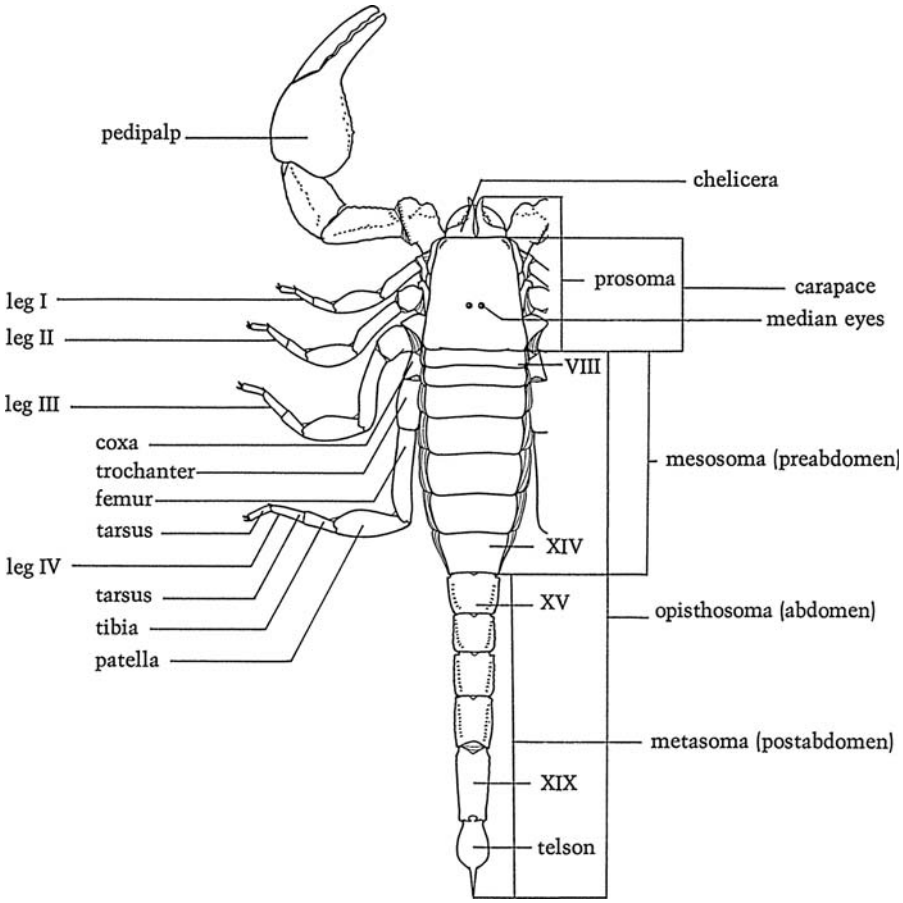


FIGURE 23.4 Scorpion morphology, adult, dorsal view. *Keegan (1980), with permission of University Press of Mississippi.*

bears at its posterior end a stinging structure called the **telson**.

The dorsal aspect of the prosoma consists of a single sclerotized plate referred to as the **carapace**. It is marked by various furrows, grooves, depressions, and keels that indicate internal apodemes and other surfaces for the attachment of muscles associated with the legs and other appendages. A pair of **median eyes** is located on an **ocular tubercle** along the midline of the carapace. Two additional groups of smaller **lateral eyes** are situated at the anterolateral margins of the carapace. There may be as many as five pairs of lateral eyes, whereas eyes may be lacking altogether in some cave-dwelling species. The ventral aspect of the prosoma consists of a posteromedian **sternum** and the broad coxae of the legs. The sternum is basically pentagonal in shape but may appear to be more triangular in some taxa.

The mesosoma is divided dorsally into seven apparent segments, each bearing a **tergite**. Ventrally there are five **sternites**, the first four of which bear a pair of **spiracles** (Fig. 23.5). The **genital aperture** is located anteriorly between the coxae of the fourth pair of legs and is covered by a pair of small plates called the **genital opercula**. The genital opercula are commonly fused in females but not in males. Just behind the sternum is a pair of appendages unique to scorpions called **pectines** (sing., *pecten*) (Fig. 23.6). These structures function primarily as chemoreceptors that enable males to locate females by tracking

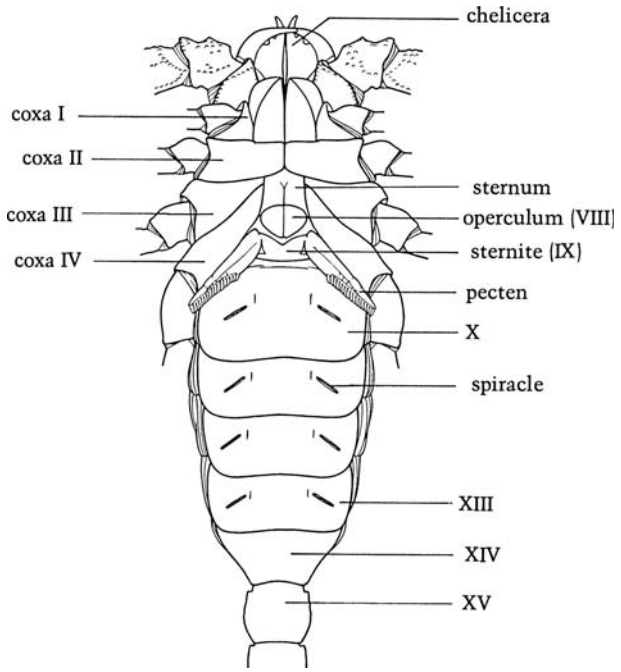


FIGURE 23.5 Scorpion morphology, adult, ventral view; with legs beyond coxae and most of metasoma (postabdomen) not shown. *Keegan (1980), with permission of University Press of Mississippi.*

her pheromones. They also serve as mechanoreceptors that can sense the nature of the substrate and apparently aid in detecting substrate vibrations. Although quite variable when all scorpion genera are considered, the **pecten** typically consists of three anterior marginal lamellae, the middle lamellae, a row of triangular fulcra, and a posterior series of fleshy lamellae called **pectinal teeth** (Fig. 23.6). The ventral surface of each pectinal tooth is covered with mechanoreceptors in the form of tiny sensory pegs visible only at high magnification. Up to 1,200 sensory pegs per pectinal tooth have been reported in *Leiurus quinquestriatus*.

The metasoma or "tail" is divided into five segments, plus the telson (Fig. 23.7). The segments are well sclerotized and may bear longitudinal ridges or **keels** along their dorsal, lateral and ventral surfaces. The nature and location of these keels can serve as important taxonomic characters. The **telson** consists of a bulbous base, called the **vesicle** or **ampulla**, and a curved, sharply pointed terminal spine, the **aculeus**. Just below the aculeus the telson also may bear a small, median **subaculear tubercle** or **accessory spine** (Fig. 23.7D). The vesicle contains a pair of **venom glands** and associated musculature. The venom glands may be simple and saclike or more complexly folded with pouch-like extensions that greatly increase the surface area of the secretory epithelium. The venom is discharged by contraction of the muscles surrounding the glands, which compress the glands against the vesicle wall. The venom is forced out through the pair of **venom ducts** that open near the tip of the aculeus.

The prosomal appendages of scorpions are a pair of chelicerae (Fig. 23.8B), a pair of pincer-like pedipalps (Fig. 23.8A), and four pairs of walking legs. Each **chelicera** consists of three segments. The terminal, third segment serves as a movable finger that opposes the second

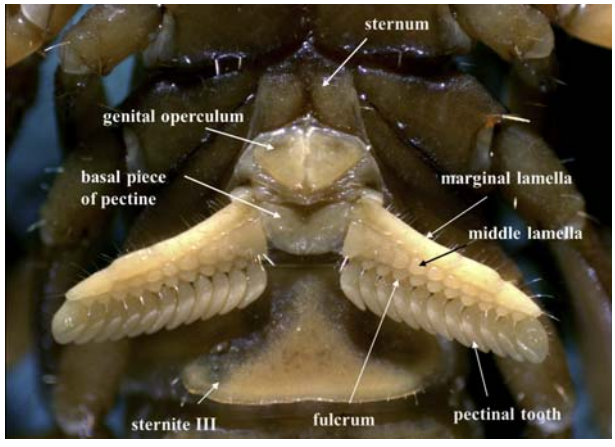


FIGURE 23.6 Sternum, genital opercula, and pectines of *Pseudouroctonus apacheanus* (Vaejovidae), Arizona (USA); located on venter of scorpion at level of third and fourth pairs of legs. *Photograph by W. David Sissom.*

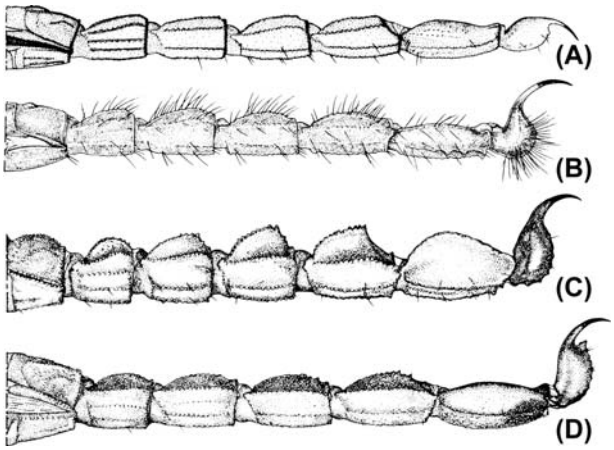


FIGURE 23.7 Metasoma (postabdomen, including terminal telson) of scorpions, showing various morphological features. (A) *Centruroides vittatus* (Buthidae), with relatively slender segments and inconspicuous keels. (B) *Leiurus quinquestriatus* (Buthidae), with slender segments and numerous, long sensory hairs. (C) *Androctonus australis* (Buthidae), with enlarged, robust segments and prominent keels. (D) *Tityus serrulatus* (Buthidae), with well-developed keels and distinct subaculear tubercle on telson. *Adapted from Keegan (1980), with permission of University Press of Mississippi.*

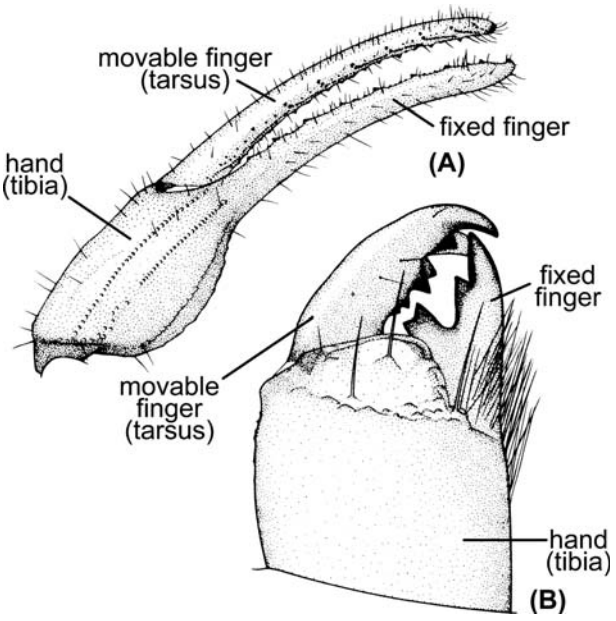


FIGURE 23.8 *Centruroides vittatus* (Buthidae). (A) Chelate (pincer-like) tibia and tarsus of pedipalp, used for seizing prey. (B) Chelicera, showing the movable tarsus and fixed finger of the tibia, used to crush and tear prey. *Adapted from Keegan (1980), with permission of University Press of Mississippi.*

segment, or hand (*manus*), bearing an anterior apophysis called the fixed finger. Both fingers are armed with teeth that facilitate the grasping and tearing of food. The **pedipalps** are six-segmented, each consisting of a coxa, trochanter, femur, patella (genu), tibia, and tarsus. The distal-most segment (tarsus) forms a movable finger, which opposes the fixed finger of the tibia, or hand. The various numbers and arrangements of keels, tubercles, denticles, granules, and **trichobothria** (sensory hairs) on the hand, patella, and femur of the pedipalp are important taxonomically. The walking legs consist of the same six segments as the pedipalps, plus a seventh terminal segment, the **pretarsus**, which bears a pair of lateral claws and a single, small median claw. The tarsus is divided into two tarsomeres. The presence and number of tibial spurs and pedal spurs on the legs can be helpful in identifying certain groups of scorpions.

Although scorpions are sexually dimorphic, it generally takes a specialist to reliably distinguish males and females. There are no uniform gender-specific, external morphological characters for determining the sex that apply to all scorpions. In general, however, males are smaller and less robust than females of the same species. Males tend to be relatively more slender than females, but there are many exceptions. In some genera, the pedipalp chelae of males are longer and more slender, whereas in other genera they are shorter and thicker. The pedipalp chela fingers of males of some species have special depressions for accepting the female's pedipalp fingers during mating. Usually, the metasoma of males is relatively longer and more slender than that of females; however, it is sometimes shorter and thicker, being different with each species. The pectines are often strikingly different, with the males having larger pectines with more pectinal teeth. In some cases the males can be recognized by the presence of a pair of genital papillae protruding posteriorly from beneath the genital operculum.

LIFE HISTORY

Although scorpions as a group differ in details relating to their biology and behavior, they follow a similar life-history pattern. Females are viviparous, giving birth to young following a developmental period that varies from 2 to 18 months, depending on the species. Scorpions are unusual among arachnids in this respect, the only other known case being one family of mites, the Pyemotidae. The average brood size for all scorpions is about 26, but it can range from as few as one up to 105. The sex ratio at birth is about equal, even though this ratio later shifts significantly toward females after reaching maturity. Parthenogenesis has been reported in eight species of scorpions, including five species of the genus *Tityus* from South America, *Hottentotta hottentotta* from West Africa, *Ananteris coineaui* from French Guiana, and *Liocheles australasiae* from islands in the South Pacific. Sexual populations are known from some of these species. All but one of the parthenogenetic species produce only female

offspring; the exception is based on a female of *Tityus metuendus* that gave birth to three consecutive all-male litters in captivity.

Following their birth, the newborns immediately crawl onto their mother's back, where they remain through the first instar without feeding. If removed during this period, they die without successfully molting. A major contributing factor seems to be their dependence on obtaining water from their mother's cuticle; they are prone to desiccation due to the permeability of their cuticle at this critical time. In most cases, dispersal of the young occurs shortly after they molt to the second instar, usually within 3–14 days after birth. In other cases, the young may remain in the burrow with their mother, where she cares for them and may even feed them captured prey. This type of social behavior appears to be common in the scorpionoids and has been described in other taxa (e.g., *Euscorpius*).

Most species molt five or six times before becoming adults, although the number of molts varies from four to nine depending on the species. Males generally undergo fewer molts than females of the same species. Maturity is reached in as few as 6 months in some of the smaller species (e.g., *Centruroides* spp.) but may take as many as 3–7 years in some of the largest species (e.g., *Pandinus* spp.). Buthid scorpions develop the fastest, averaging about 18 months, whereas the mean developmental time for all other scorpions is about 3 years. The longevity of most scorpions is probably about 3–7 years. It seems likely that scorpions in temperate environments may take longer to mature (one molt per year) and thus live longer overall due to short growing seasons. Scorpions may live still longer in captivity. The longest reliable report is 8 years for *Pandinus gambiensis*.

BEHAVIOR AND ECOLOGY

Scorpions are well adapted for surviving in a wide range of habitats, including deserts, grasslands, savannas, and both temperate and tropical forests. In addition, they are found from intertidal zones at sea level to snow-covered mountains at elevations of over 5,500 m, and in cave systems at depths of more than 800 m. They can tolerate highly varied environmental conditions, including extremes of temperature, both heat and cold, complete immersion in water for hours, and prolonged periods of drought and starvation. In large part, these adaptations are due to behavioral thermoregulation, low metabolic rates, and high efficiency in conserving water. To moderate their body temperatures, scorpions are typically nocturnal, retreating to the protection of burrows and other sheltered sites during the daytime. They experience minimal water loss via their cuticle, spiracles and book-lungs, while excreting nitrogenous wastes in almost insoluble forms such as guanine, xanthine, and uric acid. Similarly, their feces are extremely dry.

Most scorpions live on or very near the ground, where they typically are found under objects, in forest litter, or excavated burrows. The major exception is the large and important family Buthidae in which the species are often excellent climbers. They commonly are found under the bark of trees, in the tops of palms and other plants, and crevices of rocky cliffs. Upon entering houses, these species are likely to be seen on the walls and even ceilings, not infrequently gaining access to the upper floors of multistory buildings. Such climbers include some of the most venomous scorpions, notably, members of the genus *Centruroides*. Even some of the common vaejovid scorpions, such as *Vaejovis carolinianus* in the southeastern United States, are excellent climbers. They frequently enter homes where they may be seen on walls or clinging upside-down on ceilings.

Scorpions feed on a variety of prey, notably, soft-bodied insects and arachnids. Heavily sclerotized insects and other invertebrates such as certain isopods are often rejected. Common prey items include spiders, solpugids, other scorpions, millipedes, centipedes, gastropods, and other invertebrates. The larger scorpions also will attack and feed on small vertebrates such as lizards, snakes, and rodents. Owing to poor vision, scorpions depend primarily on their sensory hairs and their ability to sense ground vibrations as a means of detecting, locating, and recognizing suitable prey. Using mechanoreceptors on their tarsi, they can detect potential prey up to 15 cm away. Some arboreal scorpions even can capture flying insects that approach close enough for them to detect via air movements with the trichobothria on their pedipalps.

Scorpions with large, robust pedipalps can often subdue their prey with little or no use of their venom. Smaller species with weaker, more slender pedipalps are more dependent on stinging their prey upon seizing it with their chelate pincers. The thrust with their stinger is usually carefully delivered to penetrate the softer areas of the prey's integument between sclerites or other hard body parts. After locating a suitable site, sufficient venom is injected to immobilize the prey, following which the sting is withdrawn. This is in strong contrast to the defensive strikes directed toward threatening enemies in which the telson lashes forward to sting its target, inject the venom, and be quickly withdrawn.

Once a scorpion has captured a prey item, it crushes it with the coxal bases of its pedipalps and the first two pairs of legs while tearing at it with its chelicerae. Digestive juices from specialized glands in the gut flow through a preoral cavity formed by the coxae of the pedipalps and anterior legs. Following extraoral digestion, the semidigested food is drawn into the mouth assisted by the chelicerae; undigested parts are trapped by setae in

the preoral cavity and expelled. The feeding process is slow, taking as long as 2.5 h to consume an item such as a blow fly. Owing to the efficiency in storing digested food in the hepatopancreas and a low metabolic rate, a well-fed scorpion can survive for months without further feeding.

When it comes time to seek a mate, the female recognizes conspecific males by a behavior called **juddering**, in which the male displays a series of shaking movements, rocking back and forth with his pectines spread out and quivering. The resulting vibrations are communicated via the substrate to the female. There is also evidence to suggest that pheromones may be involved in sex recognition in at least some species. Having located one another, the male initiates courtship by grasping the female's pedipalps with his own and guiding her through a complex courtship behavior in the form of a mating dance, or **promenade**. During the promenade they may engage in cheliceral massages or kissing, in which the male grasps the female's chelicerae with his chelicerae and gently kneads them (Fig. 23.9), apparently serving to suppress her aggressiveness. In many species the male actually stings the female, usually in the tibial joint of the pedipalp, where the inserted sting may be held for 3−20 min or longer. This appears to reduce her aggression and render her more docile during the courtship.

Throughout the promenade the male uses his pectines to sweep back and forth across the ground, sensing the substrate to determine if it is suitable for depositing a spermatophore. Having found an acceptable site, he extrudes a complexly structured spermatophore from his genital aperture and attaches it to the substrate in an upright position with the sticky basal plate firmly anchoring it in place. He then guides the female over the spermatophore to make contact with her genital valves. As the spermatophore bends under pressure from her mesosoma, the sperm is released directly into her genital tract. Contact with the spermatophore may last anywhere from a few seconds to several minutes. Once insemination is completed, the male abruptly disengages from the female and effects an immediate escape, lest he be attacked and eaten by his no longer receptive mate.

When the gravid female is ready to give birth, she assumes a position known as **stilting**, in which she raises the anterior part of her body and forms a **birth basket** with her pedipalps and first two pairs of legs. While she maintains this posture, the young emerge one at a time from the genital aperture and drop into the birth basket. From there they clamber onto their mother's back (Fig. 23.10). The birth time for an individual varies from 1 min up to an about an hour, with the total birth process lasting from less than 12 h to as long as 3 days.

More than 150 species of predators have been reported to feed on scorpions. Among the most common are birds and lizards, followed by various mammals, frogs, toads, and snakes. Several invertebrates also are natural enemies, including spiders, solpugids, ants, centipedes, and other scorpions. In many cases scorpions are resistant to their own venoms; however, they readily fall prey to attacks by other species of scorpions, including situations involving cannibalism and predation. Scorpions comprise a significant part of the diet of burrowing owls, elf owls, and grasshopper mice. They also are hosts for mermithid nematodes and the ectoparasitic larvae of certain mites.

PUBLIC HEALTH IMPORTANCE

Scorpion sting cases can be categorized as two general types: those involving only localized, transitory symptoms usually lasting from a few minutes to several hours and those involving systemic reactions. Localized responses are characterized by immediate pain followed by moderate

FIGURE 23.9 *Paruroctonus luteolus* (Vaejovidae) during courtship and mating; the male grasps the female's chelicerae with his own chelicerae and gently kneads them to suppress her aggressiveness. *Photograph by W. David Sissom.*

FIGURE 23.10 *Centruroides vittatus* (Buthidae). Female carrying first-instar young on her back. *Photograph by W. David Sissom.*

swelling at the sting site, often likened to the sting of a wasp or bee. In some cases, the sting may result in a raised, reddened, indurated lesion, even in the case of relatively harmless scorpions (e.g., *Vaejovis carolinianus*). In cases involving cytolytic toxins (e.g., scorpionids and hormurids), swelling may persist up to 72 h, followed by development of hemorrhages and blood-filled blisters near the sting site. Sloughing of skin may occur, but this varies greatly in severity. Other localized effects include goose flesh, sweating, and muscle spasms near the sting site. In cases of buthid stings, pain usually radiates from the site of the sting up the affected limb. The pain tends to concentrate in the joints, especially the armpits and groin, and often crosses from one armpit to the other.

In cases of **systemic reactions**, the clinical signs and symptoms are highly variable, ranging from mild to life-threatening. Systemic reactions commonly are mild and are not necessarily indicators of a serious problem. Often there is no appreciable swelling or discoloration of the skin at the sting site. An intense aching and burning sensation may spread to adjacent tissues, which in turn often throb, sometimes becoming numb. The acute pain at the sting site turns into a chronic, dull pain accompanied by a feeling of numbness around the edge of the sting site, which may persist for one to several days. Numbness in the face, mouth, and throat is fairly common. Muscles may become spasmodic, resulting in muscular twitching, slurred speech, difficulty swallowing, tightness or cramps in the chest and back, rapid heartbeat, and nausea. Often these systemic responses persist less than an hour after the sting and are not considered serious.

In more severe systemic reactions, neurologic effects can lead to profuse sweating and salivation, restlessness, extreme nervousness, respiratory and cardiovascular problems, mental confusion, and convulsions. As the clinical symptoms indicate, the principal components of the venom of dangerous scorpions are neurotoxins. These toxins act on the autonomic, sympathetic, and neuromuscular systems, causing the wide range of systemic reactions reported in sting victims. They act by disrupting the voltage-sensitive sodium and potassium channels of nerves, which in turn causes neural depolarization, prolonged action potentials, repetitive firing, and uncontrolled release of vasodilators and neurotransmitters, which affect virtually every major organ system. The effect on neurotransmitters results in depletive release of catecholamines (e.g., adrenaline, noradrenaline) that can severely damage the heart and other organs.

The most commonly reported cause of death in scorpion sting cases is cardiac failure. In other cases, respiratory failure may be the cause, especially in patients with upper respiratory infections or related problems. Death usually occurs several days after envenomation. If symptoms subside during the first 2−12 h following a sting, the prognosis for recovery is generally good. Mortality rates are quite variable, depending on the species and amount of venom injected. The rates are much higher among children than adults. For further details on the clinical toxicology and symptoms of scorpion stings, see Dehesa-Davila et al. (1995) and Ismail (1995).

Scorpion **venom** is a very complex mixture of substances that differs significantly among the various taxa, within families, and among genera. Differences also occur in different geographic populations of the same species and even within the same population. The toxins are low-molecular-weight proteins that are among the most powerful toxins known. They are comparable in some species to the neurotoxins of certain deadly snakes. Two recognized types of neurotoxins are **α-scorpion toxin**, characteristic of the genera *Androctonus*, *Leiurus*, and *Buthus*; and **ß-scorpion toxin**, characteristic of *Centruroides*. *Tityus* spp. appear to have both types. The chemical structures of two neurotoxins of *Leiurus quinquestriatus*, **agitoxin** and **scyllatoxin**, are shown in Figure 3.7. The effects of envenomation by any given scorpion species can differ significantly, owing to a wide range of contributing factors. These include the quantity of venom injected and the age, size, and general health of the victim.

The sting of most scorpions usually requires no special treatment, although the application of ice to the sting site helps to relieve local pain. Incisions, as used in cases of poisonous snake bites, should never be made. Nor is the use of most drugs recommended in uncomplicated cases because antihistamines, steroids, analgesics, and sedatives usually have little or no effect. In more severe cases involving systemic reactions, medical attention should be sought immediately.

Substances that have been found to be effective in treating scorpion stings are atropine to counter effects on the parasympathetic system, calcium gluconate given intravenously to relieve muscle spasms, and sodium phenobarbital administered intravenously to prevent convulsions. Insulin also has been reported to be beneficial in treating cases in India. Morphine and Demerol generally are not recommended as pain relievers because of their tendency to act as synergists, increasing the toxicity of certain venoms (e.g., *Centruroides sculpturatus*).

Antivenins are recommended where available. However, caution in the use of antivenins must be noted since many antivenins are of poor quality and often are administered at doses far below the level required to produce effective results. They may adequately neutralize the larger venom peptides but not necessarily the more important small-molecular-weight toxins. To be effective, they must be administered within the first hour following the sting. Even then, the cessation of systemic symptoms within about an hour may not necessarily reflect the use of the antivenin, since this is the same period of time that symptoms often subside without treatment of any kind. Another limitation is the fact that most scorpion antivenins are very specific, often for a single species, and are produced only on a limited regional basis (Theakston and Warrell, 1991; Lucas and Meier, 1995).

The development of scorpion-specific antivenin (Anascorp) derived from horse serum for *Centruroides* venom has been shown to be highly effective in treating cases of severe envenomation (Boyer et al., 2009). It has been used in treatment of stings from *C. limpidus, C. tecomanus, C. noxious,* and *C. suffusus* in Mexico and *C. sculpturatus* in Arizona. In a randomized, double-blind study involving 15 children (ages 6 months to 18 years) in Arizona, all eight children receiving Anascorp had resolution of the symptoms within 4 h, in contrast with only one in seven of the placebo group.

For further information on the nature of scorpion toxins and their potential medical applications for the treatment of various types of cancer and cell-mediated autoimmune diseases, see Chapter 3. *Arthropod Toxins and Venoms*.

SCORPIONS OF MEDICAL IMPORTANCE

Some of the more dangerous species of scorpions for which toxicity data have been reported are shown in Table 23.1. Based on mammalian toxicity, *Leiurus quinquestriatus*, *Androctonus australis*, and *A. mauretanicus* are generally recognized as the most venomous. *Leiurus quinquestriatus* (Fig. 23.11) is known in the Middle East and Sahel region of northern Africa as the **yellow scorpion**, in the Sudan as the **Omdurman scorpion**, and in the pet trade as the **deathstalker scorpion** The *Androctonus* spp. are commonly called **fattail scorpions**, referring to the marked thickness and width of their postabdominal segments. The fattail scorpion, *Androctonus australis* (Fig. 23.12), which occurs primarily in arid mountainous regions, causes more deaths than any other species in North Africa. Based on the numbers of cases and fatalities, it is probably the most deadly scorpion worldwide. The common yellow scorpion (*Buthus occitanus)*, a widely distributed species in the Middle East and North Africa, is the only medically important species in southern Europe. Its toxicity varies markedly in different parts of its range, apparently reflecting different subspecies. The **Indian red scorpion** (*Hottentotta tamulus*) is the most medically important scorpion on the Indian subcontinent.

The more important venomous genera in the New World are *Centruroides* and *Tityus*. *Centruroides* spp. occur primarily in Mexico, Central America, and the West Indies. They are often called **bark scorpions** due to their habit of hiding under loose tree bark or in crevices of dead logs and trees. They are commonly found around domestic settings in piles of wood, stones, bricks, and discarded debris. Stings are likely to occur when they are disturbed in their hiding places or when they enter homes at night in search of prey. The **Arizona bark scorpion**, *Centruroides sculpturatus* (Fig. 23.13), is often cited as the most dangerous scorpion in the United States. Deaths from its sting, however, are rare.

FIGURE 23.11 *Leiurus quinquestriatus* (Buthidae). A dangerous scorpion in North Africa and the Middle East. *Photograph by W. David Sissom.*

FIGURE 23.12 *Androctonus australis* (Buthidae). A highly toxic scorpion found in North Africa and the Middle East. *Photograph by W. David Sissom.*

FIGURE 23.13 *Centruroides sculpturatus* (Buthidae). The only scorpion of significant medical importance in the United States; southwestern states. *Photograph by W. David Sissom.*

A number of species of *Centruroides* are able to produce severe envenomation in Mexico, including *C. limpidus, C. elegans, C. balsasensis, C. tecomanus, C. infamatus, C. noxious, C. suffuses, C. sculpturatus,* and others. A recent study (Ponce et al., 2016), based on data for 1997−2013 from the Mexican Ministry of Health indicates a human mortality rate of about 1,100 deaths/year, with the highest numbers in seven states: Morelos, Pueblo, Mexico, Michoacan, Nayarit, Jalisco, and Guerrero. Some of these states are showing an increase in incidence of both morbidity and mortality, believed to be caused by reduction in pesticide use to control kissing bugs (vectors of the agent of Chagas disease) and mosquitoes, as well as human encroachment into more natural habitats occupied by scorpions.

Tityus spp. are similar to *Centruroides* spp in size, general appearance, and behavior. As a group of over 100 species, they occur throughout South America and the Caribbean Basin. In most places where they occur, all *Tityus* species are considered dangerous. The most venomous is the Brazilian species *T. serrulatus*, which is common in urban areas and readily enters homes. Second only to *T. serrulatus* in its medical importance is another house-infesting scorpion in Brazil, *T. bahiensis* (Fig. 23.14). Related *Tityus* spp. that also are highly venomous include *T. cambridgei*, a forest-dwelling species in the Amazon Basin and northern South America; *T. trinitatis*, which can be a serious problem in coconut groves and cane fields in Venezuela and Trinidad; and *T. trivittatus*, a house-infesting scorpion in Argentina.

The presence of dangerously venomous scorpion species in the pet trade (e.g., *Androctonus, Leiurus, Buthus, Centruroides,* and *Tityus*) introduces the potential for accidental envenomation.

FIGURE 23.14 *Tityus bahiensis* (Buthidae). A dangerous scorpion in Brazil that commonly enters homes. *Photograph by João O, Burini.*

VETERINARY IMPORTANCE

There is some evidence to indicate that scorpions dangerous to humans can also pose a significant threat to domestic animals, including cats and dogs. Although the site of a scorpion sting in a dog is sometimes difficult to locate, local pain is almost ubiquitous and in severe cases is characterized by intense, burning pain that may involve the whole limb. This pain persists for several hours, but hyperesthesia or paresthesia may continue for several days (Gomez-Ortiz et al., 1972). Dogs may also show systemic symptoms, such as agitation, shivering, sweating, erythema, drowsiness, and coma. Hemorrhagic pancreatitis is known to be associated with venoms of *Leiurus quinquestriatus* and *Tityus serrulatus* (Machado and Filho, 1976; Pantoja et al., 1983). A Brazilian terrier stung by *Tityus bahiensis* on a paw exhibited vocalization, drowsiness, pain, aggressiveness, tachypnea, and tachycardia, as well as slight erythema at the site of the sting. The animal fully recovered within 24 h of treatment with 10 mL of 2% lidocaine hydrochloride, without vasoconstrictor (Cardosa et al., 2004).

Cordeiro et al. (2006) examined the effects of two dosages of *Tityus serrulatus* venom in dogs and concluded that normal doses (0.4 mg/animal) produced pain, prostration, and excessive salivation, which lasted about an hour. However, when the dose was increased to a higher experimental level (0.25 mg/kg), dogs exhibited prolongation of these symptoms, as well as sneezing, vomiting, diarrhea, and, in some cases, piloerection and tremors; there was also an increase in leukocyte count and myocardium-specific creatine kinase MB levels, indicating cardiovascular effects. There was no mortality.

PREVENTION AND CONTROL

Pesticides are not generally recommended for controlling scorpions indoors or for preventing their entering homes. Instead, appropriate measures can be taken to scorpion-proof buildings or otherwise significantly reduce the prospects of them entering homes. Entry can be discouraged by raising the floor level at least 20 cm above the ground.

A single step to reach the threshold is better than multiple steps and should be separated from the wall of the structure by a gap of 6 cm or more. The installation of a horizontal row of glazed ceramic tiles on the vertical surfaces of steps and around the entire perimeter of a building also can provide a barrier that scorpions cannot readily climb. Smooth exterior wall surfaces, such as planed cement, further impede their climbing ability. Worn weather stripping around doors and windows should be replaced, and potential entry sites around water pipes and electrical conduits in foundations should be sealed. To prevent scorpions from gaining access to the roofs of structures, a row of ceramic tiles can be applied to the outer walls just below the roof line.

Scorpions can be discouraged from frequenting the immediate vicinity of homes by trimming plantings that touch buildings and removing piles of firewood, lumber, bricks, and other materials that serve as harborage. The use of coarse bark mulches around plants near the foundation of buildings should be avoided for the same reason.

In areas where climbing scorpions commonly infest homes, measures can be taken to reduce the risk of envenomation. A sheet of muslin or other suitable cloth can be suspended from the ceiling over the sleeping quarters to catch any scorpions that might drop from overhead structures. Mosquito netting over beds affords similar protection. Regularly shaking out clothing and footwear before putting them on is highly recommended.

In temperate regions, the greatest number of complaints of scorpions entering homes is often seasonal, most commonly in the early spring and late fall. Heavy or frequent rains in the spring can saturate the soil and ground litter around building foundations, driving scorpions indoors as they search for drier sites. With the onset of colder weather in the late fall, scorpions are similarly, apt to find their way indoors while seeking warmer temperatures. Another circumstance that contributes to scorpion problems is the construction of new homes or subdivisions in previously undisturbed habitats where scorpions are abundant. The clearing of such areas and the associated disturbance of ground litter often causes displaced scorpions to wander extensively. In the process they frequently find their way inside nearby homes. Sealing or blocking potential access sites is the only practical means of preventing their entry.

REFERENCES AND FURTHER READING

Anderson, R. C. (1975). *Scorpions of Idaho* (Vol. 18, pp. 1−17). Tebiwa.

Balozet, L. (1971). Scorpionism in the old world. In W. Bücherl, & E. E. Buckely (Eds.), *Venomous animals and their venoms* (Vol. 3, pp. 349−371). New York: Academic Press.

Bettini, S. (Ed.). (1978). *Arthropod venoms*. Berlin: Springer-Verlag, 977 p.

Boyer, L., Theodorou, A., Berg, R., Mallie, J., Chávez-Méndez, A., García-Ubbelohde, W., et al. (2009). Antivenom for critically ill children with neurotoxicity from scorpion stings. *The New England Journal of Medicine, 360*, 2090−2098.

Briggs, D. E. G. (1987). Palaeontology: Scorpions take to the water. *Nature, 326*, 645−646.

Bücherl, W. (1978). Systematics, distribution, biology, venomous apparatus, etc. of Tityinae; venom collection, toxicity, human accidents and treatment of stings. In S. Bettini (Ed.), *Arthropod venoms* (pp. 371−378). Berlin: Springer-Verlag.

Cardoso, M. J. L., Sakate, M., Ciampolini, P., Moutinho, F. Q., & Cherubini, A. L. (2004). Envenomation by scorpion in dog − case report. *Journal of Venomous Animals and Toxins Including Tropical Diseases, 10*, 98−105.

Couraud, F., & Jover, E. (1984). Mechanisms of action of scorpion toxins. In A. T. Tu (Ed.), *Handbook of natural toxins* (Vol. 2, pp. 659−678). New York: Dekker.

Cloudsley-Thompson, J. L. (1990). Scorpions in mythology, folklore, and history. In G. A. Polis (Ed.), *The biology of scorpions* (pp. 462−485). Stanford, CA: Stanford University Press.

Cloudsley-Thompson, J. L. (1993). Spiders and scorpions (Araneae and Scorpiones). In R. P. Lane, & R. W. Crosskey (Eds.), *Medical insects and arachnids* (pp. 659−682). Chapman & Hall.

Coddington, J. A., Larcher, S. F., & Cokendolpher, J.C. (1990). The systematic status of Arachnida, exclusive of Acari, in North America north of Mexico. In M. Kosztarab & C. W. Schaefer (Eds.), *Systematics of the North American insects and arachnids: Status and needs. Virginia Agricultural Experiment Station Information series 90-1* (pp. 5−20). Blacksburg: Virginia Polytechnic Institute and State University.

Cordeiro, F. F., Sakate, M., Fernandez, V., & Cuyumjian, P. R. (2006). Clinical and cardiovascular alterations produced by scorpion envenomation in dogs. *Journal of Venomous Animals and Toxins Including Tropical Diseases, 12*, 19−43.

Dehesa-Davila, M., Alagon, A. C., & Possani, L. D. (1995). Clinical toxicology of scorpion stings. In J. Meier, & J. White (Eds.), *Handbook of clinical toxicology of animal venoms and poisons* (pp. 221−238). Boca Raton., FL: CRC Press.

Diniz, C. R. (1978). Chemical and pharmacological aspects of Tityinae venoms. In S. Bettini (Ed.), *Arthropod venoms* (pp. 379−394). Berlin: Springer-Verlag.

Dupre, G. (2007). Conspectus Genericus Scorpionorum 1758-2006 (Arachnida: Scorpiones). *Euscorpius, 50*, 1−31.

El-Asmar, M. F. (1984). Metabolic effect of scorpion venom. In A. T. Tu (Ed.), *Handbook of natural toxins* (Vol. 2, pp. 551−576). New York: Dekker.

El-Ayeb, M., & Delori, P. (1984). Immunology and immunochemistry of scorpion neurotoxins. In A. T. Tu (Ed.), *Handbook of natural toxins* (Vol. 2, pp. 607−638). New York: Dekker.

Efrati, P. (1978). Symptomatology and treatment of Buthinae stings. In S. Bettini (Ed.), *Arthropod venoms* (pp. 312−316). Berlin: Springer-Verlag.

Ennik, F. (1972). A short review of scorpion biology, management of stings, and control. *Califfornia Vector Views, 19*, 69−80.

Fet, V., & Selden, P. A. (Eds.). (2001). *Scorpions. In memoriam Gary A. Polis*. Burnham Beeches, Bucks: British Arachnological Society.

Fet, V., Sissom, W. D., Lowe, G., & Braunwalder, M. E. (2000). *Catalog of the scorpions of the world (1758-1998)*. New York: The New York Entomological Society, 690 p.

Fet, V., Soleglad, M. E., Neff, D. P. A., & Stathi, I. (2004). Tarsal armature in the superfamily Iuroidea (Scorpiones: Iurida). *Revista Ibérica de Arachnología, 10*, 17–40.

Gertsch, W. J., & Allred, D. M. (1965). Scorpions of the Nevada test site. *Brigham Young Univeristy Science Bulletin, Biological Series, 6*, 1–15.

Gertsch, W. J., & Soleglad, M. E. (1972). Studies of North American scorpions of the genera *Uroctonus* and *Vejovis* (Scorpionida, Vejovidae). *Bulletin of the American Museum of Natural History, 148*, 551–608.

Gomez-Ortiz, S., Leal, O., & Osuna, S. (1972). Contribution to the study of scorpions. *Revista Da Faculdade de Medicina Veterinaria e Zootecnia Da Universidade de Sao Paulo, 34*, 25–41.

Gonzalez-Santillan, E., & Prendini, L. (2013). Redefinition and generic revision of the North American vaejovid scorpion subfamily Syntropinae Kraepelin, 1904, with descriptions of six new genera. *Bulletin of the American Museum of Natural History, 382*, 1–71.

Goyffon, M., & Kovoor, J. (1978). Chactoid venoms. In S. Bettini (Ed.), *Arthropod venoms* (pp. 395–418). Berlin: Springer-Verlag.

Gueron, M., & Ovsychcher, I. (1984). Cardiovascular effects of scorpion venoms. In A. T. Tu (Ed.), *Handbook of natural toxins* (Vol. 2, pp. 639–658). New York: Dekker.

Hassan, F. (1984). Production of scorpion antivenin. In A. T. Tu (Ed.), *Handbook of natural toxins* (Vol. 2, pp. 577–606). New York: Dekker.

Hjelle, J. T. (1972). Scorpions of the northern California coast ranges (Arachnida: Scorpionida). *Occasional Papers of the California Academy of Science, 92*, 1–59.

Ismail, M. (1995). The scorpion envenoming syndrome. *Toxicon, 33*, 825–858.

Johnson, J. D., & Allred, D. M. (1972). Scorpions of Utah. *The Great Basin Naturalist, 32*, 154–170.

Kaestner, A. (1968). Order Scorpiones, scorpions. In *Invertebrate Zoology: Vol. II. Arthropod relatives, Chelicerata and Myriapoda* (pp. 101–114). New York: Interscience.

Keegan, H. L. (1980). *Scorpions of medical importance*. Jackson: Univ. Press of Mississippi, 140 p.

Kovařík, F. (2009). *Illustrated catalog of scorpions. Part I. Introductory remarks; keys to families and genera; subfamily Scorpioninae with keys to* Heterometrus *and* Pandinus *species*. Prague, Czech Republic: Clarion Publications, 169 p.

Kovařík, F., & Onjanguran Affilastro, A. (2013). *Illustrated catalog of scorpions. Part II. Bothriuridae; Chaerilidae; Buthidae I., genera* Compsobuthus, hottentotta, Isometrus, Lychas, *and* Sassanidotus. Prague, Czech Republic: Clarion Publications, 400 p.

Lucas, S. M., & Meier, J. (1995). Biology and distribution of scorpions of medical importance. In J. Meier, & J. White (Eds.), *Handbook of clinical toxicology of animal venoms and poisons* (pp. 205–219). Boca Raton, FL: CRC Press.

Machado, J. C., & Filho, J. F. S. (1976/77). Indução de pancreatite hemorrágica aguda no cão por veneno escorpiônico de *T. serrulatus*. *Memorias do Instituto Butantan, 40/41*, 1–9.

Meerdink, G. L. (1983). Bites and stings of venomous animals. In R. W. Kirk (Ed.), *Current veterinary therapy VIII: small animal practice* (p. 155). Philadelphia: W.B. Saunders.

Muma, M. H. (1967). *Scorpions, whip scorpions and wind scorpions of Florida. Arthropods of Florida and Neighboring Land Areas* (Vol. 4). Gainesville, FL: Florida Dept. Agric, 28 p.

Pantoja, J. L., Renner, I. G., Abramson, S. B., & Edmonson, H. A. (1983). Production of acute hemorrhagic pancreatitis in the dog using venom of the scorpion, *Buthus quinquestriatus*. *Digestive Diseases and Sciences, 28*, 429–439.

Polis, G. A. (Ed.). (1990). *The biology of scorpions*. Stanford, CA: Stanford Univ. Press, 587 p.

Ponce-Saavedra, J., Francke, O. F., Quijano-Ravell, A. F., & Cortés Santillán, R. (2016). Alacranes (Arachnida: Scorpiones) de importancia para la salud Pública en México. *Folia Entomologica Mexicana, 2*, 45–70.

Possani, L. D. (1984). Structure of scorpion toxins. In A. T. Tu (Ed.), *Handbook of natural toxins* (Vol. 2, pp. 513–550). New York: Dekker.

Prendini, L., & Wheeler, W. C. (2005). Scorpion higher phylogeny and classification, taxonomic anarchy, and standards for peer review in online publishing. *Cladistics, 21*, 446–494.

Rankin, W., & Walls, J. G. (1994). *Tarantulas and Scorpions: Their care in captivity*. Neptune City, NJ: T.F.H. Publications, Inc., 64 p.

Rein, J. O. (2017). *The scorpion files*. Trondheim: Norwegian University of Science and Technology. Available from: http://www.ntnu.no/ub/scorpion-files/.

Shulov, A., & Levy, G. (1978). Systematics and biology of Buthinae. In S. Bettini (Ed.), *Arthropod venoms* (pp. 309–312). Berlin: Springer-Verlag.

Simard, J. M., & Watt, D. D. (1990). Venoms and toxins. In G. A. Polis (Ed.), *The biology of scorpions* (pp. 414–444). Stanford, CA: Stanford Univ. Press.

Sissom, W. D. (1990). Systematics, biogeography, and paleontology. In G. A. Polis (Ed.), *The biology of scorpions* (pp. 64–160). Stanford, CA: Stanford Univ. Press.

Soleglad, M. E., & Sissom, W. D. (2001). Phylogeny of the family Euscorpiidae Laurie, 1896: A major revision. In V. Fet, & P. A. Selden (Eds.), *Scorpions 2001. In memoriam gary a. Polis* (pp. 25–111). Burnham Beeches, Bucks: British Arachnological Society.

Soleglad, M. E., & Fet, V. (2003). High-level systematics and phylogeny of the extant scorpions (Scorpiones: Orthosterni). *Euscorpius, 11*, 1–175.

Soleglad, M. E., & Fet, V. (2006). Contributions to scorpion systematics. II. Stahnkeini, a new tribe in the scorpion family Vaejovidae (Scorpiones: Chactoidea). *Euscorpius, 40*, 1–32.

Soleglad, M. E., & Fet, V. (2008). Contributions to scorpion systematics. Iii. Subfamilies Smeringurinae and Syntropinae (Scorpiones: Vaejovidae). *Euscorpius, 71*, 1–115.

Stockwell, S. A. (1992). Systematic observations on North American Scorpionida with a key and checklist of the families and genera. *Journal of Medical Entomology, 29*, 407–422.

Theakston, R. D. G., & Warrell, D. A. (1991). Antivenoms: A list of hyperimmune sera currently available for the treatment of envenoming by bites and stings. *Toxicon, 29*, 1419–1470.

Tu, A. T. (Ed.). (1984). *Handbook of Natural Toxins*, Vol. 2. Insect poisons, allergens, and other invertebrate venoms. Dekker, New York. 732 pp.

Wainschel, J., Russell, F. E., & Gertsch, W. S. (1974). Bites of spiders and other arthropods. In H. F. Conn (Ed.), *Current therapy* (pp. 865–867). Philadelphia: W. B. Saunders Company.

Whittemore, F. W., & Keegan, H. L. (1963). Medically important scorpions in the Pacific area. In H. L. Keegan, & M. V. Macfarlane (Eds.), *Venomous and poisonous animals and noxious plants of the Pacific region"* (pp. 107–110). New York: Permagon Press.

Williams, S. C. (1969). Birth activities of some North American scorpions. *Proceedings of the Caliornia Academy of Sciences Series, 4*(37), 1–24.

Williams, S. C. (1976). The scorpion fauna of California. *Bulletin of the Society for Vector Ecology, 3*, 1–4.

Williams, S. C. (1980). Scorpions of Baja California, Mexico and adjacent islands. *Occasional Papers of the California Academy of Sciences, 135*, 1–127.

Zlotkin, E., Miranda, F., & Rochat, H. (1978). Chemistry and pharmacology of Buthinae scorpion venoms. In S. Bettini (Ed.), *Arthropod venoms* (pp. 317–370). Berlin: Springer-Verlag.

Chapter 24

Solpugids (Solifugae)

Gary R. Mullen
Department of Entomology and Plant Pathology, Auburn University, Auburn, AL, United States

The order Solifugae (Solpugida) includes 12 families, more than 150 genera, and approximately 1,100 species worldwide (Zhang, 2013; Integrated Taxonomic Information System; https://itis.gov). They occur most commonly in tropical and subtropical deserts in Africa, the Middle East, western Asia, and the Americas. They occur in the United States and southern Europe, but not in Australia or New Zealand. The two major families in North America are Ammotrechidae and Eremobatidae, together represented by 11 genera and about 120 species. Most of the species in the United States occur in arid regions of the western states. The exception is *Ammotrechella stimpsoni,* which is found under the bark of termite-infested tree stumps in Florida. For a comprehensive treatment of solpugids, including keys to the families and genera worldwide, see Punzo (1998). For further information on solpugids in the United States and the Western Hemisphere, see Muma (1951, 1970, 1976), and Muma and Muma (1988).

Members of this group are variously known as **solpugids, sun-spiders, wind-spiders, wind-scorpions, camel-spiders, barrel-spiders, false-spiders**, and **romans**. Local names in the United States include **bulldozer-spiders** in the Big Bend area of Texas and **sand-puppies** in Wyoming. They also are known by the British terms **jerrymander** and **jerrymunglum.** In Mexico they are called *mata venado* ("deer killer") in the mistaken belief that they can kill large animals. In southern Africa, solpugids are called *haarskeerders* ("hair-cutters") and *baardskeerders* ("beard-cutters"). According to local lore, females are attracted to the hair of sleeping humans and other animals, which they clip with their chelicerae and carry to their burrows, or other retreats, to line their nests in preparation for egg laying.

Solpugids are usually yellow or brownish and rather hairy. The body length varies from 1 to 7 cm (0.4–3 in.), with the largest species having a leg span up to 12 cm (4.7 in.). The prosoma and opisthosoma are broadly joined, with the latter being visibly segmented (Fig. 24.1). The most prominent structures are the greatly enlarged pair of chelicerae that are used to seize, cut, and tear apart their food. As a group, solpugids lack venom glands and rely on their size and powerful pincer-like chelicerae to subdue prey. A pair of flagella on each chelicera helps to distinguish males from females. The pedipalps are long and leglike, each ending in an eversible adhesive organ used to grasp prey and facilitate climbing. The first pair of legs is

FIGURE 24.1 A female solpugid, South Africa; locally called a jerrymunglum. Despite their greatly enlarged chelicerae and formidable appearance, solpugids lack venom glands and are generally harmless to humans. *Photograph by Jon Richford, Wikimedia Commons.*

Medical and Veterinary Entomology. https://doi.org/10.1016/B978-0-12-814043-7.00024-8

modified as slender tactile organs that are held outstretched as the solpugid moves about. Unique, mallet-shaped structures call **racquet organs** (malleoli) are borne on the underside of the fourth pair of legs in both sexes. They are innervated and apparently function as both tactile organs and chemoreceptors while probing various substrates, presumably to detect cues associated with prey and potential mates.

The name "sun-spider" refers to species that are active during the daytime. The name "wind-scorpion" reflects their peculiar, rapid movement as they run about the surface of desert sands hunting prey; they give the appearance of being blown across the sand and have been likened to tumbleweeds. The name "camel-spider" refers to the arch-shaped plate on the dorsum of the prosoma of many species.

Solpugids are typically crepuscular or nocturnal, hiding during the day under stones and in crevices, or burrowing into loose soil in arid habitats largely devoid of vegetation; in Africa, however, they also are found in grasslands and forests. Their food consists primarily of insects (e.g., termites, tenebrionid beetles), spiders, scorpions, and other ground-dwelling arthropods. The larger species also are known to attack and kill small lizards and snakes, mice, birds, and other vertebrates.

They produce only one generation per year. Mating typically involves the male depositing a spermatophore on the substrate, after which he flips the female on her back and transfers the sperm with his chelicerae to the female genital pore. In some species, however, insemination is direct. The female then excavates a burrow in which she deposits about 50–200 eggs, depending on the species. The females of some species guard the eggs until they hatch. The resultant offspring undergo 9–10 nymphal instars before reaching maturity as adults.

Despite their formidable appearance and aggressive posturing, solpugids are relatively harmless and cause little medical concern. Because they are not venomous, most such bites are limited to painful nips, although the powerful chelicerae of larger species can puncture or lacerate the skin. In a case involving U.S. military personnel in the Persian Gulf, an individual was bitten on the lip and required 10 stitches to close the wound (Conlon, 1991). The greatest concern is usually preventing secondary infections, which can lead to painful swellings, necrosis of tissue surrounding the bite site, and gangrene.

REFERENCES AND FURTHER READING

Aruchami, M., & Sundara-Rajulu, G. (1978). An investigation on the poison glands and the nature of the venom of *Rhagodes nigrocinctus* (Solifugae: Arachnida). *National Academy Science Letters, 1*, 191–192.

Cloudsley-Thompson, J. L. (1958). *Spiders, scorpions, Centipedes and Mites*. New York: Pergamon, 228 pp.

Cloudsley-Thompson, J. L. (1992). Solifugae and keeping them in captivity. In J. E. Cooper, P. Pearce-Kelly, & D. L. Williams (Eds.), *Arachnida. Symposium on spiders and their Allies, London (1987)* (pp. 52–56). Keighley: Chiron Publications.

Conlon, J. M. (1991). *Vectors & war. Part 2. Desert Storm. Wing Beats* (Vol. 22, pp. 16–20). Florida Mosquito Control Association.

Harvey, M. S. (2003). *Catalogue of the smaller arachnid orders of the world: Amblypygi, Uropygi, Schizomida, Palpigradi, Ricinulei and Solifugae*. Collingwood, Australia: CSIRO Publishing.

Harvey, M. S. (2011). Smaller Arachnid Orders Catalogue (SAOCat) database. In Bisby, F. A., Roskov, Y. R., Orrell, T. M., Nicolson, D, Paglinawan, L. E., Bailly, N., et al. (Eds). Species 2000 & ITIS Catalogue of Life: 2011 Annual Checklist. Digital resource at http://www.catalogueoflife.org/annual-checklist/2011. Species2000, Reading, UK.

Hickin, N. E. (1984). Solifugae. In N. E. Hickin (Ed.), *Pest animals in Buildings* (pp. 85–86). London: Godwin, 385 pp.

Maury, E. A. (1982). Solifugae de Colombia y Venezuela (Solifugae, Ammotrechidae). *Journal of Arachnology, 10*, 123–143.

Muma, M. H. (1951). The arachnid order Solpugida in the United States. *Bulletin of the American Museum of Natural History, 97*, 35–141.

Muma, M. H. (1970). A synoptic review of North America, Central America, and west indian Solpugida (Arthropoda: Arachnida). In *Arthropods of Florida and Neighboring Land areas* (Vol. 5, pp. 1–62). Gainesville: Florida Department of Agriculture and Consumer Services.

Muma, M. H. (1976). *A review of solpugid families with an annotated list of Western Hemisphere solpugids* (Vol. 2, pp. 1–33). New Mexico University, Silver City: Publication of the Office of Research.

Muma, M. H., & Muma, K. E. (1988). *The arachnid order Solpugida in the United States (Suppl. 1, a biological review, pp. 35)*. Silver City, New Mexico: Southwest Offset.

Punzo, F. (1998). *The Biology of camel-spiders (Arachnida, Solifugae)*. Dordrecht/Norwell, MA: Kluwer Academic, 301 pp.

Van der Meijden, A., Langer, F., Boistel, R., Vagovic, P., & Heethoff, M. (2012). Functional morphology and bite performance of raptorial chelicerae of camel spiders (Solifugae). *Journal of Experimental Biology, 215*(Pt 19), 3411–3418.

Wharton, R. A. (1981). Namibian Solifugae (Arachnida). *Cimberbasia Memoir, 5*, 1–87.

Zhang, Z.-Q. (Ed.). (2013). Animal Biodiversity: An Outline of Higher-level Classification and Survey of Taxonomic Richness (Addenda). *Zootaxa*, *3703*, 1–82.

Chapter 25

Spiders (Araneae)

Gary R. Mullen[1] and Richard S. Vetter[2]

[1]*Department of Entomology and Plant Pathology, Auburn University, Auburn, AL, United States;* [2]*Department of Entomology, University of California, Riverside, CA, United States*

All spiders, except the Symphytognathidae and Uloboridae, possess venom glands that are used to subdue captured prey. When threatened, however, spiders will often defend themselves by biting, thereby injecting those same toxins into vertebrate skin. In most cases, the venom produces only mild, localized reactions that do not warrant medical attention. Other spiders have much more potent venoms that can cause severe reactions in bite victims, occasionally resulting in deaths. Only about 60 species of spiders worldwide are considered to have significant medical importance. Among them are only a few genera that are dangerous to humans. Most occur in the subtropics and tropics. A few tropical species, however, have extended their ranges into temperate regions, particularly those with Mediterranean-like climates.

Envenomation by spiders is called **araneism** after Araneae, the arachnid order to which spiders belong. Separate names, however, are given to bites or syndromes associated with the more dangerous spider genera, each of which is generally characterized by typical clinical signs and symptoms. Examples include atraxism (*Atrax* spp.), latrodectism (*Latrodectus* spp.), loxoscelism (*Loxosceles* spp.), and phoneutriism (*Phoneutria* spp.). There also are cases in which individuals develop an abnormal fear of spiders such that the mere sight of one can cause panic or hysteria, a condition called **arachnophobia** or, more specifically, **araneophobia**. This should not be confused with the unfortunate disdain that many people have for spiders, often reflecting their upbringing and misconceptions about spiders in general.

TAXONOMY

More than 4,000 genera and 46,000 species of spiders have been described worldwide. In North America alone there are 68 families, 569 genera, and 3,700 species (Ubick et al., 2017). Among the 113 families of spiders, about 20 include species that reportedly cause medical concerns when they bite humans and other animals. The five more important families are the Dipluridae, Atracidae, Theraphosidae, Sicariidae (formerly Loxoscelidae), and Theridiidae.

The order Araneae is divided into two suborders: the Mesothelae and Opisthothelae. The Mesothelae include the single family Liphistiidae, a small group of primitive spiders in Southeast Asia and the Indo-Malaysian region. The Opisthothelae are composed of two groups: the Mygalomorphae (tarantula-like spiders) and Araneomorphae (all other spiders). The mygalomorphs are more primitive, represented by trap-door spiders, funnel-web spiders, and tarantulas. They include the largest spiders. The araneomorphs are a very diverse group that include wandering spiders and those that are familiar to most people by the diversity of silken webs that they produce.

An online taxonomic catalogue of the world spider fauna, which is routinely updated, can be found in the World Spider Catalog (2017). For identification keys to the families and genera of mygalomorphs, see Raven (1985), and for North American families and genera of spiders, see Ubick et al. (2017).

The following is a synopsis of the major families of spiders of medical-veterinary importance.

MYGALOMORPH SPIDERS

Actinopodidae

This small group is closely related to the typical trap-door spiders of the family Ctenizidae, which they resemble both morphologically and behaviorally. They construct vertical, silk-lined burrows in the soil, the opening at the surface of which is covered by a hinged trap door. Their venom is weakly neurotoxic to vertebrates and causes no necrosis. *Actinopus* in Central and South America has been reported biting humans, producing only local pain

Medical and Veterinary Entomology. https://doi.org/10.1016/B978-0-12-814043-7.00025-X

and transient muscle contractions. Similarly, the bite of a *Missulena* sp. in Australia has been reported to cause a moderate reaction in a child.

Barychelidae

Members of this family are closely related to the Theraphosidae, or tarantulas, and are largely restricted to southern Africa and Australia. *Idiommata blackwalli* occurs widely throughout Australia in dry areas where it constructs silk-lined burrows provided with a saucer-shaped door that fits tightly into the opening at the ground surface. Its bite is painful, causing local redness and edema in humans. Most encounters occur when wandering males enter homes during the late summer and early fall, or when individuals are dislodged from their burrows in new suburban areas when people rake or otherwise disturb their yards.

Dipluridae

Known as sheet-web or **funnel-web-building tarantulas**, diplurids construct burrows in the ground in a wide range of habitats. The family includes 24 genera. They are particularly abundant in the Southern Hemisphere and Australian region. The venom of *Trechona* species is especially toxic to humans and has been reported to cause human deaths in South America. A species of particular importance is *T. venosa*, which occurs in tropical forests and coastal areas, where they are encountered on vegetation and along trails. They are very aggressive and, if disturbed, will readily bite.

Atracidae

Formerly included in the Hexathelidae, some of the members of this family are regarded as among the most toxic of spiders for humans (Isbister et al., 2005). Among the recognized genera, *Atrax* and *Hadronyche* are the more dangerous. They are known as **funnel-web spiders**, referring to the extensive silk about the entrance, and extending into their shallow silk-lined burrows in the ground, among rocks, or in stumps and rot holes of trees. The most serious bites are caused by six species, most notably, the Sydney funnel-web sider (*A. robustus*) in Australia.

Macrothelidae

Previously considered to be in the Hexathelidae, the mygalomorph family Macrothelidae is represented by only one genus of medical significance, i.e., *Macrothele*. They occur primarily in Asia and Java.

Theraphosidae

This is the largest mygalomorph family, with 140 recognized genera. They are best known for their large size and hairy appearance, familiar to most people as **tarantulas**. As a group, they are primarily tropical and subtropical, occurring widely throughout both the Old World and New World where they are variously known as bird spiders, bird-eating spiders, monkey spiders, or baboon spiders. In North America, they extend into the southwestern United States but do not naturally occur east of the Mississippi River. Although the bite of most species is relatively harmless, several genera can cause severe envenomation, particularly in South America, where the genera dangerous to humans are *Acanthoscurria*, *Pamphobeteus*, *Phormictopus*, and *Sericopelma*. On the Indian subcontinent, the genus *Poecilotheria* has venom that causes a reaction similar to widow venom, perhaps making it the most dangerous tarantula.

ARANEOMORPH SPIDERS

Agelenidae

Agelenid spiders are called **funnel weavers**, not to be confused with the mygalomorph funnel-web spiders (Dipluridae and Atracidae). They typically build horizontal sheet webs with a tubular retreat, or "funnel," leading into a protected recess. When their webs are constructed in vegetation, they are often called grass spiders. A few species occur in homes, especially in basements and cellars, where their chances of encounters with humans are greatest. In North America, the only species that has drawn medical attention is the hobo spider, *Eratigena agrestis*, although it is currently considered unlikely to be toxic to humans (see later). In Europe, *Agelena labyrinthica* has been reported to bite humans. A person bitten on the finger by *Pireneitega pyrenaea* (formerly *Coelotes obesus*) is said to have experienced intense pain and localized paralysis that persisted for a few hours (Maretic and Lebez, 1979).

Araneidae

This is one of the largest families of spiders, familiar to most people because of the symmetrical spiral-like webs which they construct for snaring flying insects. Known as **orbweavers**, they are commonly found around homes and other dwellings where they take advantage of artificial lights to attract prey at night. They seldom bite humans or other vertebrates and usually cause only minor, temporary discomfort when they do. Even the North American black and yellow garden spider, *Argiope aurantia*, produces only

localized pain, redness, and edema on the rare occasions in which it has been known to bite. This is a large and colorful species that often attracts the attention of homeowners in the late summer and fall. *Argiope lobata* in Europe causes a similar, mild reaction. The only araneids that have been reported as a medical concern are members of the genus *Mastophora*, known as **bolas spiders**, and in Peru, Bolivia, and Chile, as *podadoras*. The bite of some South American species is said to cause localized, necrotic skin lesions, with general pain, edema, and hematuria (Bucherl, 1971). However, based on the rarity of encounters and the lack of documented cases in the literature, there is reason to question the significance of bolas spiders as a cause of bites to humans.

Ctenidae

Members of this family are wandering spiders that do not build webs. Most are of moderate size (1.5−2.5 cm in body length) and occur primarily in ground litter and low vegetation where they hunt prey. They resemble wolf spiders (Lycosidae) in both their general appearance and behavior. The taxa of greatest medical concern are the South American species *Phoneutria nigriventer* and *P. keyserlingi* from coastal Brazilian forests. These are relatively large species (>3 cm body length) that occasionally cause severe envenomation; most bites in human adults result in only mild effects. In Central America, several species of *Cupiennius* have been involved in bites, with pain lasting only about 30 min (Barth, 2001). Both *Phoneutria* and *Cupiennius* spp. have been found in international cargo, most commonly bananas. For information on identification of these spiders and medical aspects of their bites, see Barth (2001), Vetter and Hillebrecht (2008), and Vetter et al. (2014).

Desidae

This family of cribellate spiders is of veterinary importance in Australia primarily because of *Balumna insignis*, a species reported to cause painful bites and inflammation in horses. This, and related species, are dark, robust spiders that construct funnel-shaped webs, not to be confused with that of the funnel-web spiders (Atracidae). Their web forms an untidy sheet leading to funnel-like entrances in logs, tree trunks, rock walls, and crevices around window frames of buildings. They are not aggressive and only infrequently bite humans and domestic animals.

Dysderidae

Although there are over 250 *Dysdera* species worldwide, only *D. crocata*, has become established worldwide via commerce. It has a dark cinnamon-colored cephalothorax, gray-tan abdomen, and very large fangs that it presents when threatened. Its bite is painful, probably due to fang penetration, not venom toxicity, with mild effects that dissipate within 1 hour (Vetter and Isbister, 2006).

Eutichuridae

Previously the Clubionidae was a large, diverse family known as the sac spiders, referring to the silken tubular retreats that they typically make in rolled-up leaves, in other ground litter, and under bark and stones. This family has been divided into many smaller families, of which only members of the genus *Cheiracanthium* (now in the family Eutichuridae) are typically involved in envenomations. Although bites can be painful like a bee sting, most result in cases of minor medical importance (Vetter et al., 2006; McKeown et al., 2014). They are nocturnal, vagrant spiders that commonly are found hunting prey on plants and incidentally enter houses and other buildings.

Gnaphosidae

These wandering spiders are commonly found under stones, in rolled leaves, and ground debris and are known as ground spiders. The bites of most gnaphosids are relatively harmless. *Herpyllus ecclesiasticus* and *Scotophaeus blackwalli* (formerly *H. blackwalli*) have been reported to cause painful bites in the United States, usually on entering homes at night.

Lamponidae

Members of this family are similar to gnaphosids in their habitats and behavior. The white-tailed spider (*Lampona cylindrata/murina* complex) has been reportedly associated with necrotic skin lesions for more than 20 years in Australia. These cases have not been corroborated, however, as being caused by *Lampona* venom. White-tailed spiders were shown to be virtually harmless when a total of 130 verified white-tailed spider bites in humans resulted in only mild effects and no necrosis (Isbister and Gray, 2003b).

Lycosidae

Commonly known as **wolf spiders**, lycosids represent a highly successful family of hunting spiders that are noted for their relatively large size (up to 4 cm), and hairy appearance. Their posterior median and posterior lateral eyes are greatly enlarged and aid them visually in capturing prey. Members of the genus *Lycosa* possess cytotoxic venoms that can cause painful bites; however, they also

have powerful cheliceral musculature, so some of the pain could be due to fang penetration. Included in this genus are the so-called "tarantulas" of Europe, such as *L. tarentula* of tarantism fame. Although the bites of many wolf spiders are painful, they generally cause only temporary, localized discomfort.

Miturgidae

The miturgid spider that has drawn medical attention is *Elassoctenus harpax* of Western Australia, which reportedly can inflict a painful bite. This spider used to be in the family Zoridae. Also, the sac spiders of the genus *Cheiracanthium* were moved from the Clubionidae to the Miturgidae, and then to the Eutichuridae.

Oxyopidae

Members of this largely tropical and subtropical family are called **lynx spiders**. They are active hunters that rely on their keen eye sight, speed, and agility to capture prey while climbing in foliage. Although the family is not generally regarded as being medically important, females of the green lynx spider (*Peucetia viridans*) are known to forcibly expel venom from their fangs as a defensive response, especially when guarding their egg sacs. Droplets can be squirted up to 20 cm (Fink, 1984), and on contact with human eyes can cause impaired vision and moderately severe conjunctivitis.

Pisauridae

This family is closely related to the wolf spiders, which they strongly resemble. They occur most frequently near water, where members of the genus *Dolomedes* are adept at moving about on the water surface to capture prey, hence their common name **fishing spiders**. Spiders of the genus *Pisaurina* are known as **nursery-web spiders** because of the habit of females suspending their egg sacs in a protective silken "nursery" in vegetation and guarding the resultant spiderlings until they disperse. Because of their large size (body length up to 4 cm or more) and powerful chelicerae, they can bite if handled, causing local pain and transient swelling. The bite of the European species *Dolomedes fimbriatus* is reported to cause a reaction similar to that of the agelenid *Pireneitega pyrenaea*.

Salticidae

This is the largest family of spiders, with 620 genera and nearly 6,000 species widely distributed throughout the world. They are known as **jumping spiders** because of their habit of stalking and pouncing on prey or jumping to escape when threatened. The anterior median eyes are complex and greatly enlarged, providing them with the keenest vision of all spiders. Some of the larger species can be aggressive and inflict painful bites when handled or pressed against the skin. The venom of at least some species contains cytotoxins that cause necrotic lesions at the puncture site, often being slow to heal. The bite of *Phidippus johnsoni* can cause a dull, throbbing pain that may persist for a few hours, in addition to swelling, tenderness, and itching that may last for 1 to 4 days following the bite (Russell, 1970).

Segestriidae

Members of this relatively small family live in silken retreats under stones and bark or in crevices of wood and rocks. They are active nocturnal hunters that may enter homes or construct their retreats in and around human dwellings. Despite their large, well-developed chelicerae, they are not very aggressive, rarely bite, and are not considered to be very toxic. Nonetheless a few cases of human bites by the European species *Segestria florentina* reportedly have involved local pain, redness, and swelling, and occasionally nausea and vertigo.

Sicariidae (Including the Former Loxoscelidae)

The sicariids are a small group of relatively primitive araneomorphs. Included in this family are the **recluse spiders** in the genus *Loxosceles*, which can cause severe necrosis in a minority of cases. Approximately 85 species of *Loxosceles* have been described in the Americas and nearly another 50 species worldwide. They are generally similar in appearance and are difficult to recognize from one another by the nonspecialist. They typically are found in ground litter and under bark or stones; a few species occur in caves, and some are decidedly synanthropic, living in close association with humans. The genus *Sicarius* in Africa also has been shown to be highly toxic in laboratory studies, although its behavior (e.g., burying itself in sand under rocks) and remote distribution limit its encounter with humans.

Theridiidae

Members of this large family are called **cobweb weavers** or **comb-footed spiders**. The latter refers to a row of serrated bristles on the hind tarsus that is used to comb the silk from the spinnerets during construction of their irregular webs or wrapping prey. The only genus considered particularly toxic to humans and domestic animals is *Latrodectus*, which includes the **widows**, or

shoe-button spiders. Another genus that is less toxic to humans is *Steatoda*. It shares venom protein similarities with *Latrodectus* (Garb and Hayashi, 2013), which accounts for the mild latrodectism symptoms in cases of envenomation. Several species in South America, including *S. andina,* the well-known *cirari* of Bolivia, Chile, and Paraguay, are said to cause serious envenomation (Southcott, 1984). Although venom of the Mediterranean species *Steatoda paykulliana* has been shown to be neurotoxic to guinea pigs, this spider has not been reported to bite humans. The cosmopolitan species *S. grossa*, known as the false black widow, causes only a local bite reaction with mild effects resembling *Latrodectus* bites (Isbister and Gray, 2003a). Another spider, *S. nobilis*, has generated concern since its introductions to England, California (USA), and Chile during the past 15−20 years. Despite verified bites by this species being only mild, the media in Great Britain continue to publish reports of dire envenomations by this species without substantiating evidence. *Steatoda* spiders are commonly misidentified as black widows by both patients and physicians. In one case of mistaken identity, black widow spider antivenin was administered for a *Steatoda* bite, which appeared to ameliorate the envenomation symptoms.

Thomisidae

Members of this family are called **crab spiders** because of their generally flattened appearance, laterigrade legs, and crablike gait. They usually are cryptically colored and ambush their prey from camouflaged sites such as tree bark and flower heads. As a group they are considered harmless to humans and other animals. Some species of *Misumenoides*, however, have been suspected, but not proved, of causing relatively minor bites in humans.

Trachelidae

These spiders were formerly included as a subfamily of the Clubionidae. Some *Trachelas* spp. (e.g., *T. volutus*) have been reported to bite humans in the United States, causing a stinging sensation and localized erythema and swelling.

MORPHOLOGY

The body of a spider is divided into two regions: the anterior **cephalothorax** (prosoma), which represents a fusion of the head and thoracic segments, and the abdomen (opisthosoma) (Fig. 25.1). The cephalothorax bears the chelicerae, pedipalps, eyes, and legs. The **chelicerae** (sing.

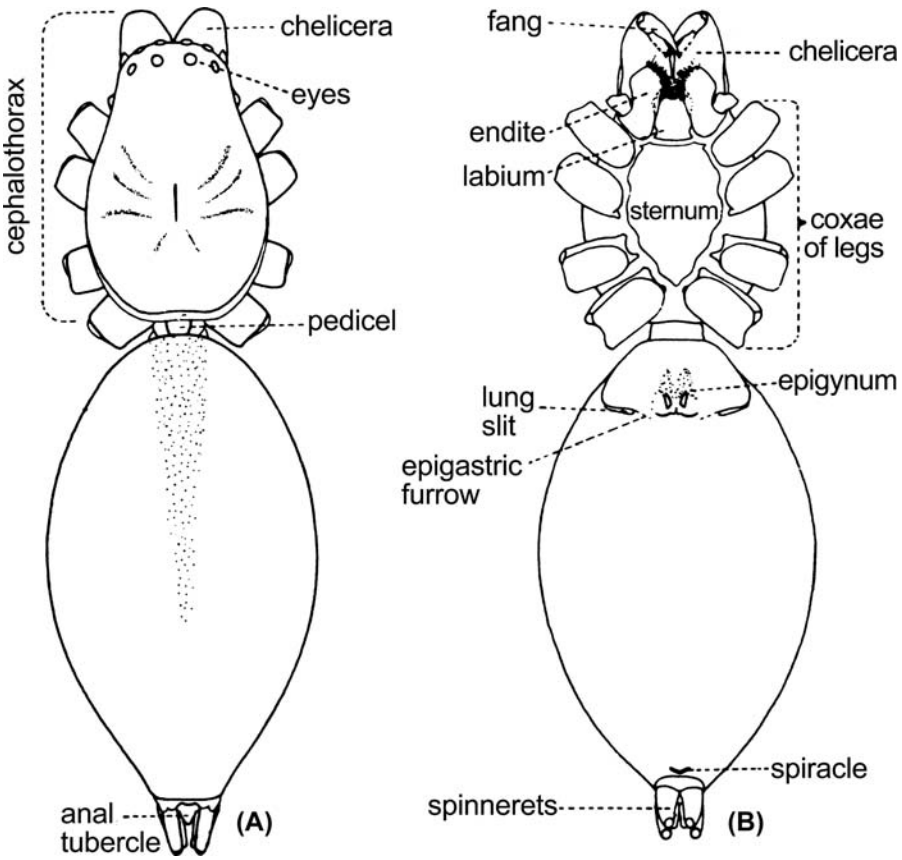


FIGURE 25.1 Morphology of representative spider. (A) Dorsal view. (B) Ventral view. *Modified from Kaston (1978).*

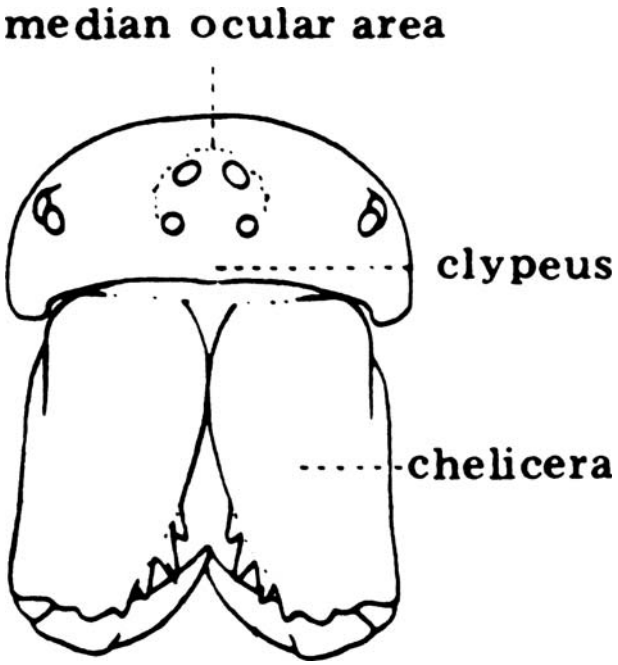


FIGURE 25.2 Head and chelicerae of representative spider, frontal view. *Modified from Kaston (1978).*

chelicera) are paired structures used to seize prey or to bite defensively when threatened (Figs. 25.1 and 25.2). They also serve other functions in different groups of spiders, such as digging by burrowing species, transporting prey, and carrying eggs sacs by some spiders. Each chelicera consists of two parts: a stout basal portion and a movable fang. The fang rests in a groove and is extended when the spider bites or otherwise attempts to grasp something. Near the tip of each fang is a tiny opening to the venom duct that leads from the **venom gland**; the latter is located in the basal part of the chelicera and usually extends back into the cephalothorax in araneomorphs. In mygalomorphs, the venom glands are restricted to the chelicerae. The mouth is located just behind the bases of the chelicerae. The chelicerae of mygalomorphs are oriented parallel to the long axis of the body and move parallel to one another in a vertical plane. These spiders strike downward when seizing prey. The chelicerae of araneomorphs are oriented perpendicular to the long axis of the body and are opposed to one another, moving together in a pincher-like motion.

Most spiders have eight eyes located anteriorly on the cephalothorax (Fig. 25.2). They are simple eyes (ocelli) usually arranged in two rows. Some spiders lack one or more pairs of eyes, as in *Loxosceles* spp. that have only six eyes. In other species, such as wolf spiders and jumping spiders, some eyes may be enlarged, reflecting their greater visual acuity. The size and arrangement of the eyes often serve as valuable taxonomic characters.

The **palps** (pedipalps) are a pair of six-segmented appendages that arise immediately behind the mouth. They are primarily tactile structures used in sensing the substrate, perceiving contact stimuli from conspecifics, and both detecting and manipulating prey. Whereas the palps of immature spiders and adult females tend to resemble legs, the palps of adult males are modified as copulatory organs. In such cases the terminal segment (palp tarsus) is enlarged with a ventral, bowl-shaped cavity enclosing a complex of specialized structures formed from the pretarsus that serve as an intromittent organ for inseminating females. Adult males usually can be recognized by the swollen terminus of their palps and their often smaller body size relative to females of the same species.

Spiders have four pairs of legs, each with seven segments: coxa, trochanter, femur, **patella**, tibia, **metatarsus**, and tarsus (Fig. 25.3). Spiders that run about on the ground and other substrates without building a trapping web typically have only two tarsal claws on each leg. Many of these hunting spiders possess dense tufts of hairs (**scopulae**) directly beneath the pair of claws or along the ventral side of the tarsus and metatarsus. They provide physical adhesion to facilitate climbing on smooth surfaces and grasping prey. The scopulae are especially prominent in tarantulas. Three tarsal claws are characteristic of web-building spiders. The single median claw on each leg is used to hold onto silken threads by those spiders that hang suspended in their webs.

The abdomen is connected with the cephalothorax by a narrow **pedicel**, which provides great flexibility and movement between the two body regions. One or

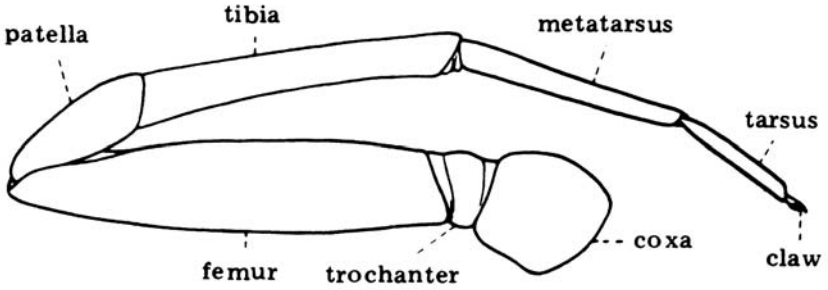


FIGURE 25.3 Leg of spider showing the seven leg segments. Spiders possess a patella and metatarsus not found in insects. *From Kaston (1978).*

two pairs of slit-like openings to the **book lungs**, the principal respiratory organs in spiders, are located ventrally on the second and third abdominal segments. The more "primitive" spiders tend to retain two pairs of book lungs, whereas most spiders have only one pair. The second pair of book lungs in some spiders is modified to form tubular tracheae that open via a spiracle, or pair of spiracles, on the third abdominal segment. Most spiders, however, have only a single spiracle located in front of the spinnerets.

The genital opening of both sexes is located ventrally on the second abdominal segment between the book lungs. In females of the more "advanced" spiders, a sclerotized copulatory structure called the **epigynum** (Fig. 25.1B) is located just in front of the genital opening and leads to the **spermathecae** where sperm is stored after mating. The presence of the epigynum is helpful in distinguishing adult females from immatures and males and in identifying species.

Located at the posterior end of the abdomen are the **spinnerets** (Fig. 25.1B) through which silk from several types of internal silk glands are extruded via small spigots. Most spiders have three pairs of spinnerets, the size and relative lengths of which provide useful taxonomic characters. In some groups of araneomorph spiders, a sieve-like plate of minute spigots called the **cribellum** is present in front of the anterior pair of spinnerets. These taxa, called cribellate spiders, also possess a row of specialized setae on the metatarsi of the fourth pair of legs called a **calamistrum**. The calamistrum is used to comb silk from the cribellum by rhythmic movements of the hindlegs. Most araneomorph spiders lack a cribellum and calamistrum and are referred to as ecribellate spiders.

LIFE HISTORY

Most reproductive activity of spiders occurs during the late spring and early summer, when mating typically takes place and eggs are deposited. Some species, however, are reproductively active in the late summer and fall. Embryonic development commonly takes about 2 weeks, during which time one or two prelarvae and a first-instar larva are formed within individual eggs contained in a silken egg sac. The larva subsequently molts into the second instar inside the sac and later emerges from the egg sac as a nymph. The young nymph, or **spiderling**, is a miniature of the adult spider with functional spinnerets and poison glands. The nymphs undergo two to 12 molts as juveniles, depending on the spider species, before reaching sexual maturity as adults.

In temperate regions, juvenile spiders are present throughout the summer months, with overwintering typically occurring as late-instar nymphs. However, if egg sacs are constructed by females late in the year (e.g., some orb-weaving spiders), the resultant spiderlings remain inside the egg sac until spring. In other cases, spiders overwinter as mature females. The life cycle for most spiders is 1 year, after which the adults die. Males are shorter lived than females and usually die soon after mating. Some of the more "primitive" spiders live much longer; purse-web spiders (Atypidae) may live 7 years, whereas the larger tarantulas (Theraphosidae) may live 20–30 years, especially in captivity.

BEHAVIOR AND ECOLOGY

Mating behavior in spiders varies greatly from one group to another and in some cases involves complex courtship displays. Prior to mating, the male typically deposits a droplet of sperm from the genital pore onto a small, silken platform called a sperm web. The droplet is then drawn into the copulatory organ at the tip of each male palp in a process called **sperm induction**. Sperm is stored in the palp until mating takes place. Following acceptance of a male's advance by a female, copulation is accomplished by indirect insemination in which the male transfers the sperm from his palps to the spermathecal ducts on the underside of the female's abdomen. Although the males of many species die soon after mating, others refill their palps with sperm and may mate with other females one or more times thereafter. Contrary to popular belief, most males either walk away or hurriedly retreat without being attacked and eaten by the female.

Oviposition typically occurs within a few weeks after mating. The female spins a silken sheet onto which she extrudes fertilized eggs from her genital opening, forming a mass in which the individual eggs are cemented together. She then protects the eggs by spinning multiple layers of silk to form the egg sac. More than one egg sac may be produced sequentially over a period of weeks, as commonly observed in black widow spiders. Although some species of spiders remain with the eggs to protect them until they hatch, most spiders provide no maternal care and leave the eggs to hatch unguarded.

Spiders have been very successful in exploiting a wide range of ecological habitats, including islands. This is accomplished, in part, by their specialized dispersal behavior called **ballooning**. Spiderlings, and even adults of many small species, are carried aloft on silken threads by which they can be conveyed by wind currents over considerable distances. To become airborne the spider orients into the wind, lifts the tip of its abdomen, and extrudes a strand of silk from the spinnerets. When the strand is long enough to provide enough buoyancy to support its body weight, the spider releases its grip on the substrate and drifts away.

Spiders can be categorized in three major groups based on their general behavior and feeding strategies:

burrowers, vagrants or wanderers, and web builders. Burrowing spiders usually excavate their burrows in the soil of suitable sites where they remain more-or-less permanently throughout their lives. They typically capture prey that comes within reach of the burrow entrance, or make short excursions to capture food and return to the safety of the burrow. Among this group are many of the ground-dwelling tarantulas, trap-door spiders, and burrowing wolf spiders. Vagrant spiders tend to wander extensively and may or may not regularly return to a given location where they have constructed a retreat. They do not produce a silken web for capturing food, but instead hunt or ambush their prey. Examples include many wolf spiders, jumping spiders, sac spiders, gnaphosids, and ctenids. These spiders commonly enter homes during their hunting forays and occasionally bite humans. The brown recluse spider also falls in this group. It differs from the others, however, by actually living indoors rather than wandering inside incidentally, and it makes a flimsy web. Web-building spiders construct various silken structures to detect and capture potential prey. The webs may be sheetlike, as in diplurids and agelenids, or more complex trapping webs like those of comb-footed spiders and orb weavers.

Burrowing and vagrant spiders usually rely on their physical size and strength to capture food, together with sufficiently potent venoms to quickly subdue their prey. The size of acceptable prey items is often positively correlated with the size of the spider itself. Web-building spiders, on the other hand, tend to rely on the use of silk to ensnare or immobilize their prey. They are able to use a wider range of prey items, often capturing and feeding on insects and other arthropods much larger than themselves. Those that construct aerial webs also have access to a wider variety of flying insects than do ground-dwelling spiders. The venom of web-building spiders is typically less potent than that of non-web-building species, accounting in part for the fact that bites of even the larger web builders are usually quite harmless.

PUBLIC HEALTH IMPORTANCE

Recognizing that virtually all spiders possess venom glands, it is not surprising that when they are threatened, species with chelicerae large enough to pierce the skin will bite humans and other animals. In most cases the reaction is minor, usually limited to mild, localized pain and slight to moderate swelling at the bite site. The severity of the reaction is dependent on the species of spider, its size, and the amount of venom injected.

The **venom** varies greatly among different spider taxa in terms of chemical composition and its effects on different animals on injection. Components include a wide range of proteases, esterases, polyamines, free amino acids, histamine, and specific toxic compounds unique to individual groups or species. Whereas some venoms are primarily cytolytic, causing the destruction of cells and tissues with which they come in contact, others act as neurotoxins or disrupt normal blood functions. For details on the biochemistry and pharmacology of spider venoms, see Bucherl (1971), Bettini (1978), Duchen and Gomez (1984), Geren and Odell (1984), Rash and Hodgson (2002), da Silva et al. (2004), Isbister and White (2004) and Gremski et al. (2014).

Most problems warranting medical attention are not due directly to bites but to infections that are mistaken for spider bites. In other cases involving the more toxic species, reactions can be much more severe, occasionally causing deaths.

See the following sources for information on toxic spiders in different regions of the world: North America (Wong et al., 1987), South America (Lucas, 1988; Bucaretchi et al., 2000), Europe (Maretic and Lebez, 1979), South Africa (Newlands and Atkinson, 1988) and Australia (Southcott, 1976; Sutherland, 1990), Isbister and White (2003), Isbister et al. (2005), Vetter and Isbister (2008), and Vetter (2013). For a review of antivenins, see Isbister et al. (2003) and Pauli et al. (2006). The following accounts address spider problems of particular medical importance.

Tarantism

The term **tarantism** has special significance from a medical viewpoint. It refers to a condition in which individuals allegedly bitten by a "tarantula" spider experience a range of symptoms including tremors, hyperactivity, difficulty breathing, muscular rigidity and priapism (painful penile erection in males), sweating, and uncontrolled crying. In its most extreme form it can lead to fainting spells, delirium, and convulsions. Although descriptions of this syndrome can be traced back as early as Aristotle's writings in the fourth century BC, it was most prevalent in Europe during the Middle Ages. It is believed to have been named after Taranto, Italy, where an epidemic of tarantism occurred in 1370. From there the phenomenon spread throughout Italy to present-day Croatia, Spain, and other parts of the Mediterranean. The only cure was thought to be prolonged and vigorous dancing to special, lively music to induce profuse sweating and eventual collapse from shear exhaustion. Municipalities sometimes hired musicians to play in shifts for 3–4 days at a time as victims danced themselves into frenzies, seeking relief from their affliction. Not uncommonly this led to mass hysteria among local residents and shameless exhibitionism on the part of some individuals. Some have linked this choreomania to Saint Vitus' dance, a nervous disease with involuntary jerking motions.

The bite of the European wolf spider *Lycosa tarentula*, commonly called the "tarantula," traditionally has been

blamed as the cause of tarantism. The reason for this connection is uncertain; in fact this species seldom comes in contact with people. Its bite causes only mild pain and slight swelling at the bite site and none of the neurological effects characteristic of tarantism victims. Convincing evidence suggests that the spider involved was actually a *Latrodectus* species. The tarantellas were danced in the autumn, after the harvest. The European widow spiders live in wheat; harvesters usually pressed wheat against their left forearms during harvest where many envenomations occurred, being an occupational hazard. Autumn was also the time when the pagans celebrated their postharvest bacchanalias. Even as late as the 1950s, spiders of this genus were called *tarantola* in southern Italy, and cases involving their bites were noted in medical records as a tarantola bite or tarantolism. Today tarantism is regarded as largely a psychosomatic response to real or imagined spider bites rooted in legend, ignorance, or superstition linked to cases of latrodectism. Because latrodectism victims tend to move incessantly in response to the pain caused by the neurotoxin, there may be some truth to the notion that dancing helped to ameliorate the effects in envenomation cases (Maretic and Lebez, 1979).

Tarantulas

Tarantulas (Theraphosidae) (Fig. 25.4) are typically ground dwellers living in silk-lined burrows. They often leave their burrows at night to hunt prey; at such times they may enter homes and other shelters or otherwise come in contact with people. During the mating season, males are more likely to be encountered as they wander in search of females; they are particularly aggressive at this time and are easily provoked. Other circumstances that contribute to human encounters are disturbances of their burrows, land development, and flooding or other natural disasters that tend to displace them. About a dozen genera of tarantulas are considered toxic enough to humans to require medical treatment of bite victims (Lucas et al., 1994; Ahmed et al., 2009). They are found primarily in the tropics of South America, Africa, and Australia, where most of the serious cases of human envenomation occur.

Despite their large size, powerful fangs, and intimidating appearance (Fig. 25.5), most tarantulas are not very toxic. Bite reactions vary from almost painless to moderately or intensely painful with reddening about the puncture site. The sensation is commonly likened to that of a bee sting, except that the pain is not immediate and develops more slowly. The pain subsides gradually, seldom persisting for longer than 30 min. This may be accompanied by a burning sensation, localized swelling, and tightening of the muscles near the bite wound.

In cases of more dangerous tarantulas, neurotoxic components in the venom can cause severe, sometimes life-threatening reactions. These toxins are designed to act quickly in subduing vertebrate prey such as frogs, lizards, and birds on which some species feed. When injected into a human bite wound, the venom can cause not only intense pain but also muscle spasms, edema, inflammation of lymphatic vessels, and systemic reactions that can lead to shock and vascular collapse. Other effects reported in laboratory animals include local necrosis, hemoglobin in the urine, and jaundice, indicating the presence of necrotoxic and haemolytic components in the venom of some species.

Members of the genus *Harpactirella* are among those sometimes called **baboon spiders**. They occur in South Africa where they live in silk-lined tunnels under logs, stones, and other debris. They are aggressive hunters, frequently entering homes and animal shelters during their wanderings. The bite of *H. lightfooti* causes an immediate

FIGURE 25.4 Tarantula, *Vitalius sorocabae* (Theraphosidae), Brazil. *Photograph by João P. Burini.*

FIGURE 25.5 Tarantula, ventral view; showing large body size, exposed pair of chelicerae, and extended palps. *Photograph by João P. Burini.*

burning pain followed by paleness, vomiting, and severe systemic reactions that can lead to shock and collapse. The bite is not fatal, with recovery usually occurring within 24 h. None of the tarantulas in the United States are considered to be dangerously venomous. However, the venom of the Texas brown tarantula, *Aphonopelma hentzi*, contains a necrotoxin that has been shown to damage myocardial tissues in mice.

Many tarantulas possess tiny (0.2−1.2 mm long), specialized **urticating hairs** on their abdomen, which are readily detached when stroked with their hindlegs (Figs. 25.6 and 25.7). Only New World theraphosids in the subfamilies Avicullariinae, Ischnocolinae, and Theraphosinae are known to possess such hairs; the latter includes the genera *Brachypelma* and *Aphonopelma*, representatives of which are commonly sold as pets. These hairs are armed with spines and barbs designed to penetrate vertebrate skin and other sensitive tissues (i.e., eyes, nasal cavity) with which they come in contact. Some species have up to 10,000 of these hairs/mm^2, totaling well over 1 million urticating hairs per individual.

FIGURE 25.6 Tarantula (Theraphosidae), with bald patch on dorsal aspect of abdomen where urticating hairs have been defensively flicked off by the hindlegs. *Photograph by Nathan Burkett-Cadena.*

Four basic types of hairs are described by Cooke et al. (1972), although there are additional unusual types in a few other species. Type I hairs enter the skin at a shallow angle, do not penetrate very deeply, and cause only a mild reaction. This is the only type found in species in the United States. Type II hairs are not flicked off but are incorporated into the silk lining the tarantula's retreat, eggs sacs, or silk mats used during molting. Type III hairs penetrate the skin up to 2 mm, causing a persistent urticaria and inflammation that may last for 2−3 weeks; this type is characteristic of many Mexican, Caribbean, Central American, and South American species. Type IV hairs cause inflammation of the respiratory tract in small mammals, although little is known regarding their effects on humans. A given species may have more than one type of urticating hair. Bald patches on the back of the abdomen (Fig. 25.6) are usually evidence that a tarantula has defended itself by dislodging these specialized hairs. They are replaced with each molt, even in adult females that continue to molt after reaching maturity.

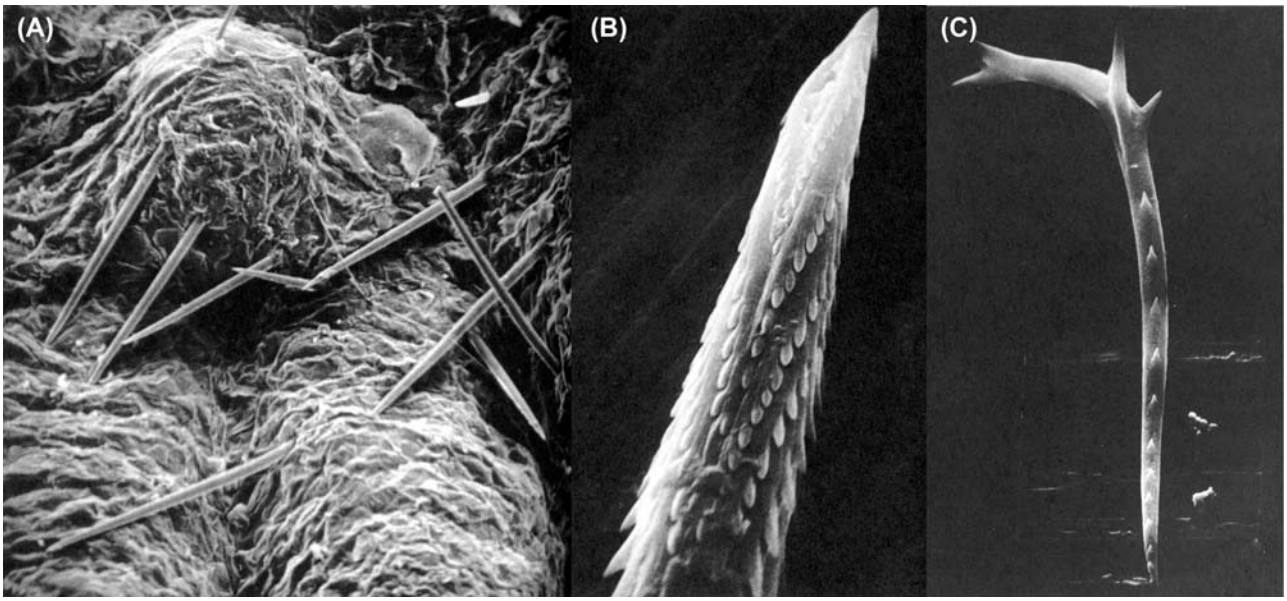


FIGURE 25.7 Urticating hairs of tarantulas (Theraphosidae). (A) Hairs of *Avicularia surinamensis* embedded in skin of young mouse. (B) Basal tip of type II hair of *A. surinamensis*, with backward-directed barbs that help to work the hair into skin. (C) Type IV hair of unidentified tarantula from Chile that causes inflammation of respiratory passages of small mammals. *From Cook et al. (1972); courtesy of the American Museum of Natural History.*

When threatened, the tarantula flicks a cloud of these hairs at the offender, usually rodents and other small mammals that try to attack them in their burrows. In addition to causing irritation to the skin (Fig. 25.7), they can cause severe inflammation of the eyes, mouth, and respiratory passages, serving as an effective deterrent against predators. The effects are solely mechanical and do not involve chemical substances. In the case of certain tarantulas (e.g., *Megaphobema* and *Theraphosa* spp.), the abdominal hairs are incorporated with silk into the egg sacs or the silk mats on which they molt as a defensive barrier to attack by potential predators and parasites.

Humans experience similar reactions to that of other animals when handling or provoking certain tarantulas, including species sold as pets. Common symptoms are urticarial dermatitis, mild edema, and vascular dilation. When the setae come in contact with the eyes, they cause an immediate burning or stinging sensation followed by intense pruritus, lachrymation, swelling of the eyelids, and corneal abrasions. The problem is exacerbated by the natural inclination of the victim to rub the affected areas. Corneal lesions may still be evident 6–9 months after the encounter as the embedded hairs are gradually resorbed. The damage is not permanent, and full visual acuity is gradually restored.

Australian Funnel-Web Spiders

The genera *Atrax* and *Hadronyche* occur only in the eastern half of Australia, predominantly along the coast, including Tasmania, where its members are known as **funnel-web** or **tube-web spiders**. They construct their silken retreats in rock crevices or ground burrows. Six species are considered highly toxic to humans and can cause severe envenomation symptoms (Isbister et al., 2005). The venom of males is more dangerous than that of females in some species.

The **Sydney funnel-web spider,** *A. robustus* (Fig. 25.8), the more commonly encountered species, is largely restricted to areas within a 160-km radius of Sydney (New South Wales). It is a large species with males, the larger of the sexes, measuring about 25 cm in body length. They construct their tubular webs under logs, amid rocks, and in various ground debris. They are often found in suburban gardens where they are attracted to damp, well-watered sites with abundant ground litter. Most bites by this species occur during the summer when roaming males are most likely to enter homes. Wandering males are extremely aggressive, readily attack when provoked, and account for the majority of cases of human envenomation. The bite produces immediate pain and a wide range of neurological symptoms. These include agitation, anxiety, hypertension, generalized muscular twitching, and irregular heartbeat, in addition to pulmonary edema and intravascular coagulation. Deaths have been reported, notably, in children; no deaths have occurred since the development of antivenin. The principal toxic component of the venom has been named **atracotoxin** (Fig. 3.8). It acts by stimulating the release of acetylcholine at motor end plates and throughout the autonomic nervous system. The effects are usually transient and reversible, causing no permanent damage.

Hadronyche formidabilis is called the **North Coast funnel-web spider**, or the tree funnel-web spider. It occurs in the rain forests of southeastern Queensland and northern New South Wales. The effects of its bite are similar to that of *A. robustus*, although the toxicity of its venom is greater. It is less commonly encountered, however, with relatively few bite cases having been reported. An antivenin directed at the male toxins is available for treating both *A. robustus* and *H. formidabilis* bites. Other species of concern are *H. infensa* (Fig. 25.9), *H. versuta*, and *H. cerberea,* along the southeastern coast of Australia.

FIGURE 25.8 Sydney funnel-web spider, *Atrax robustus* (Hexathelidae), Australia. Note the raised chelicerae and threatening posture. *Photograph by Julian White ©.*

FIGURE 25.9 Funnel-web spider, *Hadronyche infensa* (Hexathelidae), male, Australia. *Photograph by Robert Raven.*

South American Wandering Spiders

The genus *Phoneutria* is widely distributed throughout South America, where members are known as wandering spiders, armed spiders, and banana spiders. They are large, aggressive spiders that actively hunt at night, feeding on both invertebrate and vertebrate prey. The venom is highly neurotoxic to humans and acts on both the central and peripheral nervous systems, producing a characteristic syndrome. The most dangerous species are *P. nigriventer* (Fig. 25.10) and *P. keyserlingi,* which occur throughout Brazil, Uruguay, and Argentina, where cases of human envenomation are quite common. It is a large species with females attaining body lengths up to 5 cm. Its venom contains a number of pharmacologically active compounds including histamine and serotonin, in addition, to neurotoxin. Its bite is extremely painful and causes a number of symptoms such as salivation, sweating, muscular spasms, painful penile erection, and visual problems. Deaths are rare and usually are attributed to respiratory failure. In one study of several hundred bite victims, 96% of 10- to 70-year-olds experienced no, or only mild, envenomation effects (Bucaretchi et al., 2000).

Yellow Sac Spiders

Only a few members of the families formerly belonging to the Clubionidae, or **sac spiders** (Fig. 25.11), pose health concerns. Most are *Cheiracanthium* spp. (Eutichuridae), which as a group occur primarily in the Eastern Hemisphere; only two species are known from North America (*C. inclusum* and *C. mildei*). Envenomation usually occurs when they are trapped against the skin after crawling into clothing or footwear, or when an individual rolls over onto one while sleeping. The bite typically causes immediate local pain, redness, and formation of a wheal, similar to the sting of a wasp or bee. In some cases the victim may experience a persistent loss of sensitivity involving the musculature and nerves at the bite site. In more severe cases, systemic responses may include mild fever, nausea, and loss of appetite. No deaths have been attributed to *Cheiracanthium* spiders.

FIGURE 25.10 *Phoneutria nigriventer* (Ctenidae), female on tree trunk, Brazil. *Photograph by João P. Burini.*

FIGURE 25.11 Sac spider, *Clubiona obesa* (Clubionidae), female. *From Gertsch (1979).*

Cheiracanthium punctorium is the species most commonly involved in cases of cheiracanthism in Europe. Its bite causes a painful burning sensation and associated swelling that may persist for several days. Other reported symptoms include chills, general muscular aches, and tenderness of regional lymph glands. Envenomation by *C. japonicum* in Japan is similar to that by *C. punctorium* but also may involve local petechiae (purplish, hemorrhagic spots on the skin), nausea, vomiting, and, rarely, shock. Reactions to one of the two species found in North America, *C. inclusum*, are painful but relatively mild compared with *C. punctorium* and *C. japonicum*. Although *C. mildei* has been circumstantially reported to cause necrosis similar to a recluse spider bite (Spielman and Levi, 1970), studies involving verified bites demonstrated only mild effects and no necrosis (Vetter et al., 2006; McKeown et al., 2014). Several of the *Cheiracanthium* spp. of medical importance are fairly recent introductions from other parts of the world. These include *C. mildei* introduced to the United States from Europe and *C. mordax* introduced to Hawaii and Fiji from Australia.

Hobo Spiders

Some agelenid spiders in the genus *Eratigena* occur in close association with humans. On entering homes, they may become established in basements and other relatively dark, damp locations where they construct sheet webs with

FIGURE 25.12 Hobo spider, *Eratigena agrestis* (Agelenidae), female. *Photograph by Richard S. Vetter.*

FIGURE 25.13 Brown recluse spider, *Loxosceles reclusa* (Sicariidae), female, dorsal view. Note the dark, violin-shaped marking on the cephalothorax and arrangement of the six eyes in three groups of two eyes each. *Photograph by Gary R. Mullen.*

a funnel-like retreat characteristic of the Agelenidae. In Europe, these species are called house spiders.

One species previously implicated as medically important in North America is the **hobo spider**, *Eratigena agrestis* (formerly *Tegenaria agrestis*) (Fig. 25.12), which was inadvertently introduced from Europe to the Pacific Coast of the United States. It was first reported at Seattle in 1930 but did not become common in the Pacific Northwest until the 1960s. The hobo spider is now well established in British Columbia, Washington, and Oregon, with the eastern and southern edge of its distribution extending to Montana, Wyoming, Colorado, and northern Utah, with a stable population in Ontario, Canada. Around human habitation it has been found in basements and cellars, window wells of homes, and crawl spaces; around house foundations, in wood piles, under rocks and wood used in landscaping, and other suitable sites at ground level. The males tend to wander at night in search of females, at which time they enter homes and are more likely to be encountered than females. In Europe, it is considered harmless. It was not until the 1980s that *E. agrestis* was believed to be the cause of bites in the form of necrotic skin lesions in the Pacific Northwest (USA). However, the ability of this species to cause dermal necrosis is now in question and has been seriously challenged (Binford, 2001; Vetter and Isbister, 2004).

Recluse Spiders

The clinical syndrome called loxoscelism is caused by the bite of *Loxosceles* spp. known as **recluse spiders**, **fiddle-back spiders**, and **violin spiders**. It is also called necrotic arachnidism because of cytolytic components in the venom that cause necrosis of tissues around the bite wound. The common names of these spiders refer to a usually distinct fiddle- or violin-shaped marking on the dorsum of the cephalothorax, the neck of which is directed posteriorly. The base of the "violin" encompasses the eyes and is darkly contrasted against the lighter, general body color in several but not all species (Fig. 25.13). The eyes are distinctive among spiders in that there are only six, rather than the usual eight, and that they are arranged in three groups of two eyes each (Fig. 25.13). The combination of a violin-shaped marking and this eye pattern distinguishes *Loxosceles* from all other spider genera. The body color and legs are usually light, tawny brown but may be dark brown or even grayish in some populations. *Loxosceles* spp. quite closely resemble one another, usually requiring a specialist to make species determinations. The legs are relatively long and slender, making them agile spiders that can move quickly. They are primarily nocturnal hunters, either catching prey that comes in contact with their irregular webs or actively wandering from the security of their silken retreats to capture food items. They do not wrap their prey but rely on their potent venom to quickly subdue it.

Approximately 134 species of *Loxosceles* have been described, with about 80% of them being found in the Americas (Gertsch and Ennik, 1983). The other species occur primarily in Europe and Africa. Thirteen *Loxosceles* spp. are found in the United States (Fig. 25.14), including *L. arizonica* (Arizona), *L. blanda* (Texas), *L. deserta* (California, Arizona), *L. devia* (Texas), *L. laeta* (Massachusetts, California), *L. reclusa* (southeastern except Florida, south-central, and midwestern states), and *L. rufescens* (very disjunct, localized sites in many states; often in a single building). *Loxosceles laeta* and *L. rufescens* are non-native species, from South America and the Mediterranean region, respectively. *Loxosceles rufescens* is the most widely distributed member of the genus. It is endemic in southern Europe and northern

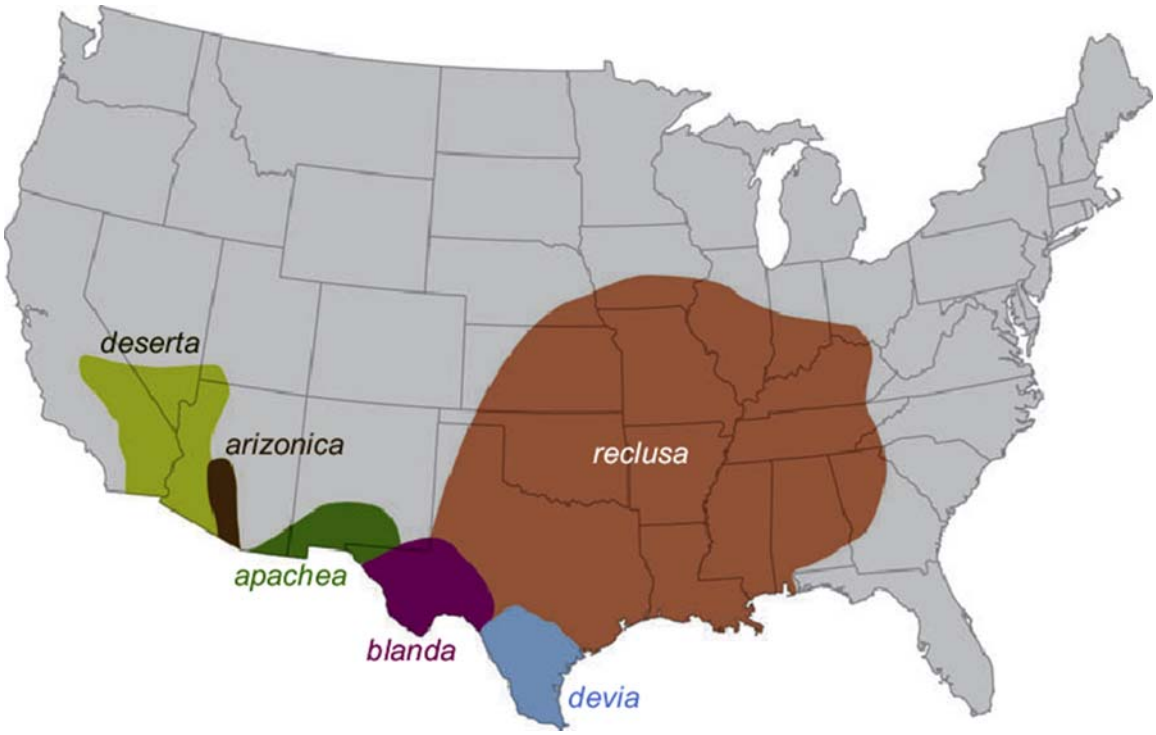


FIGURE 25.14 Approximate distribution of the more common *Loxosceles* species in the continental United States. *Reproduced with permission from the* Annual Review of Entomology, *Volume 53 © 2008 by Annual Reviews.*

Africa, from where it has spread to northern Europe and parts of the Middle East, and it has been introduced to Australia, Japan, Madagascar, and North America. The most important species are *L. reclusa* in North America, *L. laeta* in South America, and *L. rufescens* in the Mediterranean. The latter two species have been introduced into non-native areas through commerce, although their prevalence is very spotty. A review of aspects relating to *Loxosceles* envenomations can be found in da Silva et al. (2004), Vetter (2008, 2013, 2015), and Gremski et al. (2014).

Brown Recluse Spider (Loxosceles reclusa)

The **brown recluse spider** (Fig. 25.15) occurs primarily in the southeastern and central United States. Records outside this area are believed to represent scattered but not well established populations, unlike *L. rufescens*. It is typically found indoors in warm, dry, undisturbed areas such as closets, attics, basements, storage areas, utility rooms, heated garages, lofts of feed mills, storerooms of broiler houses, and heated warehouses. It also is found hiding in cabinets and furniture, behind baseboards, door facings, and wall hangings, and in crevices and corners of rooms. Particularly common sites to find them are in old boxes and accumulations of materials that have not been disturbed for some time.

When mature, *Loxosceles reclusa* females are 7−12 mm in body length but may look much larger because of their long legs. The males are slightly smaller (mean 8 mm), have longer legs than the females, and are easily recognized by their bulbous pedipalps. Mating occurs from February to October but most commonly in June and July. The inseminated female produces 1−5 egg sacs, each containing 20−50 eggs. The egg sacs are white, about 17 mm in diameter, flattened on the underside and convex

FIGURE 25.15 Brown recluse spider, *Loxosceles reclusa* (Sicariidae), female. This spider is typically tawny-brown in color, with relatively long legs. *Photograph by Richard S. Vetter.*

above, and are constructed in the spider's silken retreat. The spiderlings usually emerge in 3–7 weeks and remain in the web with the female until after the first or second molt. Development is relatively slow, requiring 7–8 months under favorable conditions. During this time they molt another 6–7 times, undergoing eight instars before becoming adults. Adults commonly live up to 2.5 years and have been known to survive 5–10 years under laboratory conditions.

The web of *L. reclusa* is constructed in poorly lighted, undisturbed, out-of-the-way places where the spider spends most of its time. It is a rather irregular, nondescript tangle of silken strands, which continues to grow in thickness as new silk is laid down. Freshly deposited silk is mechanically "sticky" like Velcro but soon becomes covered with dust, contributing to the unkempt appearance of the webbing. In addition, to being a retreat, the silk serves to detect the presence of potential prey. When food is scarce, *L. reclusa* will leave the web at night to roam in search of prey. It is under such circumstances that they are most likely to come in contact with humans.

Although being nonaggressive and very retiring as its common name implies, the brown recluse spider will bite if provoked. Despite common misperceptions, 90% of brown recluse bites are of minor incidence and self-healing and involve only inflammation (Tutrone et al., 2005). Severe wounds with massive tissue damage are the cases that tend to be publicized, such that extreme reactions are often misconceived to be typical. Most encounters occur either at night when a person rolls onto a recluse spider in bed or when putting on clothes or footwear into which the spider has crawled. Often the victim is not aware of the bite until 2–3 h later, whereas in other cases it may be immediately felt as a stinging sensation. This is usually followed by intense local pain with the formation of a small blister at the bite site. In a study of North American loxoscelism cases, based on a pain scale of 1–10 (10 being the highest), patients reported an average pain level of 2.4 on the first day postbite; this increased to 6.7 the next day, with several reporting scores of 9 and 10 (Payne et al., 2014).

The area around the bite becomes reddened and swollen as the venom seeps into the surrounding tissues, making it very sensitive to touch. The extent of the skin area involved is usually evident within 6–12 h. The venom is highly cytotoxic, killing any cells it contacts. Within 24 h, the involved skin tissue may turn dusky or purplish in more severe wounds as the blood supply and oxygen to the affected area are cut off. It is a positive sign if the bite area retains red coloration or if a red area turns from white to red after being pressed. In more severe cases, the result is necrosis of tissues and formation of an ulcer (Fig. 25.16) within 7–14 days. Histological evidence indicates acute injury to the blood vessels and

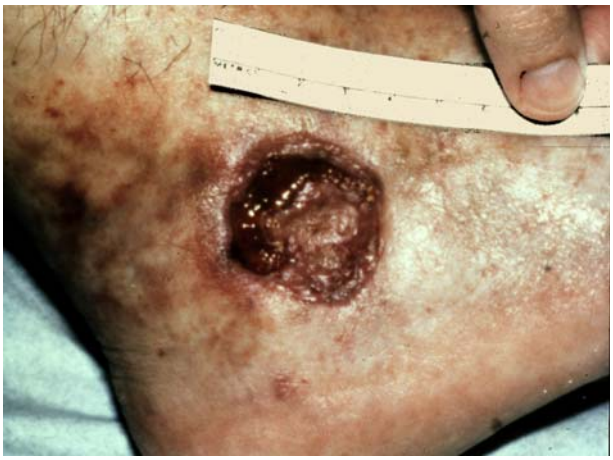


FIGURE 25.16 Slow-healing skin lesion just below ankle caused by bite of the brown recluse spider, *Loxosceles reclusa*; 4 months following envenomation, before the patient underwent skin grafts to repair the tissue damage. *Courtesy of Kevin Humphreys.*

infiltration by white blood cells at the site. Phospholipase, a major component of the venom, induces this white blood cell response while also causing platelets in the blood to aggregate and the liberation of inflammatory substances that contribute to development of the skin lesion.

The extent of tissue damage is largely dependent on the amount of venom injected at the time of the bite. Small doses can elicit very little response such that many, if not most, *L. reclusa* bites go virtually unnoticed or do not result in ulcerations. A high dose, on the other hand, can result in destruction not only of the skin but also the underlying muscles into which the venom seeps due to gravity. The irregular shape of the skin lesion itself also reflects the effect of gravity, most evident in bites on the arms and legs. Some of the more severe cases result when the bite occurs in areas associated with fat tissue. Previously, it was thought that the dermonecrosis was caused solely by the venom enzyme **sphingomyelinase D**. It since has been shown that several isoforms of this enzyme exist; now the group of toxic compounds are collectively referred to as **phospholipases D** (Gremski et al., 2014). The venom components readily destroy lipid cells, causing saponification and extensive damage to the vasculature. This can cause severe tissue damage to the eyelids and face and to "baby fat" of infants.

Healing in cases of severe bites occurs very slowly, often requiring 2–3 months. The edges of the wound become thickened and raised as the central area begins to undergo scar formation. The necrotic tissue gradually sloughs away, often exposing the underlying muscles. As the wound heals from beneath, a black scablike **eschar** develops over the damaged area, protecting it during the healing process (Fig. 25.17). Although the literature

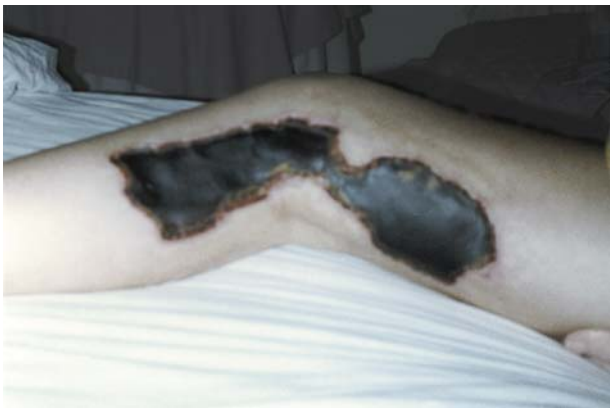


FIGURE 25.17 Severe case of envenomation by brown recluse spider, *Loxoxceles reclusa*, on inner surface of leg of 19-year-old woman. The bite occurred at night while she was sleeping in bed. The large, black eschar denotes extent of tissue damage, as evident 3 months after the bite when picture was taken. *Courtesy of Carolyn Grissom, Shelbyville, TN.*

cautions about secondary infection, Anderson (1998) states that in the 1,000 cases on which he consulted in his career, recluse bites never developed infection. The end result is typically a sunken scar varying in size from about 2 cm up to 10 cm or larger.

A very small percentage of victims of brown recluse spider bites experience systemic reactions, usually within 24−48 h after envenomation. These may include fever, malaise, nausea, vomiting, joint pains, jaundice, yellowing of the eyes, and a generalized pruritic rash. Occasionally the systemic symptoms can be even more serious in the form of hemolytic anemia, intravascular coagulation, and renal failure; the latter is indicated by dark-colored urine, due to the presence of free hemoglobin. The anticoagulant heparin can be administered to reduce the risk of intravascular coagulation. Also, aggressive therapy to counter hemolysis and the use of dialysis in cases of renal failure, may be required. Systemic loxoscelism is predominantly seen in children, with treatment for hemolysis often involving transfusions, hydration, and dialysis. Systemic loxoscelism is of particular concern in children because it can be fatal in 12−30 h, which is prior to expression of dermatologic evidence, making accurate diagnosis difficult.

Treatment of *L. reclusa* bite victims remains controversial. No single approach has been accepted by the medical community, in part because studies never incorporate a control group to determine what happens without treatment, and the fact that recluse bites often heal without medical intervention (Swanson and Vetter, 2005). Treatments that have been used with varying success include cleansing the wound with hydrogen peroxide and applying hyperbaric oxygen and burn creams to prevent secondary infection and promote healing. Corticosteroids injected directly into the lesion have been used, but to be effective they must be done within a few hours following the bite. An antiquated remedy was prompt surgical excision of the affected skin, particularly in severe cases, in an effort to remove the venom before it could do further damage. Excising affected tissues, however, often can result in more damage than simply allowing the affected area to heal without surgical intervention. Excision therefore is recommended only after the wound has stopped enlarging and the healing process can begin. Skin grafting and other forms of reconstructive surgery may be required in severe cases to repair extensive tissue damage that otherwise can lead to permanent, disfiguring scars.

Antivenins are available for treatment of *L. laeta* and other *Loxosceles* spp. in South America; however, despite the development of antivenin for treatment of *L. reclusa* bites in North America, its production has not proved to be commercially feasible.

Many cases of focal skin lesions and necrotic "bite" wounds are often misdiagnosed as brown recluse bites. In some cases, other spiders may be involved; more commonly, however, the cause is unknown and is not spider-related at all. Examples of the latter include secondary infections of arthropod bites and stings (e.g., ticks, fire ants), skin abscesses and ulcerous lesions, and slow-healing wounds of diabetic patients. In recent years, an increasing number of cases of skin infections and soft-tissue injury due to methicillin-resistant *Staphylococcus aureus* (MRSA) is being misdiagnosed as loxoscelism. Even when a spider is involved in a suspected case, all too often the specimen is not recovered for identification; when it is, typically the species is not confirmed by a spider specialist. The situation is further confounded by the tendency of physicians and the general public to blame "bites" of unknown origin on spiders, with no supporting evidence to corroborate it. The result is that the magnitude of misdiagnosed brown recluse bites is difficult to determine and no doubt varies significantly, depending on the geographic area. To date no reliable diagnostic test for recluse envenomation has been developed, although one test has shown promise (Gomez et al., 2002; Stoecker et al., 2006).

In an effort to reduce the number of loxoscelism misdiagnoses, Stoecker et al. (2017) created an acronym of NOT RECLUSE, where each letter represents a dermatological sign that should exclude recluse bite from consideration. For example, the USE in NOT RECLUSE stands for **U**lcerates too early, **S**wollen and **E**xudative. Recluse bites typically ulcerate after 7 days, do not show swelling below the neck or above the feet, and are dry. If a wound ulcerates prior to 7 days, exhibits swelling in an arm, or is oozing pus or blood, these are indications that a diagnosis other than loxoscelism should be considered.

South American Violin Spider (Loxosceles laeta)

Loxosceles laeta is the largest species in the genus and poses a significant health concern in South America. It closely resembles *L. reclusa*, from which it is generally distinguished by its more reddish coloration and the fourth pair of legs of the female being longer than the others. In addition, to its common names South American violin or brown recluse spider, it is called *araña de los rincones*, or the corner spider, because of its occurrence indoors in the corners of rooms. This spider has been introduced to three or four locations in North America and Europe where isolated local populations have become established; e.g., in museums in New England (USA) and Finland. In 1960 an infestation was discovered on the Harvard University campus at Cambridge, Massachusetts (USA), where it was believed to have been present for some 20 years, with no building occupants reporting medical events. Established populations of *L. laeta* have been documented at several locations in southern California (USA), where they have been known to occur since the 1960s. They have not spread much since that time.

Females produce multiple egg sacs, each containing about 50 eggs, which are deposited in a dense, cottony part of the web usually at floor level. The number of egg sacs per female varies significantly and may be as high as 15 under laboratory conditions. In natural settings, females produce an average of three to seven egg sacs following a single mating. Most eggs are produced during the spring and summer (October–January) in South America. The developmental time from egg hatch to adult ranges from as short as 6–8 months to a year or more. The adults are relatively long-lived, with mated and unmated females surviving about 3 and 4 years, respectively. Males live only about half as long as the females. Like other *Loxosceles* spp., both sexes of *L. laeta* are able to survive prolonged periods without food and water, reportedly up to 2 years for some females. *Loxosceles laeta* often produces extensive webbing that is particularly noticeable in corners of rooms and along floor-level runways that they follow at the base of walls. These are composed of multilayers of coarse silk, the amount of which reflects the degree of spider activity and duration of the infestation.

For many years before the cause was determined in 1947, skin lesions resulting from the bite of *L. laeta* in South America were known as **gangrenous spot syndrome**. The bite reaction is similar to that of *L. reclusa*, producing a necrotic lesion that heals slowly. However, it is more often accompanied by systemic effects that can be life-threatening. Such cases are referred to as **viscerocutaneous loxoscelism** in which the lungs, kidneys, liver, and central nervous system may be damaged. The venom causes severe inflammatory, cytotoxic, necrotic, and degenerative changes in tissues, leading to fever, jaundice, blood or hemoglobin in the urine, and sensorial involvement.

Widow Spiders

The term **latrodectism** is a syndrome caused by the bite of any of several *Latrodectus* spp. The venom of these spiders contains potent neurotoxins that cause generalized pain, nausea, vomiting, faintness, dizziness, perspiration, and neuromuscular involvement in the form of muscle weakness, stiffness, cramps, tremors, incoordination, numbness or prickling sensations, paralysis, disturbed speech, and difficulty breathing. The main toxic fraction of the venom of *L. mactans* is a protein called **α-latrotoxin** that acts on the motor-nerve endings at the neuromuscular junctions. It causes depletion of the synaptic vesicles and the selective release of neurotransmitters that cause contraction of voluntary muscles. The autonomic nervous system is also affected. In severe cases the victims typically experience painful abdominal and leg cramps, profuse sweating, lachrymation, and spasms of the jaw muscles that distort the face and cause grimacing. Although symptoms often appear within 10–60 min, the syndrome may take several hours to develop; symptoms usually persist for 20–48 h. A diagnostic sign of latrodectism is sweating at the bite site. This may be accompanied by localized swelling and redness, increased blood pressure, and the development of various types of rashes, either generalized or limited to the bite area. The fatality rate is relatively low (about 5%) even in untreated cases.

Prompt treatment of *Latrodectus* bite victims significantly reduces the severity of symptoms and promotes recovery. **Antivenin** can be very effective as a treatment, especially in cases involving *L. mactans*. Nonetheless, North American physicians, in general, are somewhat reticent to administer antivenin due to possible allergic reaction to the horse-serum base. Considering the efficacy of the antivenin and quickness of pain relief, however, physicians in other parts of the world (e.g., Australia) are more inclined to use it, being vigilant to watch for anaphylaxis. Antiquated remedies include hot baths, tourniquets, and the consumption of whiskey. Calcium gluconate, a commonly recommended muscle relaxant, is now considered ineffective (Clark et al., 1992). Current remedy, other than antivenin, entails the use of opioid and non-opioid analgesics and benzodiazepines, although their utility is still questionable (Vetter and Isbister, 2008). Children are more likely to have serious reactions to *Latrodectus* bites and should receive medical attention as quickly as possible.

Latrodectus taxonomy continues to be subject to many changes, with the number of recognized species differing

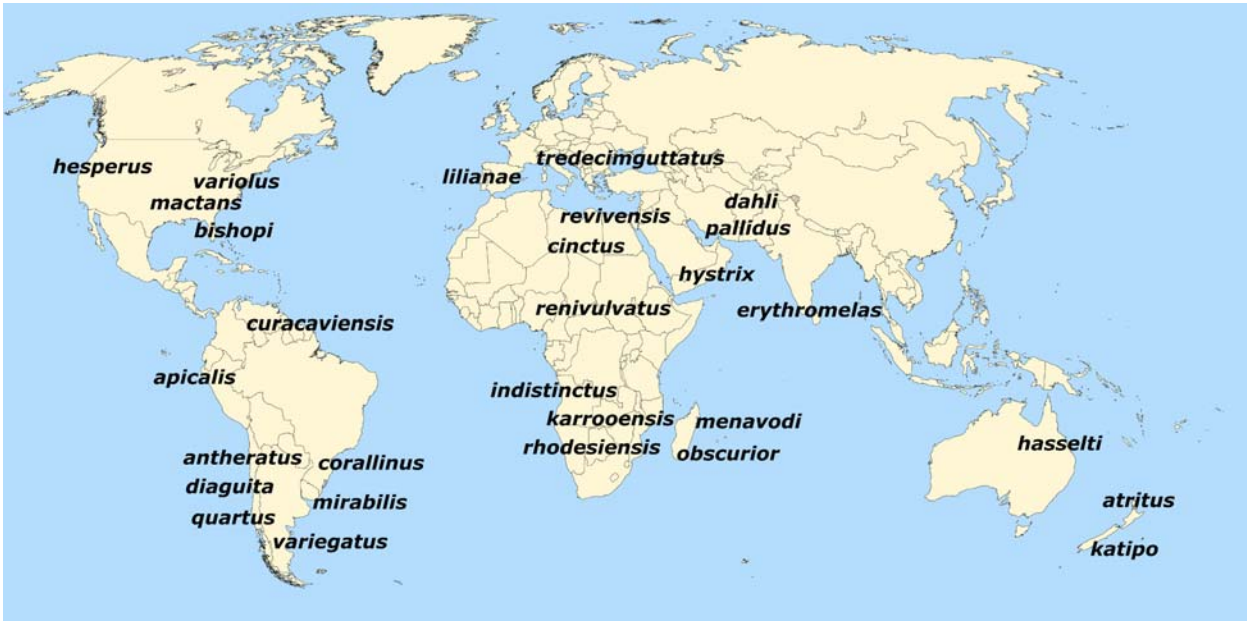


FIGURE 25.18 Generalized distribution of *Latrodectus* species. Not shown is the brown widow (*L. geometricus*), which occurs in multiple locations around the world. *Modified and reproduced with permission, the* Annual Review of Entomology*, Volume 53 © 2008 by Annual Reviews.*

significantly, depending on the author (e.g., 16 species by Levi 1959; compared to 30 species by Garb et al., 2004). This reflects, in large part, differences of opinion as to which taxa represent species versus subspecies. The following currently recognized species, for example, were previously considered to be subspecies of *Latrodectus mactans*: *L. hesperus, L. hasselti*, *L. menadovi*, and *L. tredecimguttatus*. The worldwide distribution of *Latrodectus* spp. is shown in Fig. 25.18.

As a group, *Latrodectus* spp. are medium-sized spiders, seldom more than 1.3 cm in body length, with globose abdomens and relatively long legs. They are generally recognized by their shiny black color and a red or orange hourglass marking on the underside of the abdomen. Some species, however, are drab in appearance or may be colorfully patterned as in the case of *L. bishopi*. They are known by a variety of common names in different parts of the world. In North America they are usually called **black widow spiders** and, less commonly, **hourglass spiders** or **button spiders**. The term "widow" is derived from the misconception that the females invariably devour the male after mating, whereas the term "button" refers to the resemblance of the shiny black, round abdomen of the female to the buttons on old-fashioned shoes. Other common names include shoe-button spiders in South Africa, jockey in Arabia, *karakurt* (black wolf) in Russia, night stinger or *katipo* in Australia and New Zealand, *la malmignatte* in the Mediterranean region (notably Italy and Corsica), *araña capulina*, *chintatlahua,* and *viuda negra* in Mexico, *culrouge* and *veinte-cuatro horas* in the West Indies, *lucacha* in Peru, *mico* in Bolivia, *guina* and *pallu* in Chile, and *araña del lino* (flax spider), *araña del trigo* (wheat spider), and *araña rastrojera* (stubblefield spider) in Argentina and other parts of South America.

Latrodectus spp. are shy, retiring spiders that construct their tangled webs of coarse silk in dark, undisturbed places, usually close to the ground. They are especially common under logs and stones, in abandoned animal burrows, crevices in protected earthen banks, and various materials stacked on the ground. Some species, however, build large, irregular aerial webs in shrubs and other vegetation, often up to a meter or more above the ground. The adults are primarily nocturnal, spending most of the day in the security of their silken retreats in protected recesses adjoining the web. Prey consists primarily of medium-sized to large insects and other arthropods that stumble into the web.

Following mating, the female constructs one or more egg sacs that she suspends in the web. Each sac typically contains 150−300 eggs, depending on the species. The total number of egg sacs produced by a given female varies considerably among species, with up to 10 for *L. mactans* and 20 for *L. hesperus* and *L. geometricus*. The egg sacs are usually spherical or pyriform, white or grayish, with a tough, tightly woven outer covering. The eggs hatch in 14−30 days. The spiderlings undergo their first molt within the sac 3−4 days after hatching, and then emerge via one or more tiny holes which they cut through the silken layers. The spiderlings remain in their mother's web for several weeks before dispersing. They undergo four to nine molts, depending on the species and sex. Males reach maturity in

2−5 months, whereas females require somewhat longer, usually 3.5−8 months.

Spiderlings of *Latrodectus* spp. look quite different from the adult females. Young spiderlings are pale colored with light and dark stripes on the abdomen and legs. They often exhibit patterns of white, yellow, and red bands and spots, which are gradually lost in the females as they mature. The males, however, tend to retain the color pattern of the immatures, including the abdominal markings and leg bands. On reaching sexual maturity, the males leave their webs to wander in search of a female. Mating takes place in the female's web. Contrary to popular belief, females of most *Latrodectus* spp. do not kill and devour the males following insemination any more frequently than do most other spiders. In fact, in the case of *L. bishopi* the adult male and female actually live together in the same web. However, males of the Australian redback (*L. hasselti*) and the brown widow (*L. geometricus*) often do a backflip as the mating act is finishing, placing his abdomen near the female's mouthparts; she may accept or reject his offering depending on her level of hunger.

Five *Latrodectus* spp. occur in North America: *L. bishopi*, *L. geometricus*, *L. hesperus*, *L. mactans*, and *L. variolus*. *Latrodectus mactans* is the most toxic and widespread of these species, causing most of the cases of latrodectism requiring medical attention. *Latrodectus tredecimguttatus* is the most important species in Europe, whereas *L. curacaviensis*, *L. hasselti*, and *L. katipo* are important species in South America, Australia, and New Zealand, respectively.

Southern Black Widow (Latrodectus mactans)

This is the most notorious of widow spiders because of its potent venom, widespread occurrence, and likelihood of coming in contact with humans and domestic animals. It is found throughout eastern North America from southern New England to eastern Mexico, being less common as one moves northward. The adult female typifies the general description of *Latrodectus* in being shiny black with a prominent red hourglass on the underside of the abdomen (Fig. 25.19). It usually has a small red spot just above the spinnerets and occasionally a median series of additional red spots extending anteriorly on the abdominal dorsum. It is found outdoors in various protected places under rocks, logs, boards, and other ground debris, and frequently near buildings. In North America, *L. mactans* also occurs indoors in barns, wood sheds, garages, and various other unheated storage areas, similar to *L. hesperus*. Before the days of indoor plumbing, the widespread use of outdoor privies contributed significantly to the number of human cases of envenomation, especially in males bitten on the genitalia by black widow spiders in webs under the toilet seats (Kirby-Smith, 1942).

FIGURE 25.19 Southern black widow spider, *Latrodectus mactans* (Theridiidae), female, ventral view, showing characteristic red hourglass marking. *Photograph by Richard S. Vetter.*

Although the bite of *L. mactans* may go unnoticed in some cases, it is more commonly felt as a pin prick or an immediate sharp, burning pain with little or no swelling. The pain spreads from the bite site to regional lymph nodes and other parts of the body, usually reaching its maximum intensity in 1−3 h. Thereafter the pain may be continuous or intermittent, lasting up to 48 h. The accompanying muscle spasms and cramps, especially in the abdomen and legs, can lead to tightness of the chest, boardlike rigidity of abdominal muscles, and complete prostration. In extreme cases, complications may occur in the form of shock, leukocytosis, and lesions of the liver, spleen, and kidney evidenced by blood and elevated protein levels in the urine. The death rate can be as high as 4%−5% in untreated cases, the highest of all *Latrodectus* spp.

Northern Black Widow (Latrodectus variolus)

The northern black widow closely resembles *L. mactans* in both size and general appearance. It is usually distinguished, however, by the ventral hourglass being divided into two transverse bands and a row of prominent red spots along the dorsal midline of the abdomen. Its geographic range largely overlaps that of *L. mactans* in North America, occurring widely throughout the eastern United States as far south as western Florida and eastern Texas. It is more common in the northern states, extending into southeastern Canada. Unlike L. *mactans*, it seldom is found in buildings, preferring outdoor situations such as old stumps, piles of dead tree branches, hollow logs, abandoned animal burrows, cavities in rock walls, and under debris. It is common in wooded areas where it constructs a large tangled web in shrubs and tree branches, sometimes as high as 6 m above the ground.

FIGURE 25.20 Western black widow spider, *Latrodectus hesperus* (Theridiidae), female. *Photograph by Richard S. Vetter.*

Western Black Widow (Latrodectus hesperus)

This species also is very similar in appearance to *L. mactans*. The abdominal dorsum is typically all black, only infrequently having red markings. The ventral, red hourglass is well defined and usually complete (Fig. 25.20). It occurs from Oklahoma, Kansas, and central Texas into the western United States, including the high elevations of Colorado and adjacent Mexico. The western black widow utilizes a wider range of habitats than *L. mactans*. It most commonly constructs its web near the ground, in animal burrows, or under various objects. However, it also is found above the ground in shrubs and trees, and in such places as grape arbors and bird nests. It is well adapted to semiarid and arid habitats where it can be found in soil crevices and various desert plants such as agaves and cacti. It is commonly found around human habitation, often in high densities.

Brown Widow (Latrodectus geometricus)

As its common name implies, *L. geometricus* females are brownish rather than black and have a highly variable color pattern, giving it a mottled appearance (Fig. 25.21).

FIGURE 25.21 Brown widow spider, *Latrodectus geometricus* (Theridiidae), female, dorsolateral view. *Photograph by Sturgis McKeever.*

Typically there are three pairs of irregular-shaped spots along the dorsal midline of the abdomen. These vary from simple white spots with black borders to multicolored bull's-eye spots marked with white, yellow, orange, reddish brown, tan, gray, or aqua. The hourglass is generally dull orange, complete, and commonly bordered with yellow. Occasionally individuals are nearly black, more closely resembling other widow spiders.

Although the brown widow is pantropical around the globe (Marie and Vetter 2015; Muslimin et al., 2015), it is thought to have originated in Africa. Introductions to the United States have led to established populations in the Gulf Coast states, Georgia, South Carolina, southern California, and Hawaii. In non-native areas where it has colonized, it has been found in a wide range of domestic settings and is rarely found in native habitat (Baerg, 1954; Vetter et al., 2012). It constructs a relatively small tangled retreat in corners and chinks of brick and cement walls, building foundations and fences, under overhangs of steps, playground equipment, and even in vegetation in gardens and landscaping. In addition, to urban areas, *L. geometricus* is common in South America along ocean beaches where it seems to prefer to construct its web in low, running plants such as the morning-glory *Ipomoea biloba* (Convolvulaceae) above the high tide mark. It is even shyer than other widow spiders, is not aggressive, and rarely has been recorded biting humans. In verified bites involving humans, symptoms are typically limited to pain during fang penetration and a red dermal mark (Müller, 1993). Brown widow bites do not typically result in the severe symptoms commonly associated with latrodectism.

Red Widow (Latrodectus bishopi)

The cephalothorax and legs of the red widow are orange or reddish, contrasted with a black abdomen that has red or

FIGURE 25.22 Red widow spider, *Latrodectus bishopi* (Theridiidae), female, ventral view. This widow spider is unusual in lacking the red hourglass marking; instead the marking is typically reduced to a transverse bar or single, triangular spot. *From Short and Castner (1992); courtesy of University of Florida-IFAS.*

orange spots with yellow borders (Fig. 25.22). The ventral hourglass is pale and usually reduced to a transverse bar or a single triangular spot. The red widow is known to occur only in the sand-pine scrub habitat of peninsular Florida (USA), where it commonly builds its web in saw palmettos (Aracaceae). Little is known about the toxicity of *L. bishopi* venom. Regardless, red widows are of little or no medical concern due to their rarity and lack of interaction with humans.

European Black Widow (Latrodectus tredecimguttatus)

Latrodectus tredecimguttatus is the most common widow spider in Europe. It occurs primarily in the Mediterranean region, extending eastward into Eurasia. In Italy and Corsica it is called *la malmignatte*, and in Russia, *karakurt* or "black wolf." It occurs exclusively outdoors where it constructs its web in a variety of herbaceous and shrubby vegetation, including cultivated crops and arbors. The European black widow is characterized by 13 dorsal red spots and the absence of an hourglass. It poses a significant occupational hazard for farmers and field workers who are commonly bitten while harvesting or threshing wheat, handling hay, picking fruit, or working in vineyards.

Araña del Trigo (Latrodectus curacaviensis)

In South America, *L. curacaviensis* is frequently encountered by humans in buildings, garages, and privies. It also is found in cultivated crops such as wheat, accounting for its common name *araña del trigo*, or wheat spider. It closely resembles *L. mactans*, with which it is easily confused. Like L. *mactans*, its bite can cause serious illness and death.

FIGURE 25.23 Australian red-back spider, *Latrodectus hasselti* (Theridiidae), female, postero-ventral view, showing characteristic red hourglass marking and wide, reddish stripe along dorsal midline of abdomen. *Photograph by Julian White ©.*

Australian Red-Back Spider (Latrodectus hasselti)

This species is black with a prominent red stripe along the dorsal midline of the posterior half of the abdomen (Fig. 25.23), hence the common name "red back." The immatures of other *Latrodectus* spp. (e.g., *L. mactans*) sometimes have a dorsal red stripe, which has led to misidentifications as *L. hasselti*. The leg length of females is 2−3 cm. The Australian red-back spider occurs in sheltered, usually dry sites such as hollows of trees and under logs and rocks. It often builds its retreat and web around building foundations and in ventilator gratings, trash cans, and gas-meter boxes. Although this spider is not aggressive, the female causes a painful bite that can lead to systemic envenomation and fatalities if untreated (about 5%). In typical cases, the initial bite is relatively painless, comparable to a pin prick. Thereafter the pain intensifies from a few minutes to a half hour, often accompanied by localized perspiration and edema, and nausea and vomiting. Localized sweating at the bite site is an important diagnostic sign (Wiener, 1961). In untreated, systemic cases, recovery is usually protracted and may take 3−4 weeks. The severity of red-back spider bites was dramatically reduced in Australia with the introduction of an antivenin against *L. hasselti* in 1956; since that time, no fatalities have been recorded. Sutherland and Trinca (1978) summarized the effects of 2,144 red-back spider bites in Australia.

Katipo Spider (Latrodectus katipo)

This spider is primarily a coastal species found high on ocean beaches and in river beds, under driftwood, and at the base of vegetation. Although found primarily in New Zealand, where it is known as the **New Zealand redback**, *L. katipo* also is said to occur in the Caribbean basin on coastal beaches of Jamaica (Southcott, 1976). It is being displaced from its highly restricted distribution in New Zealand by other theridiid spiders: *Steatoda capensis* and the Australian redback, *L. hasselti.*

VETERINARY IMPORTANCE

Under certain circumstances spiders can pose health threats to household pets, livestock, and other domestic animals. Most cases occur either in stables or in pasture situations where animals are grazing. In the latter situation, high density of a toxic species can cause significant veterinary concerns. Although the evidence of spider bites in nonhuman animals often is circumstantial, enough cases have been documented to show that theridiid and theraphosid spiders are the more common causes of serious spider bites of veterinary importance worldwide.

Most cases of envenomation by spiders in grasslands and pastures are caused by *Latrodectus* spp. Outbreaks involving *L. tredecimguttatus* (published as *L. erebus*) were reported in the steppes of southern Russia in the 1830s. Grazing animals were severely bitten, causing some to stampede due to the pain and to run until they dropped. Fatalities as high as 12% in sheep, 17% in horses, and 33% in camels were reported. Notable outbreaks of latrodectism affecting cattle and agricultural workers also occurred in Spain in the 1830s and 1840s and caused deaths in horses, sheep, and other livestock in Chile in the 1870s. For other examples of latrodectism involving goats, sheep, cattle, and horses in Europe, South Africa, and Indonesia, see Maretic and Lebez (1979). Latrodectism does not seem to present a significant problem for livestock in North America or South America.

Cases of *Latrodectus* envenomation in dogs and cats can be serious, with onset of clinical signs usually during the first 8 h following a bite. Moderate to severe cases typically result in extreme pain, abdominal rigidity without tenderness, hypertension, and, in the case of cats, paralysis. Cats are particularly sensitive to the venom, with a significantly higher mortality rate than in dogs. The primary treatment for cases of *Latrodectus* envenomation in veterinary practices is administration of specific antivenin (Peterson, 2006). Extracts of *Latrodectus* eggs have been shown to elicit significantly different toxicological effects than do widow venom when injected into experimental animals (Buffkin et al., 1971). The egg toxin has been described by Li et al. (2014).

Caution must be used when examining the experimental effects of *Loxosceles* venom in nonhuman mammals. Rabbits, guinea pigs, and humans develop dermonecrotic lesions, whereas rats and mice do not; additionally, in rabbits, the wounds are smaller and heal faster than in humans (da Silva et al., 2004). Also, recluse venom will lyse human and pig red blood cells but not those of dogs, rats, or guinea pigs. The effects of brown recluse spider venom in companion pets were reviewed by Pace and Vetter (2009). When brown recluse venom was injected into rabbits and dogs, rabbits developed typical necrotic lesions, whereas in dogs, the lesions were much smaller and local. The dogs also showed poor feeding, apathy, and dehydration for several days but with full recovery. A lethal dose was not reached until eight spider equivalents were injected. Dogs thus appear to be somewhat resilient to recluse envenomation. Information regarding feline reaction to challenge with recluse venom is lacking.

Envenomation by *Badumna insignis* (formerly *B. robusta*), a member of the family Desidae, has been reported as a problem in stables in Queensland, Australia. Known as the black house spider, common black spider, and window spider, *B. insignis* is nocturnal and has been known to bite horses while they are bedded down for the night. It can inflict a painful bite with associated inflammation and swelling, usually about the head and neck (Southcott, 1976).

Tarantulas have been implicated in several reported cases of envenomation of pastured animals in Central America. *Aphonopelma seemani* and *Sphaerobothria hoffmani*, two theraphosid species that construct deep burrows in pasture soils, are believed to be the cause of necrotic lesions in cattle and horses in Costa Rica (Herrero and Bolanos, 1982). These and other theraphosids in the Americas are suspected as the cause of vesicular lesions on the muzzle, hooves, and udder of cattle, horses, and swine.

PREVENTION AND CONTROL

The best way to avoid being bitten by spiders is not to handle them or allow direct contact while working in areas where they are likely to occur. Gloves can be worn to protect the hands while gardening, potting plants, stacking or handling firewood, moving rocks and other ground materials, or involvement in various other outdoor activities. Spiders can be discouraged from constructing webs around windows and under eaves of buildings by regular removal of their webs with a broom and turning off unnecessary lights at night that attract insect prey. Lumber, trash, garden materials, or other items piled next to buildings should be removed, and cluttered areas in basements, attics, and outbuildings that can serve as protected sites for spiders should be eliminated. Windows and doors should be tightly closed or adequately screened to prevent wandering spiders from entering homes during their activity periods. Regular removal of cobwebs and vacuuming the corners of rooms, window frames and sills, baseboards, and the underside of furniture help to prevent spiders from establishing themselves indoors. The elimination of household insects on which indoor spiders depend for food also helps to reduce infestations.

If necessary, pesticides registered for indoor control of spiders can be used. They are available in the form of sprays, dusts, aerosols, and fogs that help to reach spiders that may escape sweeping and vacuuming by retreating into cracks and crevices. Products with residual activity can be applied to outdoor surfaces such as under eaves, crawl spaces under houses, and around decks and patios.

Widow spiders are seldom found inside homes; they prefer garages and areas around homes. To avoid bites by black widow spiders and related *Latrodectus* spp, precautions should be taken to minimize contact with them in wood piles, under rocks and logs, in outbuildings, and at other sheltered outdoor sites. Avoid putting unprotected hands into recesses at ground level, such as water-meter encasements and accumulations of trash and other debris that can serve as retreats. Special precaution should be

taken when using outdoor privies and portable toilets to avoid contact with webs of black widow spiders on the underside of toilet seats.

In the case of *Loxosceles laeta*, *L. reclusa*, and *L. rufescens*, which occur indoors, appropriate measures can be taken to minimize the risk of being bitten in infested premises. Visually inspect clothes closets, water-heater closets, utility rooms, basements, attics, and other storage areas, killing and removing any specimens that are found. Inspect dry, relatively undisturbed hiding places such as old boxes, wooden cabinets, behind wall hangings, and under beds and other furniture. In one study, when a shop vacuum was used, the violent tumbling through the hose killed all stages of recluse spiders. Shake out clothing that has been hanging unused for some time to avoid encounters with spiders in sleeves and trouser legs. Inspect shoes and other footwear before putting them on, and visually check towels or shake them before use. Where infants or small children are involved, cribs or beds should be pulled away from the wall, and the bed clothes should be kept from reaching the floor. Items in attics and garages should be stored in large zipper-closure bags and well-taped boxes and positioned away from the wall.

Established *Loxosceles* populations can be difficult to eliminate without the use of pesticides to reach individuals that are not accessible to sweeping and vacuum cleaning. Sprays of appropriate materials can be applied in attics, crawl spaces, along baseboards, in corners of rooms, under furniture, behind cabinets, and in other out-of-the-way places to kill the spiders. Treating any webs that are found with pesticide dusts generally helps to reduce infestations more quickly than applying materials to other surfaces. Sticky traps can be used to catch *Loxosceles* spp. and to monitor their activity before or after treatments.

REFERENCES AND FURTHER READING

Ahmed, N., Pinkham, M., & Warrell, D. (2009). Symptom in search of a toxin: Muscle spasms following bites by old world tarantula spiders (*Lampropelma nigerrimum*, *Pterinochilus murinus*, *Poecilotheria regalis*) with review. *Quarterly Journal of Medicine, 102*, 851–857.

Akre, R. D., & Myhre, E. A. (1991). Biology and medical importance of the agressive house spider, *Tegenaria agrestis*, in the Pacific Northwest (Arachnida: Araneae: Agelenidae). *Melanderia, 47*, 1–30.

Anderson, P. C. (1998). Missouri brown recluse spider: A review and update. *Missouri Medicine, 95*, 318–322.

Baerg, W. J. (1954). The brown widow and the black widow spiders in Jamaica (Araneae, Theridiidae). *Annals of the Entomological Society of America, 47*, 52–60.

Barth, F. G. (2001). *A spider's work: Senses and behavior*. Berlin: Springer-Verlag.

Bettini, S. (Ed.). (1978). Arthropod Venoms. *Handbook of experimental pharmacology* (Vol. 48). New York: Springer-Verlag, 977 p.

Bettini, S., & Brignoli, P. M. (1978). Review of the spider families, with notes on the lesser-known poisonous families. In S. Bettini (Ed.), *Arthropod venoms. Handbook of experimental pharmacology* (Vol. 48, pp. 103–120). New York: Springer-Verlag.

Bettini, S., & Maroli, M. (1978). Venoms of Theridiidae, genus *Latrodectus*. In S. Bettini (Ed.), *Arthropod venoms. Handbook of experimental pharmacology* (Vol. 48, pp. 149–184). New York: Springer-Verlag.

Binford, G. J. (2001). An analysis of geographic and intersexual chemical variation in venoms of the spider *Tegenaria agrestis* (Agelenidae). *Toxicon, 39*, 955–968.

Breene, R. G., Dean, D. A., Edwards, G. B., Hebert, B., Levi, H. W., Manning, G., et al. (2003). *Arachnid common names in North America*. http://www.americanarachnology.org/assets/pdfs/arachnid_common_names2003.pdf.

Bucaretchi, F., Deus Reinaldo, C. R., Hyslop, S., Madureira, P. R., De Capitani, E. M., & Vieira, R. J. (2000). A clinico-epidemiological study of bites by spiders of the genus *Phoneutria*. *Revista do Instituto de Medicina Tropical de Sao Paulo, 42*, 17–21.

Bucherl, W. (1971). Spiders. In W. Bucherl, & E. Buckley (Eds.), *Venomous animals and their venoms* (Vol. 3, pp. 197–277). New York: Academic Press.

Clark, R. F., Wethern-Kestner, S., Vance, M. V., & Gerkin, R. (1992). Clinical presentation and treatment of black widow spider envenomation: A review of 163 cases. *Annals of Emergency Medicine, 21*, 782–787.

Cooke, J. A. L., Roth, V., & Miller, F. H. (1972). The urticating hairs of theraphosid spiders. *American Museum Novitates, 2498*, 1–43.

da Silva, P. H., da Silveira, R. B., Appel, M. H., Mangili, O. C., Gremski, W., & Veiga, S. S. (2004). Brown spiders and loxoscelism. *Toxicon, 44*, 693–709.

Duchen, L. W., & Gomez, S. (1984). Pharmacology of spider venoms. In A. T. Tu (Ed.), *Handbook of natural toxins* (Vol. 2, pp. 483–512). New York: Marcel Dekker.

Fink, L. S. (1984). Venom spitting by the green lynx spider, *Peucetia viridans* (Araneae, Oxyopidae). *Journal of Arachnology, 12*, 372–373.

Foelix, R. F. (2011). *Biology of spiders* (3rd ed.). Oxford University Press.

Garb, J. E., Gonzalez, A., & Gillespie, R. G. (2004). The black widow spider genus *Latrodectus* (Araneae: Theridiidae): Phylogeny, biogeography, and invasion history. *Molecular Phylogenetics and Evolution, 31*, 1127–1142.

Garb, J. E., & Hayashi, C. Y. (2013). Molecular evolution of α-latrotoxin, the exceptionally potent vertebrate neurotoxin in black widow spider venom. *Molecular Biology and Evolution, 30*, 999–1014.

Gertsch, W. J., & Ennik, F. (1983). The spider genus *Loxosceles* in North America, Central America and the West Indies (Araneae, Loxoscelidae). *Bulletin of the American Museum of Natural History, 175*, 263–360.

Gomez, H. F., Krywko, D. M., & Stoecker, W. V. (2002). A new assay for the detection of *Loxosceles* species (brown recluse) spider venom. *Annals of Emergency Medicine, 39*, 469–474.

Gorham, J. R., & Rheney, T. B. (1968). Envenomation by the spiders *Chiracanthium inclusum* and *Argiope aurantia*. *Journal American Medical Association, 206*, 1958–1962.

Gray, M. R., & Sutherland, S. K. (1978). Venoms of Dipluridae. In S. Bettini (Ed.), *Arthropod venoms. Handbook of experimental pharmacology* (Vol. 48, pp. 121–148). New York: Springer-Verlag.

Gremski, L. H., Trevisan-Silva, D., Ferrer, V. P., Matsubara, F. H., Meissner, G. O., Wille, A. C. M., et al. (2014). Recent advances in

the understanding of brown spider venoms: From the biology of spiders to the molecular mechanisms of toxins. *Toxicon, 83*, 91−120.

Herrero, M. V., & Bolanos, R. (1982). Life-history and tunnels of 2 'horse-biting' spiders from Costa Rica (Araneae: Theraphosidae). Preliminary observations (in Spanish). *Brenesia, 19/20*, 319−324.

Isbister, G. K., Graudins, A., White, J., & Warrell, D. (2003). Antivenom treatment in arachnidism. *Journal of Toxicology − Clinical Toxicology, 41*, 291−300.

Isbister, G. K., & Gray, M. R. (2002). A prospective study of 750 definite spider bites, with expert spider identification. *Quarterly Journal of Medicine, 95*, 723−731.

Isbister, G. K., & Gray, M. R. (2003a). Effects of envenoming by comb-footed spiders of the genera *Steatoda* and *Achaearanea* (family Theridiidae: Araneae) in Australia. *Journal of Toxicology − Clinical Toxicology, 41*, 809−819.

Isbister, G. K., & Gray, M. R. (2003b). White-tail spider bite: A prospective study of 130 definite bites by *Lampona* species. *Medical Journal of Australia, 179*, 199−202.

Isbister, G. K., Gray, M. R., Balit, C. R., Raven, R. J., Stokes, B. J., Porges, K., et al. (2005). Funnel-web spider bite: A systematic review of recorded clinical cases. *Medical Journal of Australia, 182*, 407−411.

Isbister, G. K., & White, J. (2004). Clinical consequences of spider bites: Recent advances in our understanding. *Toxicon, 43*, 477−492.

Kaston, B. J. (1970). Comparative biology of American black widow spiders. *Transactions of the San Diego Society of Natural History, 16*, 33−82.

Kaston, B. J. (1978). *How to know the spiders* (3rd ed.). Dubuque, IA: William. C. Brown Co., 272 p.

Kirby-Smith, H. T. (1942). Black widow spider bite. *Annals of Surgery, 115*, 249−257.

Levi, H. W. (1959). The spider genus *Latrodectus* (Araneae: Theridiidae). *Transactions of the American Microscopical Society, 78*, 7−43.

Li, J., Yan, Y., Wang, J., Guo, T., Hu, W., Duan, Z., et al. (2014). Purification and partial characterization of a novel neurotoxic protein from eggs of black widow spiders (*Latrodectus tredecimguttatus*). *Journal of Biochemical and Molecular Toxicology, 27*, 337−342.

Lucas, S. (1988). Spiders in Brazil. *Toxicon, 26*, 759−772.

Lucas, S. M., Da Silva, P. I., Bertani, R., & Costa Cardoso, J. L. (1994). Mygalomorph spider bites: A report on 91 cases in the state of São Paulo, Brazil. *Toxicon, 32*, 1211−1215.

Malaque, C. M. S., Santoro, M. L., Cardoso, J. L. C., Conde, M. R., Novaes, C. T. G., Risk, J. Y., et al. (2011). Clinical picture and laboratorial evaluation in human loxoscelism. *Toxicon, 58*, 664−671.

Maretic, A., & Lebez, D. (1979). *Araneism with special reference to Europe*. Belgrade: Nolit Publishing House, 255 p.

Marie, J., & Vetter, R. S. (2015). Establishment of the brown widow spider (Araneae: Theridiidae) and infestation of its egg sacs by a parasitoid, *Philolema latrodecti* (Hymenoptera: Eurytomidae) in French Polynesia and the Cook Islands. *Journal of Medical Entomology, 52*, 1291−1298.

McCrone, J. D., & Levi, H. W. (1964). North American widow spiders of the *Latrodectus curacaviensis* group (Araneae: Theridiidae). *Psyche, 71*, 12−27.

McKeown, N., Vetter, R. S., & Hendricksen, R. G. (2014). Verified spider bites in Oregon (USA) with the intent to assess hobo spider venom toxicity. *Toxicon, 84*, 51−55.

Müller, G. J. (1993). Black and brown widow spider bites in South Africa: A series of 45 cases. *South African Medical Journal, 83*, 399−405.

Muslimin, M., Wilson, J.-J., Ghazali, A.-R. M., Braima, K. A., Jeffrey, J., Wan-nor, F., et al. (2015). First report of brown widow spider sightings in peninsular Malaysia and notes on its global distribution. *Journal of Venomous Animals and Toxins including Tropical Diseases, 21*, 11.

Newlands, G., & Atkinson, P. (1988). Review of southern African spiders of medical importance, with notes on the signs and symptoms of envenomation. *South African Medical Journal, 73*, 235−239.

Nentwig, W., Patini, P., & Vetter, R. S. (2017). Distribution and medical aspects of *Loxosceles rufescens*, one of the most invasive spiders of the World (Araneae: Sicariidae). *Toxicon, 132*, 19−28.

Ori, M. (1984). Biology of and poisoning by spiders. In A. T. Tu (Ed.), *Handbook of natural toxins* (Vol. 2, pp. 397−440). New York: Marcel Dekker.

Pace, L. B., & Vetter, R. S. (2009). Brown recluse spider envenomation: A clinical review for veterinarians. *Journal of Veterinary Emergency and Critical Care, 19*, 329−336.

Pauli, I., Puka, J., Gubert, I. C., & Minozzo, J. C. (2006). The efficacy of antivenom in loxoscelism treatment. *Toxicon, 48*, 123−137.

Payne, K. S., Schilli, S., Meier, K., Rader, R. K., Dyer, J. A., Mold, J. W., et al. (2014). Extreme pain from brown recluse spider bites: Model for cytokine-driven pain. *JAMA Dermatology, 150*, 1205−1208.

Peterson, M. E. (2006). Black widow spider envenomation. *Clinical Techniques in Small Animal Practice, 21*, 187−190.

Rader, R. K., Stoecker, W. V., Malters, J. M., Marr, M. T., & Dyer, J. (2012). Seasonality of brown recluse populations is reflected by numbers of brown recluse envenomations. *Toxicon, 60*, 1−3.

Rash, L. D., & Hodgson, W. C. (2002). Pharmacology and biochemistry of spider venoms. *Toxicon, 40*, 225−254.

Raven, R. J. (1985). The spider infraorder Mygalomorphae (Araneae): Cladistics and systematics. *Bulletin of the American Museum of National History, 182*, 1−180.

Regier, J. C., Shultz, J. W., Zwick, A., Hussey, A., Ball, B., Wetzer, R., et al. (2010). Arthropod relationships revealed by phylogenomic analysis of nuclear protein-coding sequences. *Nature, 463*, 1079−1083.

Russell, F. E. (1970). Bites by the spider *Phidippus formosus*. Case history. *Toxicon, 8*, 193−194.

Ryan, N. M., Buckley, N. A., & Graudins, A. (2017). Treatments for latrodectism—A systematic review on their clinical effectiveness. *Toxins* (Basel)*, 9*, 148.

Sams, H. H., Hearth, S. B., Long, L. L., Wilson, D. C., Sanders, D. H., & King, L. E., Jr. (2001). Nineteen documented cases of *Loxosceles reclusa* envenomation. *Journal of the American Academy of Dermatology, 44*, 603−608.

Schenberg, S., & Pereira Lima, F. A. (1978). Venoms of Ctenidae. In S. Bettini (Ed.), *Arthropod venoms. Handbook of experimental pharmacology* (Vol. 48, pp. 217−246). New York: Springer-Verlag.

Schenone, H., & Suarez, G. (1978). Venoms of Scytodidae, genus *Loxosceles*. In S. Bettini (Ed.), *Arthropod venoms. Handbook of experimental pharmacology* (Vol. 48, pp. 247−275). New York: Springer-Verlag.

Sharma, P. P., Kaluziak, S. T., Perez-Porro, A. R., Gonzalez, V. L., Hormiga, G., Wheeler, W. C., et al. (2014). Phylogenomic interrogation of Arachnida reveals systemic conflicts in phylogenetic signal. *Molecular Biology and Evolution, 31*, 2963−2984.

Short, D. E., & Castner, J. L. (1992). *Venomous spiders of Florida. Leaflet No. SP 104. Institute of food and agricultural sciences*. Gainesville: University of Florida.

Southcott, R. V. (1976). Arachnidism and allied syndromes in the Australian regions. *Records of Adelaide Children's Hospital, 1*, 97–187.

Southcott, R. V. (1984). Diseases and arachnids in the tropics. In W. B. Nutting (Ed.), *Mammalian diseases and arachnids* (Vol. 2, pp. 15–56). Boca Raton, FL: CRC Press.

Spielman, A., & Levi, H. W. (1970). Probable envenomation by *Chiracanthium mildei*; a spider found in houses. *The American Journal of Tropical Medicine and Hygiene, 19*, 729–732.

Stoecker, W. V., Green, J. A., & Gomez, H. F. (2006). Diagnosis of loxoscelism in a child confirmed with an enzyme-linked immunosorbent assay and noninvasive tissue sampling. *Journal of the American Academy of Dermatology, 55*, 888–890.

Stuber, M., & Nentwig, W. (2016). How informative are case studies of spider bites in the medical literature? *Toxicon, 114*, 40–44.

Stoecker, W. V., Vetter, R. S., & Dyer, J. A. (2017). NOT RECLUSE: A mnemonic device to avoid false diagnoses of brown recluse spider bites. *JAMA Dermatology, 153*, 377–388.

Sutherland, S. K. (1990). Treatment of arachnid poisoning in Australia. *Australian Family Physician, 19*(1), 17, 50–55, 57-61, 62.

Sutherland, S. K., & Trinca, J. C. (1978). Survey of 2144 cases of red-back spider bites: Australia and New Zealand, 1963-1976. *Medical Journal of Australia, 2*, 620–623.

Swanson, D. L., & Vetter, R. S. (2005). Bites of brown recluse spiders and suspected necrotic arachnidism. *New England Journal of Medicine, 352*, 700–707.

Tu, A. T. (1984). Handbook of natural toxins. In *Insect poisons, allergens, and other invertebrate venoms* (Vol. 2). New York: Marcel Dekker.

Tutrone, W. D., Green, K. M., Norris, T., & Weinberg, J. M. (2005). Brown recluse spider envenomation: Dermatologic application of hyperbaric oxygen therapy. *Journal of Drugs in Dermatology, 4*, 424–428.

Ubick, D., Paquin, P., Cushing, P. E., & Roth, V. (Eds.). (2017). *Spiders of North America: An identification manual* (2nd ed.). Keene, New Hampshire, USA: American Arachnological Society.

Varl, T., Grenc, D., Kostanjšek, R., & Brvar, M. (2017). Yellow sac spider (*Cheiracanthium punctorium*) bites in Slovenia: case series and review. *Wiener Klinische Wochenschrift, 129*, 630–633.

Vest, D. K. (1987). Necrotic arachnidism in the Northwest United States and its probable relationship to *Tegenaria agrestis* (Walckenaer) spiders. *Toxicon, 25*, 175–184.

Vetter, R. S. (2008). Spiders of the genus *Loxosceles* (Araneae: Sicariidae): A review of biological, medical and psychological aspects regarding envenomations. *Journal of Arachnology, 36*, 150–163.

Vetter, R. S. (2013). Spider envenomation in North America. *Critical Care Nursing Clinics of North America, 25*, 205–223.

Vetter, R. S. (2015). *The brown recluse spider*. Ithaca, NY: Cornell University Press.

Vetter, R. S., Crawford, R. L., & Buckle, D. J. (2014). Spiders (Araneae) found in bananas and other international cargo submitted to North American arachnologists for identification. *Journal of Medical Entomology, 51*, 1136–1143.

Vetter, R. S., & Hillebrecht, S. (2008). On distinguishing two often-misidentified genera (*Cupiennius*, *Phoneutria*) (Araneae: Ctenidae) of large spiders found in Central and South American cargo shipments. *American Entomologist, 54*, 82–87.

Vetter, R. S., & Isbister, G. K. (2004). Do hobo spider bites cause dermonecrotic injuries? *Annals of Emergency Medicine, 44*, 605–607.

Vetter, R. S., & Isbister, G. K. (2006). Verified bites by the woodlouse spider, *Dysdera crocata*. *Toxicon, 47*, 826–829.

Vetter, R. S., & Isbister, G. K. (2008). Medical aspects of spider bites. *Annual Review of Entomology, 53*, 409–429.

Vetter, R. S., Isbister, G. K., Bush, S. P., & Boutin, L. J. (2006). Verified bites by yellow sac spiders, (genus *Cheiracanthium*) in the United States and Australia: Where is the necrosis? *American Journal of Tropical Medicine and Hygiene, 74*, 1043–1048.

Vetter, R. S., Swanson, D. L., Weinstein, S. A., & White, J. (2015). Do spiders vector bacteria during bites? The evidence indicates otherwise. *Toxicon, 93*, 171–174.

Vetter, R. S., Vincent, L. S., Danielsen, D. W. R., Reinker, K. I., Clarke, D. E., Itnyre, A. A., et al. (2012). The prevalence of brown widow and black widow spiders (Araneae: Theridiidae) in urban southern California. *Journal of Medical Entomology, 49*, 947–951.

White, J., Hirst, D., & Hender, E. (1989). 36 cases of bites by spiders, including the white-tailed spider, *Lampona cylindrata*. *Medical Journal of Australia, 150*, 401–403.

Wiener, S. (1961). Red back spider bite in Australia: An analysis of 167 cases. *Medical Journal of Australia, 2*, 44–49.

Wong, R. C., Hughes, S. E., & Voorhees, J. J. (1987). Spider bites. *Archives of Dermatology, 123*, 98–104.

World Spider Catalog. (2017). *Natural history museum Bern*. Online at: http://wsc.nmbe.ch. version 17.5.

Yaman, M., Mete, T., Ozerr, I., Yaman, E., & Beton, O. (2015). Reversible myocarditis and pericarditis after black widow spider bite or Kounis syndrome. *Case Reports in Cardiology*. https://doi.org/10.1155/2015/768089.

This page intentionally left blank

Chapter 26

Mites (Acari)

Gary R. Mullen[1] and Barry M. OConnor[2]

[1]*Department of Entomology and Plant Pathology, Auburn University, Auburn, AL, United States;* [2]*Museum of Zoology, University of Michigan, Ann Arbor, MI, United States*

More than 250 species of mites are recognized as the cause of health-related problems for humans and domestic animals. Types of problems include (1) temporary irritation of the skin caused by bites or feeding on host skin, fur, and feathers; (2) persistent dermatitis in response to mites invading the skin or hair follicles; (3) mite-induced allergies; (4) transmission of pathogenic microbial agents and metazoan parasites; (5) serving as intermediate hosts of parasites, notably tapeworms; (6) invasion of respiratory passages, ear canals, and occasionally internal organs; (7) an abnormal fear of mites, or **acarophobia**; and (8) **delusory acariosis**, a psychological condition in which individuals are convinced that they are being attacked by mites, when in fact no mites are involved. The general term for infestations of animals by mites is called **acarinism**, whereas any disease condition caused by mites is **acariasis** (acarinosis).

For an introduction to mites in general, see Krantz and Walter (2009), Woolley (1988), or Evans (1992). For major works dealing specifically with taxa of medical–veterinary importance, the following sources are suggested: Hirst (1922), Baker et al. (1956), Strandtmann and Wharton (1958), Sweatman (1971), Yunker (1973), Nutting (1984), and Baker (1999).

TAXONOMY

Based on the classification schemes described by Zhang (2011) and Krantz and Walter (2009), mites or Acari are classified into two major groups, the superorder Parasitiformes (Anactinotrichida) and the superorder Acariformes (Actinotrichida). These are further subdivided into six orders (Table 26.1). The use of alternative names for these orders and the designation of the orders as suborders by various authors cause understandable confusion for those who are unfamiliar with mite classification. For discussions of the higher classification of mites, see van der Hammen (1972), Krantz (1978), Kethley (1982), OConnor (1984), Evans (1992), and Krantz and Walter (2009).

Members of the orders Ixodida, Mesostigmata, Trombidiformes, and Sarcoptiformes are the cause of animal health problems. Most of them are represented by the 50 families that are listed in Table 26.2 and which are covered in this chapter. Not included in the list are the many families of ectoparasitic mites found on wild mammals, birds, and reptiles, most of which cause little or no significant harm to their hosts. Given the number and diversity of taxa involved, there is no single source to which one can turn for identification of mites of medical–veterinary importance. Reliable species determinations usually require the preparation of slide-mounted specimens for microscopic examination and the assistance of an acarologist who has access to the appropriate taxonomic literature. A reference that the nonspecialist can use to identify some of the more common mite pests of public health and veterinary importance

TABLE 26.1 Higher Classification of Mites as Presented in Zhang (2011)

Class Arachnida	
Subclass Acari	
Superorder Parasitiformes (Anactinotrichida)	
Orders	Opilioacarida (Notostigmata)
	Holothyrida (Tetrastigmata)
	Ixodida (Metastigmata)
	Mesostigmata (Gamasida)
Superorder Acariformes (Actinotrichida)	
Orders	Trombidiformes (including Prostigmata, Actinedida, and Tarsonemida)
	Sarcoptiformes (including Oribatida, Astigmata, and Endeostigmata)

Alternative names for the respective orders are shown in parentheses.

Medical and Veterinary Entomology. https://doi.org/10.1016/B978-0-12-814043-7.00026-1

TABLE 26.2 Orders and Families of Mites That Include Species of Medical–Veterinary Importance or Interest

- Order Ixodida
  - Argasidae
  - Ixodidae
- Order Mesostigmata
  - Melicharidae
  - Dermanyssidae
  - Entonyssidae
  - Halarachnidae
  - Laelapidae
  - Macronyssidae
  - Rhinonyssidae
  - Spinturnicidae
- Order Trombidiformes
  - Infraorder Anystina
    - Trombiculidae
  - Infraorder Eleutherengona
    - Cheyletidae
    - Demodicidae
    - Harpirhynchidae
    - Myobiidae
    - Pyemotidae
    - Psorergatidae
    - Syringophilidae
    - Tarsonemidae
  - Infraorder Eupodina
    - Ereynetidae
- Order Sarcoptiformes
  - Hyporder Astigmata
    - Acaridae
    - Analgidae
    - Atopomelidae
    - Carpoglyphidae
    - Cytoditidae
    - Dermationidae
    - Dermoglyphidae
    - Echimyopodidae
    - Epidermoptidae
    - Gastronyssidae
    - Glycyphagidae
    - Hypoderatidae
    - Knemidokoptidae[a]
    - Lemurnyssidae
    - Listrophoridae
    - Lamninosioptidae
    - Myocoptidae
    - Pneumocoptidae
    - Proctophyllodidae
    - Psoroptidae
    - Pterolichidae
    - Pyroglyphidae
    - Rhyncoptidae
    - Sarcoptidae
    - Syringobiidae
    - Turbinoptidae
  - Hyporder Brachypylina (oribatids)
    - Ceratozetidae
    - Galumnidae
    - Oribatulidae
    - Scheloribatidae

Families are listed alphabetically under each order.
[a]*Knemidokoptidae included within Epidermoptidae by some authors (Mironov et al., 2005).*

is the *CDC Pictorial Keys: Arthropods, Reptiles, Birds, and Mammals of Public Health Significance* (Pratt and Stojanovich, 1969).

MORPHOLOGY

The basic body plan of mites is shown in Fig. 26.1. The body is divided into two major regions, the anterior **gnathosoma**, bearing the pedipalps and chelicerae, and the **idiosoma**, the remainder of the body bearing the legs and eyes (when present). The **pedipalps** are typically five-segmented but may be greatly reduced and highly modified in different groups of mites. The pedipalps are primarily sensory appendages equipped with chemical and tactile sensors that assist mites in finding food and perceiving environmental cues. In some groups, they may be modified as raptorial structures for capturing prey or as attachment devices to facilitate clinging to hosts. The mouthparts consist primarily of a pair of **chelicerae**, each of which is typically three-segmented and terminates in a **chela**, or pincer. The chela is composed of a fixed digit and a movable digit designed for seizing or grasping. In the case of certain parasitic mites, the chelicerae are highly modified as long, slender structures for piercing skin to feed on blood and other host tissues. In some groups, structures associated with the mouthparts may be modified as attachment devices to secure them to their hosts (e.g., chiggers).

The idiosoma can be divided into several regions. The anterior part bearing the legs is the **podosoma**. The posterior section behind the legs is the **opisthosoma**. Other regions include the **propodosoma**, the portion of the idiosoma bearing the first and second pairs of legs, and the **hysterosoma**, extending from just behind the second pair of legs to the posterior end of the body. The designation of these body regions is helpful to morphologists and taxonomists in locating specific setae and other structures. The size and arrangement of sclerotized plates, the chaetotaxy, and the nature and location of sensory structures on the idiosoma serve as important taxonomic characters.

Mites typically have four pairs of legs as nymphs and adults but only three pairs as larvae. The legs are divided into the following segments: coxa, trochanter, femur, genu, tibia, tarsus, and pretarsus. The pretarsus commonly bears a pair of claws, a single median empodium, and in certain groups a membranous pulvillus. These structures are highly variable among different groups of mites and aid in movement or clinging to various surfaces, including hosts.

The respiratory systems of mites often include tracheal ducts that supplement the exchange of oxygen, carbon dioxide, and other gases across the body surface. The presence of spiracular openings (stigmata) associated with the tracheal ducts and their location on the body provide important taxonomic characters for recognizing the acarine suborders. In the Prostigmata, for example, the stigmata are typically located on the gnathosoma between the chelicerae. In the Mesostigmata, they are usually located dorsolaterally to the third or fourth pairs of legs, whereas in the Oribatida (Cryptostigmata), they are typically hidden, opening ventrolaterally near the bases of the second and third pairs of legs. Tracheal systems and spiracular openings are lacking in the Astigmata.

Reproductive structures are diverse among mites, providing important characters for distinguishing the sexes and identifying taxa. Sperm transfer may be direct (e.g., insemination by transfer of sperm via the male aedeagus to the sperm storage organ, or spermatheca, of the female), or indirect (e.g., transfer of sperm via the male chelicerae to the female genital opening).

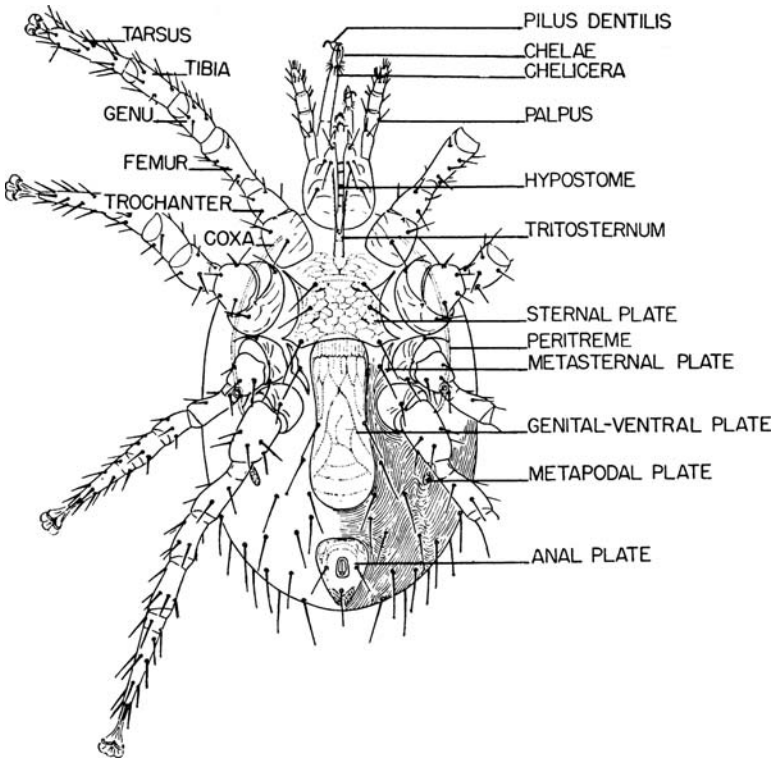


FIGURE 26.1 Generalized morphology of adult female mite (Mesostigmata, Laelapidae, *Androlaelaps fahrenholzi*), ventral view. *From Baker et al., 1956.*

A few points should be mentioned regarding the internal morphology of mites that are pertinent to species of medical—veterinary importance. The digestive system handles primarily liquefied food that has been preorally digested by enzymes secreted in the saliva. The paired salivary glands are typically located in the anterior portion of the idiosoma and open via ducts into the mouth region of the gnathosoma. In addition to digestive enzymes, these glands secrete anticoagulants in hematophagous mites. In certain groups (e.g., chiggers), they may produce cementing substances to anchor the mouthparts in host skin. Also important in certain acarine groups are **coxal glands**. They are derived from excretory structures and serve primarily in osmoregulation. Waste products, in the form of guanine, are excreted by one or two pairs of long, slender **Malpighian tubules** that open into the alimentary tract just anterior to the hindgut.

LIFE HISTORY

The basic developmental stages in the life history of mites are the egg, prelarva, larva, **protonymph**, **deutonymph**, **tritonymph**, and adult. Depending on the taxonomic group, one or more stages may be suppressed, resulting in a wide range of life-history patterns (e.g., chiggers) (Fig. 26.2). Eggs may be deposited externally or retained in the uterus until hatching. The prelarva is a nonfeeding, quiescent stage that may or may not have legs, mouthparts, or other distinct external features. The larva is typically an active form that molts to produce a nymph. The nymphs usually resemble the adults of a given taxon except for their smaller size, pattern of sclerotization, and pattern of setae. The deutonymph of certain astigmatid mites is noteworthy in that it is highly modified morphologically as a nonfeeding stage adapted for surviving adverse environmental conditions. Such deutonymphs are called **hypopi** (singular, hypopus) or hypopodes; the latter terms, however, are no longer commonly used by acarologists. They often have specialized clasping structures such as anal or ventral suckers (Fig. 26.3) that enable them to adhere to phoretic hosts, which aid in carrying them to more favorable sites where they can continue their development. Certain of these deutonymphs may become parasites in hair follicles or subcutaneous tissues of mammals and birds.

The developmental times from egg to adult and the number of generations per year are too variable to make meaningful generalizations. It is therefore important to understand the developmental biology and life-history patterns of individual groups and species. For further information on the development and life history of mites, see Krantz and Walter (2009), Woolley (1988), Schuster and Murphry (1991), and Houck (1994).

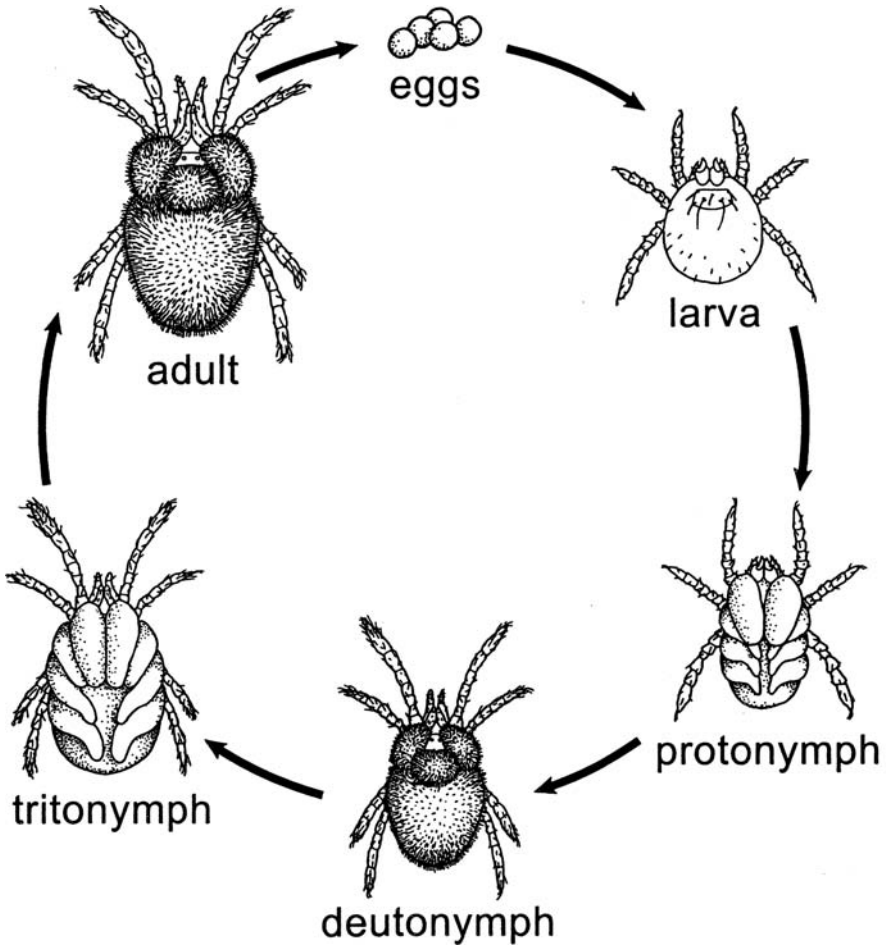


FIGURE 26.2 Developmental stages in life cycle of chiggers (Trombiculidae, Leeuwenhoekiidae, and Walchiidae). The protonymph and tritonymph are inactive stages passed within the cuticle of the engorged larva and deutonymph, respectively. *Original by Rebecca L. Nims.*

BEHAVIOR AND ECOLOGY

Because of the diversity of behavioral and ecological aspects of mites, no attempt is made to discuss them here. Instead they are addressed, where relevant, in the accounts of individual groups and species of mites that follow. For an overview of feeding, mating and reproduction, oviposition, and dispersal behavior of mites, see Woolley (1988) and Evans (1992).

PUBLIC HEALTH IMPORTANCE

Mites can adversely affect human health in many ways. They can infest homes, including carpets, mattresses and bedding, clothing, stored food products, and household pets. Usually they remain unnoticed unless individuals in the household become sensitized and develop various allergies upon subsequent exposure to these mites. Other mites that normally parasitize nonhuman hosts can cause dermatitis in humans when they bite the skin in efforts to feed on blood or other tissues. Most commonly involved in such cases are mite associates of rodents and birds that infest the premises. Such problems typically occur when the natural hosts have departed or died, forcing the mites to seek an alternative food source. A similar situation occurs outdoors when the parasitic larval stage of trombiculid mites, known as chiggers or red bugs, attempt to feed on human skin. Humans are not their normal hosts and often experience intense local skin reactions where these mites attach.

Mites also can pose occupational hazards for farmers, field hands, mill workers, warehouse operators, and others who handle mite-infested materials such as straw, hay, and grains. The mites involved normally feed on fungi, plant materials, or various arthropods; however, upon contact with humans, they can pierce the skin, sometimes causing severe dermatitis. Other mites actually invade human skin, either burrowing through cutaneous tissues (e.g., scabies mites) or infesting the hair follicles and associated dermal glands (follicle mites). Infestations

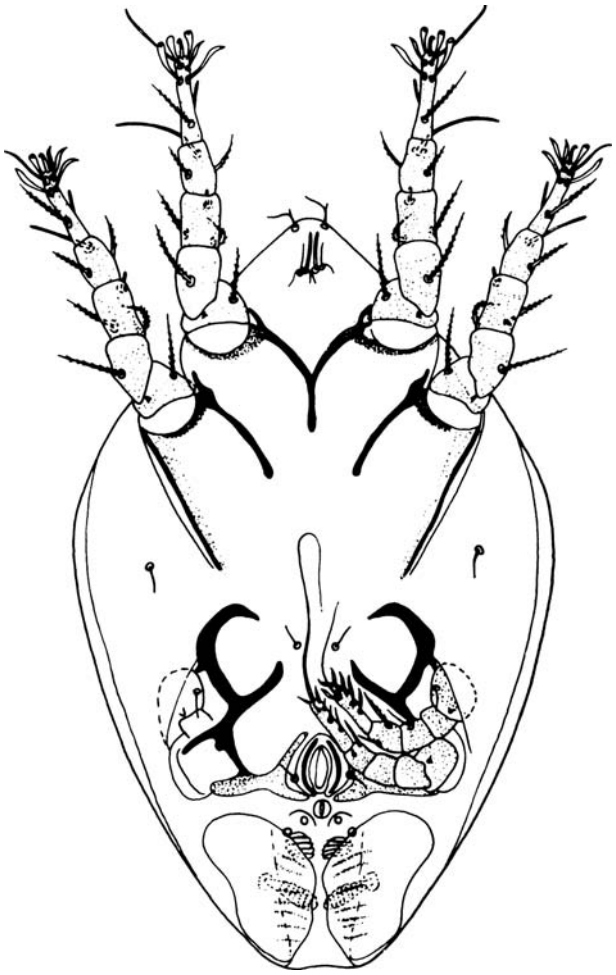


FIGURE 26.3 Deutonymph of *Glycyphagus hypudaei* (Glycyphagidae), ventral view. This is a specialized, typically phoretic, nonfeeding stage that lacks functional mouthparts. *From Whitaker, 1982; original Fain, 1969.*

of these mites can cause persistent, sometimes severe, forms of dermatitis.

In addition to the temporary discomfort or annoyance that mites can cause, some mites are responsible for more serious or chronic medical problems. A number of species may be inhaled or ingested, causing infestations of the respiratory tract and digestive system. Mites even are reported to have been recovered from bile of patients with chronic cholecystitis (inflammation of the gallbladder) and occasionally from the urinogenital tract.

The most widely recognized mite problems affecting human health are respiratory allergies caused by mites infesting house dust. In sensitized individuals, this can lead to chronic respiratory stress, bronchitis, and asthma. However, few human diseases involve pathogens transmitted by mites. The most important is tsutsugamushi disease (scrub typhus or chigger-borne rickettsiosis), which occurs primarily in southeastern Asia, Australia, and the Pacific Islands. The only significant mite-borne disease in the New World is rickettsialpox, reported in the northeastern United States.

For convenience of discussion, problems of a public health nature caused by mites can be grouped into the following categories: mite-induced dermatitis, respiratory allergies, storage mite allergies, internal acariasis, mite-borne human diseases, and acarophobia or delusory parasitosis. The mites involved represent at least 18 families in the three suborders Mesostigmata, Prostigmata, and Astigmata.

Mite-Induced Dermatitis

Species in approximately 14 families of mites are known to cause dermatitis in humans. In many cases, these represent encounters with species that infest stored products. In other cases, they are ectoparasites of other animals, notably rodents, nesting birds, and poultry. Species in only two families (Demodicidae and Sarcoptidae) use humans as their normal hosts. Skin reactions to the feeding or burrowing of these mites range from minor, localized irritation at individual bite sites to severe dermal responses in individuals who become sensitized to specific mite antigens. Still others are free-living, predatory mites that may bite upon contact with human skin.

Melicharidae

The only melicharid species thought to be involved in a human case of dermatitis is *Proctolaelaps pygmaeus,* reported in New Zealand by Andrews and Ramsay (1982). This species was formerly included in the family Ascidae, is probably cosmopolitan, and represents an incidental case. Its bites can cause red papular lesions where the mite pierces the skin.

Dermanyssidae

Members of both recognized genera of dermanyssid mites have been reported biting humans: *Dermanyssus* and *Liponyssoides*. They are ectoparasites primarily on wild and domestic birds and rodents. These mites feed on blood of their hosts by piercing the skin with their long, slender, extrusible chelicerae with highly reduced chelae at their tips. Dermanyssids spend most of their time in the nests of their hosts, crawling onto the animals primarily to feed. When they come in contact with human skin, they are prone to bite, typically causing erythematous papules at each puncture site, often accompanied by intense itching.

Chicken Mite (Dermanyssus gallinae)

Also called the **red poultry mite**, this cosmopolitan species (Fig. 26.4) is the most common dermanyssid mite that bites

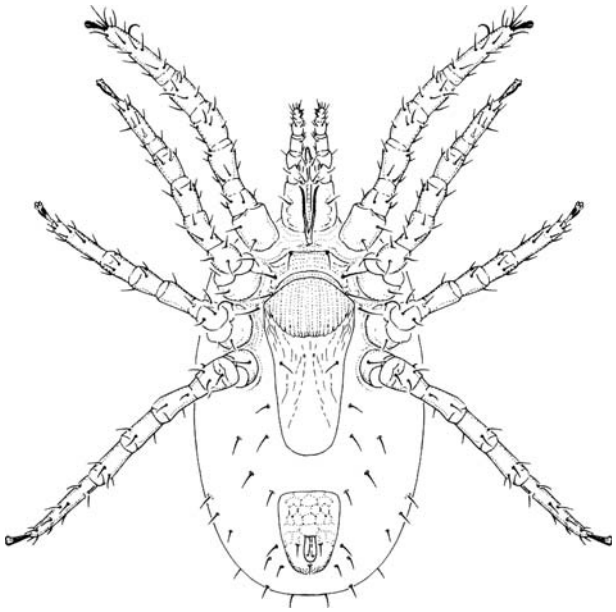


FIGURE 26.4 Chicken mite, *Dermanyssus gallinae* (Dermanyssidae), female, ventral view. *Modified from Gorham, 1991; courtesy of the US Department of Agriculture.*

people. It parasitizes a broad range of hosts. This mite is especially a problem in the Palearctic region and in the United States, where most cases occur in poultry houses or around buildings where pigeons, house sparrows, or starlings are nesting. The mites live in nesting materials, where they spend most of their time, moving onto the birds to feed on blood at night. Consequently, workers in poultry operations seldom experience a biting problem while working during the daytime, even when the houses are heavily infested. However, individuals who enter infested buildings at night may be readily bitten. Occasionally, pet canaries and parakeets also serve as sources of human infestations.

The term **pigeon mite** refers to *D. gallinae* when it infests pigeons or rock doves. The mites frequently enter buildings from pigeon roosts or nests. This tends to happen in the late spring and early summer months, when the young pigeons fledge and the nests are abandoned, forcing the mites to seek alternative hosts. Although most human bites occur at night, bites may occur during the daytime when buildings are darkened.

A number of cases have been reported in hospitals and other institutional settings, where employees and patients have been bitten by *D. gallinae*. The sources of the problem generally can be traced to nesting birds, notably pigeons, on windowsills and ledges, under eaves, and in air-intake ducts or air-conditioners mounted on the outside walls. The mites enter rooms around windows and doors, through crevices and cracks, or via ventilation ducts and air-conditioning systems. In other situations, they may drop onto individuals from roosting or nesting birds in ceilings, or from overhead sites on porches and walkways near buildings. In such cases, close inspection may reveal mites crawling on clothing, furniture, or bed linens, particularly at night when the mites are active.

Human infestations with *D. gallinae* have been variously called chicken tick rash, bird mite disease, psora dermanyssica, pseudogale, and gamasoidosis. The term "fowl mite dermatitis" is likewise used, but it also can be applied to skin reactions caused by other avian mites that attack people.

Most bites tend to occur on the arms and chest protected by clothing, rather than on exposed skin such as the hands and face. Only in exceptional cases do bites occur in the axillary and pubic areas. The bites are usually painful and typically result in red maculopapular skin lesions on the upper portions of the body and extremities. During the bite, close examination will reveal the mite to be a tiny red speck at the center of the papule. Occasionally the bites produce vesicles, urticarial plaques, and diffuse erythema, with dermatographia frequently seen. In multiple-bite cases, a pruritic rash may develop and persist until the source of the infestation has been eliminated. Itching tends to be most intense at night. The problem is usually resolved by treatment with antihistamines or topically applied steroids, combined with moving individuals from affected areas.

St. Louis encephalitis, eastern equine encephalitis, and western equine encephalitis (WEE) viruses have been isolated from *D. gallinae* infesting wild birds. However, conflicting evidence has been reported regarding the ability of *D. gallinae* to transmit any of these viruses among birds or to humans.

American Bird Mite (Dermanyssus americanus)

This mite is very closely related to *D. gallinae* but has been reported biting humans only rarely. It can cause acute, generalized, eczematous dermatitis that is easily misdiagnosed as other skin disorders unless the presence of mites is confirmed. WEE virus has been isolated from this mite infesting nests of the house sparrow, but its significance in transmission or maintenance of WEE virus is unknown.

Dermanyssus hirundinis

This hematophagous mite is a common ectoparasite of certain birds, especially swallows (*Hirundo* spp.) and the house wren (*Troglodytes aedon*) in North America and Europe. It is not unusual for hundreds or thousands of these mites to infest individual nestling birds. At least one case has been documented in Europe of *D. hirudinis* biting

a human and causing urticarial dermatitis (Dietrich and Horstmann, 1983).

House Mouse Mite (Liponyssoides sanguineus)

This mite (Fig. 26.5), referred to in the earlier literature as *Allodermanyssus sanguineus*, is an ectoparasite of domestic and wild rodents. It commonly parasitizes mice, including the house mouse (*Mus musculus*) in the United States and spiny mice (*Acomys* spp.) in North Africa. It occurs less commonly on rats (*Rattus* spp.), voles (*Microtus* spp.), and other rodents in localized areas of eastern North America, Europe, Asia, and Africa. It is primarily interesting to medical entomologists because of its role as the vector of *Rickettsia akari*, the etiologic agent of rickettsialpox in humans.

Like most other dermanyssid mites, the house mouse mite lives in nesting materials, where it spends most of its time crawling onto host animals to feed. Its life cycle and behavior are similar in many respects to *D. gallinae*. Females oviposit in rodent nests or along rodent runways 2–5 days after feeding on host blood. The eggs hatch in 4–5 days to produce larvae that do not feed but instead molt to protonymphs about 3 days later. The protonymphal stage lasts 4–5 days, during which time the mite takes a blood meal, usually engorging in less than an hour, and then molts to the deutonymph. The deutonymph lives about 6–10 days and requires a blood meal before transforming into the adult. The developmental time from egg to adult normally takes 2–3 weeks. After feeding, blood-engorged females leave the rodent host and can be found in the nests and runways, along the walls of infested premises, and especially in warmer areas of buildings such as furnace and incinerator rooms.

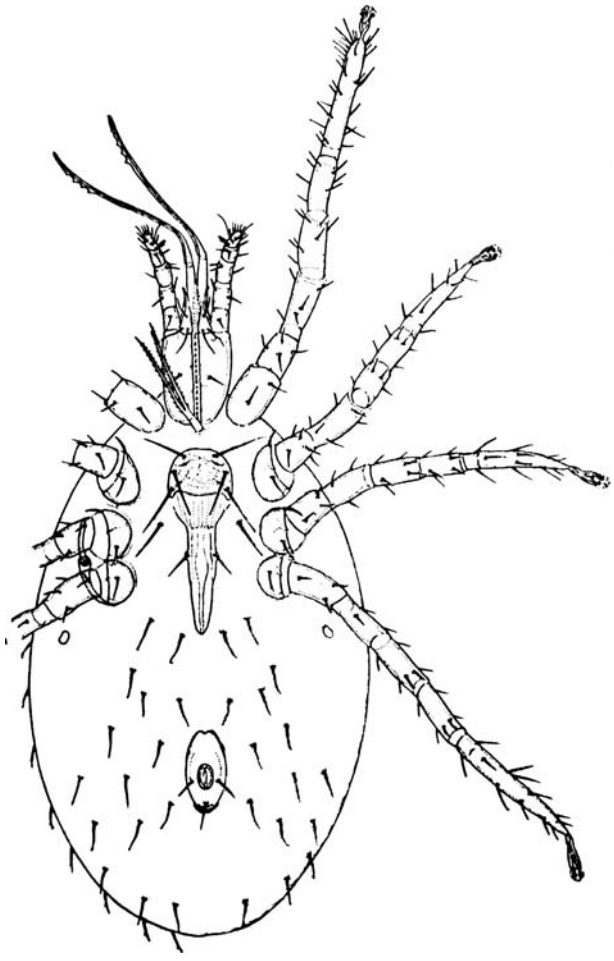


FIGURE 26.5 House mouse mite, *Liponyssoides sanguineus* (Dermanyssidae), female, ventral view. Note the pair of long, attenuated, extruded chelicerae with serrated tips for piercing skin to feed on blood. *Modified from Baker et al., 1956.*

Macronyssidae

Macronyssid mites are blood-feeding ectoparasites on reptiles, birds, and mammals. Five species account for most cases of medical interest. Three of these are *Ornithonyssus* species infesting birds or rodents; *Chiroptonyssus* is parasitic on bats whereas *Ophionyssus* is parasitic on snakes.

Tropical Rat Mite (Ornithonyssus bacoti)

This cosmopolitan mite (Fig. 26.6) is a parasite of rats, particularly the black rat (*Rattus rattus*), and other rodents in

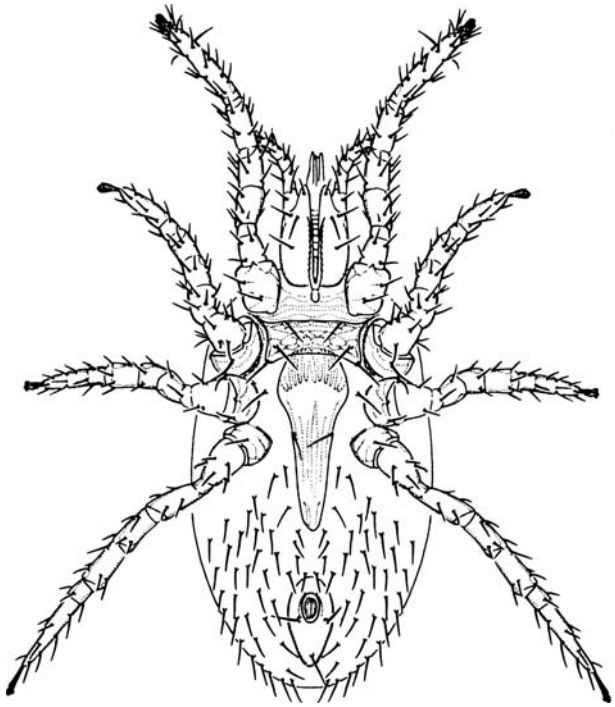


FIGURE 26.6 Tropical rat mite, *Ornithonyssus bacoti* (Macronyssidae), female, ventral view. *Modified from Gorham, 1991; courtesy of the US Department of Agriculture.*

both tropical and temperate regions. In cooler climates, this mite occurs only indoors and in nests of wild rodents. Occasionally it also infests carnivores, birds, and humans. When rats are killed or abandon their nests or runways, the mites are left behind and will readily crawl or drop onto humans and other passing animals. Rodents killed by household cats and left near human dwellings also can serve as a source of infestation. The mites are active and can move some distance from their source to enter nearby buildings.

Human bite cases involving the tropical rat mite usually occur in rodent-infested buildings. Using their long, slender chelicerae, they probe the skin in an effort to feed on blood. In some cases, they produce a prickling sensation at the bite sites, whereas in other cases the bite is painful. Multiple bites are often clustered and subsequently develop into a pruritic, erythematous, papular rash. This may be accompanied by localized swelling and occasional vesicle formation. Although they will bite almost any part of the body, they tend to bite where the clothing is tight (e.g., neck, shoulders, and waist). *Ornithonyssus bacoti* is visible to the unaided eye and may be seen crawling on the clothing or skin, floors, walls, and other structural surfaces.

This mite has been shown experimentally to be capable of being infected with or transmitting several human pathogens, including those that cause murine typhus, rickettsialpox, plague, tularemia, and coxsackie virus disease. However, their importance in the epidemiology of these diseases is regarded as negligible. On the other hand, evidence supports the possibility that *O. bacoti* may serve as both a vector and reservoir of Hantaan virus, the causative agent of Korean hemorrhagic fever (epidemic hemorrhagic fever) of humans in Asia.

Tropical Fowl Mite (Ornithonyssus bursa)

As its common name implies, the tropical fowl mite (Fig. 26.7) is distributed widely throughout subtropical and tropical parts of the world, where it parasitizes various domestic and wild birds. It occurs in the southeastern and southwestern United States, Hawaii, Central America, Colombia, South Africa, India, China, and Australia. In addition to being a poultry pest, notably attacking chickens, it parasitizes pigeons, house sparrows, grackles, and other wild avian hosts. It can also cause problems in zoos and aviaries. Human bite cases can usually be traced to nesting birds under eaves, on ledges, and in other building structures. The mites spend most of their time in the nest, moving onto the host to feed.

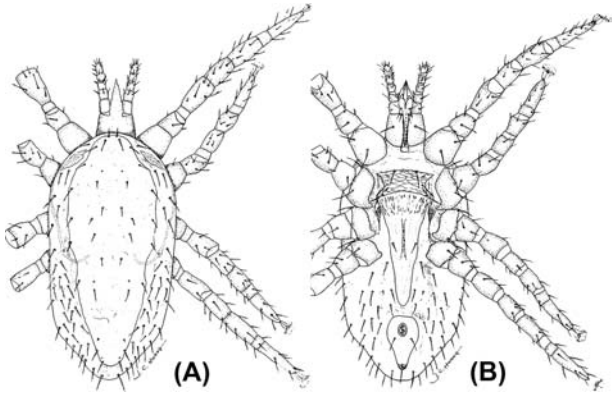


FIGURE 26.7 Tropical fowl mite, *Ornithonyssus bursa* (Macronyssidae), female. (A) Dorsal view; (B) ventral view. *Courtesy of Strandtmann and Wharton, 1958.*

Human bites typically occur when young birds leave the nest or the nest is otherwise abandoned, compelling the mites to wander in search of alternative hosts. Wild birds carrying the mites commonly infest poultry operations, which leads to workers being bitten when they come in contact with infested, commercially produced birds or their nest materials. Although less common than cases involving the tropical rat mite, human bites caused by the tropical fowl mite can be equally discomforting. The latter tend to be more sharply painful and result in more persistent itching. Although WEE virus has been isolated from *O. bursa* in house sparrow nests, there is no evidence that it actually transmits the virus. No other human pathogen has been associated with this mite.

Northern Fowl Mite (Ornithonyssus sylviarum)

The northern fowl mite (Fig. 26.8) is widely distributed throughout temperate regions of the world as a parasite of domestic fowl and wild birds. Although regarded as a major pest of chickens, it occasionally bites people. Most human cases result when poultry workers handle infested birds or when the mite enters buildings from nearby bird nests. Although bite cases may occur year-round in commercial poultry operations, most cases in homes and other work places occur about the time when young birds fledge and the adults vacate their nests. The bite reaction is similar to that of *O. bursa*, producing red papular skin lesions, often accompanied by intense itching.

The viruses that cause WEE and St. Louis encephalitis have both been detected in *O. sylviarum* from nests of wild birds in North America. In the case of WEE virus, it has been shown to persist in avian hosts and to be transmitted by bite to other birds. Newcastle disease virus has been detected in *O. sylviarum* after its feeding on infected chickens, but the virus does not establish persistent infection in the mite. The tropical fowl mite does not appear to have a significant role in the natural transmission of these or other arboviruses affecting humans.

Free-Tailed Bat Mite (Chiroptonyssus robustipes)

This mite is a common blood-feeding ectoparasite on the Brazilian (or Mexican) free-tailed bat (*Tadarida*

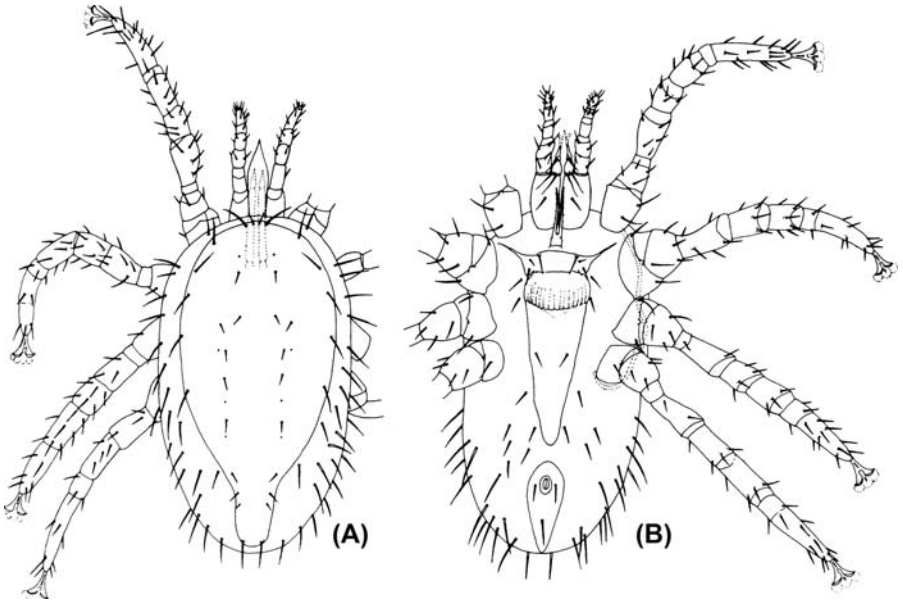


FIGURE 26.8 Northern fowl mite, *Ornithonyssus sylviarum* (Macronyssidae), female. (A) Dorsal view; (B) ventral view. *Modified from Strandtmann and Wharton, 1958.*

braziliensis) roosting or nesting in walls and attics of buildings throughout its range in the southern United States, Mexico, and Central America. Occasionally it is found in low numbers on other molossid bats roosting with *T. braziliensis*. Only the protonymphs and adult mites feed on blood and other tissue fluids of their bat hosts. Human bite cases are uncommon and usually involve zoologists handling infested bats or otherwise working with mite-infested bat colonies. The mites do not readily bite and are primarily a nuisance because they actively crawl about on the skin and clothing. On rare occasions, *C. robustipes* has been reported to invade the living quarters of homes, where it has bitten the occupants. One such case involved an 18-month-old boy in California (United States) who was bitten repeatedly on the face and abdomen, causing a persistent dermatitis. The problem was not resolved until it was discovered that a wall infestation of Brazilian free-tailed bats was the source of mites that were biting the child each time he was bathed in a bathroom sink (Keh, 1974).

Snake Mite (Ophionyssus natricis)

This mite (Fig. 26.9) is a common pest of captive snakes and only rarely has been reported biting people. Human bites occur primarily in reptile houses at zoological parks, affecting personnel who handle infested snakes. A well-documented case involved several members of a family in a household where a python was kept as a pet (Schultz, 1975). The family members had experienced skin lesions in the form of a papular rash on the forearms and other parts of the body over a 5-month period before the source of the problem was identified. Mites were observed to be attached to the skin while attempting to feed and also were found in a chair frequented by the snake. Humans do not serve as suitable hosts for this mite. The mites tend to become immobile with their legs curled underneath the body within a few minutes after they begin feeding on human blood; often they do not recover. They do not transmit any known human pathogens.

Laelapidae

Members of this family include both free-living and parasitic species and are often associated with rodents and other nest-building mammals. Some classifications treat the subfamilies Haemogamasinae and Hirstionyssinae as separate families (Beaulieu et al., 2011). The only significant laelapid species that may affect human health is the spiny rat mite. Occasionally other species cause temporary discomfort to humans, as reported in possible cases of *Haemogamasus pontiger* causing dermatitis in England (Theiler and Downes, 1973).

The laelapid *Haemogamasus liponyssoides* is an obligate blood feeder on wild rodents that has the potential to transmit human disease agents, even though it has not been reported to bite people (Furman, 1959). Other rodent-associated laelapid mites may have a role in transmitting Hantaan virus, the causative agent of Korean hemorrhagic fever, based on isolation of this human pathogen from *Laelaps jettmari* in Korea (Traub et al., 1954).

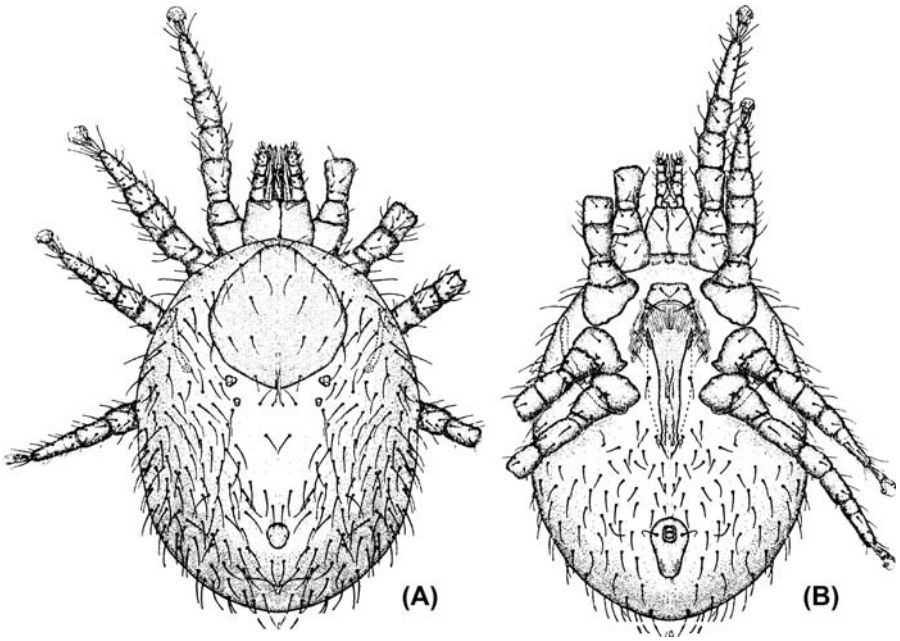


FIGURE 26.9 Snake mite, *Ophionyssus natricis* (Macronyssidae), female. (A) Dorsal view; (B) ventral view. *Modified from Strandtmann and Wharton, 1958.*

Spiny Rat Mite (Laelaps echidninus)

The spiny rat mite is a common hematophagous or tissue fluid-feeding ectoparasite of domestic rats throughout the tropical and temperate zones. Although it is capable of biologically transmitting disease agents, such as the agent of murine typhus among wild rodents, its potential role as a vector of human pathogens remains uncertain. Junin virus, which causes Argentinian hemorrhagic fever, has been isolated from *L. echidninus* and associated rodent hosts in South America (Parodi et al., 1959; Theiler and Downes, 1973).

Trombiculidae

Larvae of members of the family Trombiculidae are called **chiggers**, **red bugs**, and **berry bugs**. Larvae in the families Leeuwenhoekiidae and Walchiidae are also called chiggers and have a similar life history, but species in these families rarely interact with humans. This is the only parasitic stage in the life cycle of trombiculid mites. As a group, they feed on a wide variety of vertebrate hosts including amphibians, reptiles, birds, and mammals, with humans serving only as accidental hosts.

The life history of trombiculid mites includes the following sequence of stages: egg, prelarva (deutovum), larva, protonymph (nymphochrysalis), deutonymph (nymph), tritonymph (imagochrysalis), and adult (Fig. 26.2). The eggs are typically laid in soil or ground debris. After about 6 days, the egg shell splits to expose an inactive prelarva. After another 6 days, the active six-legged larva (i.e., chigger) is produced (Fig. 26.10). After successfully attaching to a suitable host, the larva generally feeds for 3−5 days on the host before dropping to the ground to form an inactive transitional stage, the protonymph or nymphochrysalis. This stage then develops into the active eight-legged deutonymph. The subsequent tritonymph is another quiescent stage, which yields the eight-legged adult. The deutonymph and adult are free-living predators that feed on small arthropods (e.g., collembolans) and their eggs. The duration of the life cycle requires 2−12 months or longer, depending on the species and environmental conditions. In temperate areas, there may be one to three generations per year, whereas in tropical regions generations may be continuous throughout the year.

Although trombiculid larvae usually cause little or no apparent harm to their normal hosts, they often cause dermatitis when they attach to and attempt to feed on humans and other atypical hosts. Such an infestation by trombiculid larvae is called chigger dermatitis, or **trombiculosis** (trombidiosis in the older literature).

Chiggers are just large enough (150−300 μm) to be visible to the unaided eye. They are yellowish, orange, or red and can be seen on close inspection at the center of the skin lesions they induce. Unfortunately, chiggers are often encountered in large numbers, resulting in multiple bites (Fig. 26.11). Given their preference for attaching where clothing fits snugly against the skin, the bites tend to be concentrated about the ankles, lower legs, and waist and along the elastic borders of undergarments.

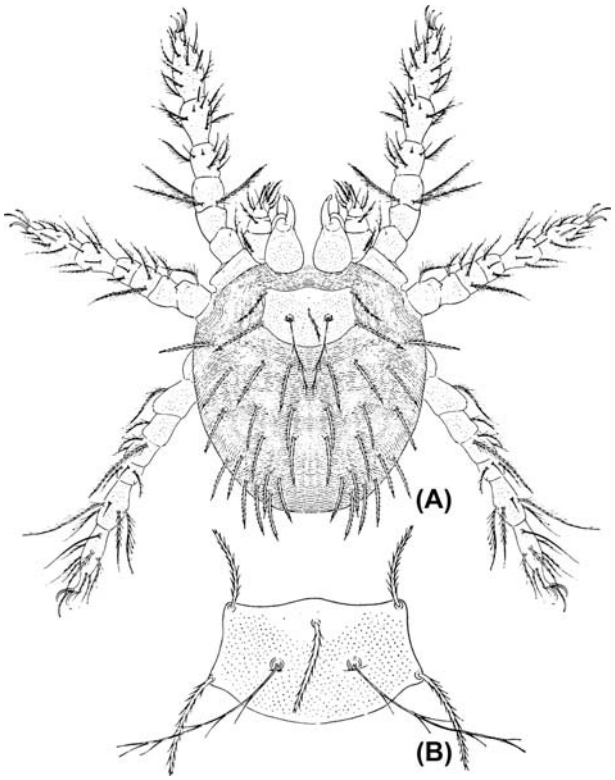


FIGURE 26.10 Larval stage (chigger) of the harvest mite, *Neotrombicula autumnalis* (Trombiculidae), of Europe. (A) Dorsal view; (B) scutum (dorsal plate), showing characteristic arrangement of setae. *Modified from Baker et al., 1956.*

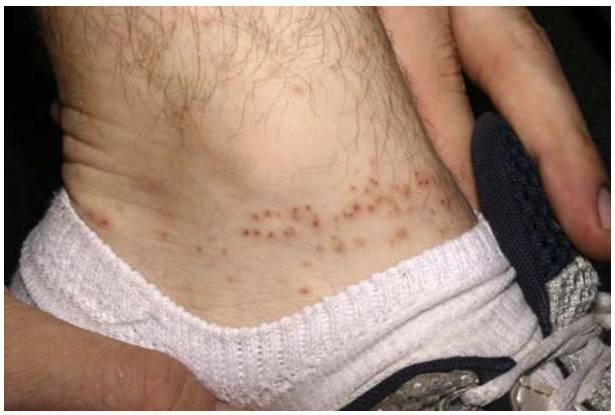


FIGURE 26.11 Multiple chigger-bite lesions on human foot, with top of ankle sock pulled down to reveal the location of attached mites where the fabric fits snugly against the skin. *Photograph by Nathan Burkett-Cadena.*

Contrary to popular belief, chiggers do not burrow into the skin of their hosts. Instead they attach by piercing the epidermis with their chelicerae and feed externally. Because of their small size and tiny mouthparts, chiggers usually attach where the skin is thin or soft. A preferred site is the opening to hair follicles. There they insert their capitulum to feed on the thin epidermal lining. In humans, this results in inflammation at the point of attachment and localized swelling of the skin around the chigger, giving the mistaken impression that it has burrowed into the skin. Their food consists primarily of partially digested skin cells and lymph broken down by saliva introduced at the attachment site. They do not feed on blood. Feeding is facilitated by the formation of a feeding tube, or stylostome, produced by the interaction of the saliva with surrounding host tissue.

With the exception of *Leptotrombidium* species, chiggers often do not survive more than 1–2 days on human hosts, owing to the adverse host reaction they cause and injury or removal caused by scratching. By then, however, the damage is already done, typically producing a discrete, persistent, itching, reddened papule at each attachment site. The lesions persist for several days but may take several weeks to heal if they become secondarily infected. The recovery time can be significantly shortened by prompt treatment to kill the chiggers when they are first detected, generally within 3–6 h after attachment, application of a topical medication to alleviate itching and prevent infection, and avoidance of scratching or otherwise excoriating the skin.

More than 50 species of trombiculid mites have been recorded attacking humans. Of this number, about 20 species are considered to be medically important, either because of the dermatitis they cause or owing to their role in transmitting disease agents. Five species of particular interest are *Eutrombicula alfreddugesi* in North America and South America, *Neotrombicula autumnalis* in Europe, and *Leptotrombidium akamushi* and *L. deliense* in the Orient. In older literature, all of these species were assigned to the genus *Trombicula*.

Eutrombicula alfreddugesi

This is the most common and widespread trombiculid mite in the Western Hemisphere, occurring from Canada to Argentina and in the West Indies. In North America, it and related species that attack humans are known as **red bugs**, especially in the southeastern United States. In Mexico, it is called *tlalzahuatl*, and in Mexico and other parts of Latin America it is called *coloradilla* and *bicho colorado*. *Eutrombicula alfreddugesi* is parasitic as larvae on a variety of amphibians, reptiles, birds, and mammals. It is particularly common in areas of secondary growth, such as shrub and brush thickets and blackberry and bramble (*Rubus* spp.) patches, and along margins of swamps and ecotones between woodlands and open fields or grasslands. The larvae are present in late summer and early fall in the more temperate parts of its range, and throughout the year in the tropics and subtropics, including southern Florida (United States). Although it is the most common cause of chigger dermatitis in the

New World, it is not involved in the natural transmission of any human disease agent.

Neotrombicula autumnalis

Known as the **harvest mite,** this is the most common chigger that attacks humans in Europe and the British Isles (Fig. 26.10). Other names include *aoutat* and *lepte automnal*. As both its common and scientific names suggest, *N. autumnalis* is particularly annoying during harvest time in late summer and fall. The larvae are present from July to the onset of winter, usually reaching peak populations in early September. They tend to be most active on warm, sunny days in grasslands, cultivated grain fields, brush lands, and thickets. The widespread occurrence of this mite reflects its wide variety of natural hosts, especially mammals and certain ground-dwelling birds. Rabbits are a particularly common host. Other hosts include voles, wood mice, hedgehogs, squirrels, cattle, sheep, goats, horses, dogs, cats, pheasants, partridges, chickens, and other domestic fowl.

Other Eutrombicula Species

Eutrombicula splendens occurs in the eastern United States from the Gulf Coast north to Massachusetts, Minnesota, and Ontario, Canada. It is especially common in the southeastern United States, where it is second only to *E. alfreddugesi* as the cause of trombiculosis in humans. Although it occurs in drier habitats with *E. alfreddugesi*, it is especially abundant in moist habitats such as swamps, bogs, and low-lying areas with rotting stumps and fallen trees. The larva is parasitic on amphibians, reptiles, birds, and mammals, but seems to prefer snakes and turtles as natural hosts. The seasonal occurrence of *E. splendens* is similar to that of *E. alfreddugesi*. Another *Eutrombicula* species that causes chigger dermatitis of humans in the United States is *E. lipovskyi*. It is restricted to moist areas, generally characterized by an abundance of decaying logs and stumps bordering swamps and streams. It occurs from Alabama and Tennessee west to Arkansas, Oklahoma, and Kansas. Reptiles, rodents, and birds serve as natural hosts.

Leptotrombidium spp.

Several members of the genus *Leptotrombidium* serve as vectors of *Orientia tsutsugamushi*, the causative agent of tsutsugamushi disease. They occur widely throughout Southeast Asia, the southwestern Pacific Islands, and northern Australia. As a group, the larvae of medically important *Leptotrombidium* species are parasitic primarily on ground-dwelling rodents (e.g., *Rattus*, *Microtus*, and *Apodemus* spp.). Other hosts include insectivores, marsupials, cattle, dogs, and cats. They occur in forests, second-growth areas along the margins of woodlands, in river valleys, and in abandoned agricultural fields where populations of rodents flourish. The principal vectors of the tsutsugamushi disease agent are *L. deliense* in Southeast Asia, the southwestern Pacific Islands, and northern Australia; *L. akamushi* in Japan; *L. arenicola*, and *L. fletcheri* in the Pacific Islands; and *L. pallidum*, *L. pavlovskyi*, and *L. scutellare* in more restricted regions of the Asian mainland, Japan, and Malaysia (Table 26.3).

Stored-Products Mites

Members of several families of mites that infest unprocessed and processed plant materials can cause human dermatitis and other health-related problems. Most cases involve people handling infested materials such as grains, flour, hay, straw, dried fruits, and vegetables. Others involve processed materials of animal origin, such as meats, hides, cheeses, dried milk, and other dairy products. Such mite infestations are the cause of occupational acarine dermatitis in farmhands, granary operators, warehouse workers, and other personnel.

Stored-products mites responsible for most human cases of acarine dermatitis are members of the families Acaridae, Glycyphagidae, Pyemotidae, and Cheyletidae.

TABLE 26.3 Major Chigger Vectors of *Orientia tsutsugamushi*, the Causative Agent of Tsutsugamushi Disease in Humans

Trombiculid Species	Geographic Occurrence
L. akamushi	Japan
L. arenicola	Malaysia, Indonesia, Thailand
L. deliense	Southeast Asia, China, southwestern Pacific Islands, northern Australia, Pakistan
L. fletcheri	Malaysia, New Guinea, Philippines, Indonesia, Melanesia
L. pallidum	Japan, Korea, Primorye region of Russia
L. pavlovskyi	Primorye region of Russia
L. scutellare	Japan, China, Thailand, Malaysia

Source: Kawamura et al., 1995.

Acaridae and Other Astigmata

Acarid mites infest a wide range of stored materials such as grains, milk products, dried fruits, straw, and animal hides in both households and commercial storage facilities. They also are common contaminants of culture media in which insects and other invertebrates are reared; of bedding materials for mice, guinea pigs, hamsters, and other vertebrates; and in animal feed and animal-holding cages in pet stores and zoos. Their numbers can build rapidly, especially when the infested materials are damp enough to support the growth of fungi on which they typically feed. Dermatitis occurs when the mites pierce the skin in attempts to feed or obtain moisture. The reaction in some cases also may involve contact allergens.

The most important acarid mite in stored products is *Acarus siro* (Fig. 26.12), a species found throughout most of the world. It is particularly a pest of processed cereal products (e.g., flour), rather than whole grains or hay and is one of the most common mites infesting cheese. The females are 350−650 μm in length, with a colorless body and yellow-to-brown gnathosoma and appendages. It can develop at temperatures of 24−32°C and a relative humidity greater than 60%. This mite tends to congregate where the relative humidity is 80%−85%, at which its reproductive rate is highest. The amount of damage it causes to grains is directly related to the moisture content; the germ is attacked only when the water content is 14% or higher. The dermatitis experienced by food handlers on contact with *A. siro* is commonly known as **grocer's itch**. Other names for dermatitis caused by *A. siro* and related mites are baker's itch, dried fruit mite dermatitis (*Carpoglyphus lactis*; Carpoglyphidae) (Fig. 26.13), wheat pollard itch (*Suidasia nesbitti*, family Suidasiidae), and vanillism, reflecting the product or commodity involved.

A mite closely related to *A. siro* that also causes human dermatitis is *Acarus farris* (Fig. 26.14). It is a widespread species that has been reported to cause skin irritation to farm workers handling infested bales of hay in England (Hughes, 1976).

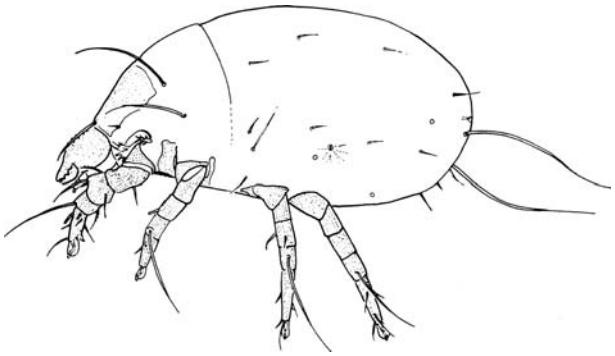


FIGURE 26.12 *Acarus siro* (Acaridae), female, lateral view. *Modified from Hughes, 1976.*

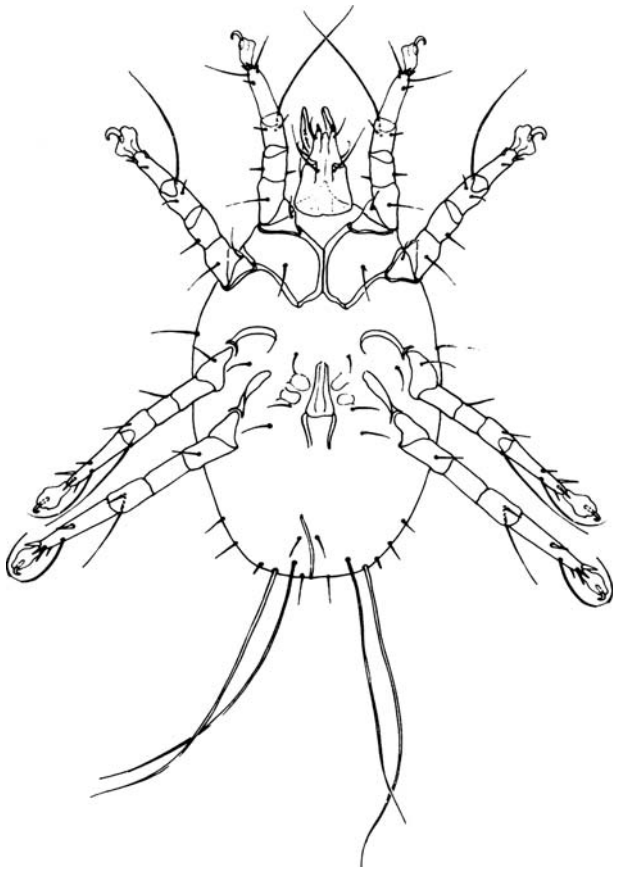


FIGURE 26.13 *Carpoglyphus lactis* (Carpoglyphidae), male, ventral view. *Modified from Hughes, 1976.*

Another common acarid mite in stored products is the cosmopolitan *Tyrophagus putrescentiae*. It is particularly a problem in foods with a high protein and fat content such as hams, cheeses, nuts, seeds, dried eggs, and fish meal. This mite feeds primarily on fungi (e.g., *Aspergillus*, *Eurotium*, *Penicillium*) that tend to thrive on foods stored at warm temperatures (>30°C) and relatively high humidities (>85%). Under such conditions, it can complete its development from one generation to the next in 2−3 weeks. It can be a pest in mycology laboratories, where it often contaminates fungal cultures. The term **mold mite** is commonly used to refer to *T. putrescentiae* and a closely related species, *T. longior* (Fig. 26.15).

In the tropics, *T. putrescentiae* causes a dermatosis called **copra itch** among workers handling copra, dried coconut kernels from which coconut oil is extracted. In Italy, human cases of cutaneous and respiratory allergies have been attributed to this mite among workers handling raw hams; the mite apparently thrives in the white dust (*ruffino*) that covers hams during the seasoning process

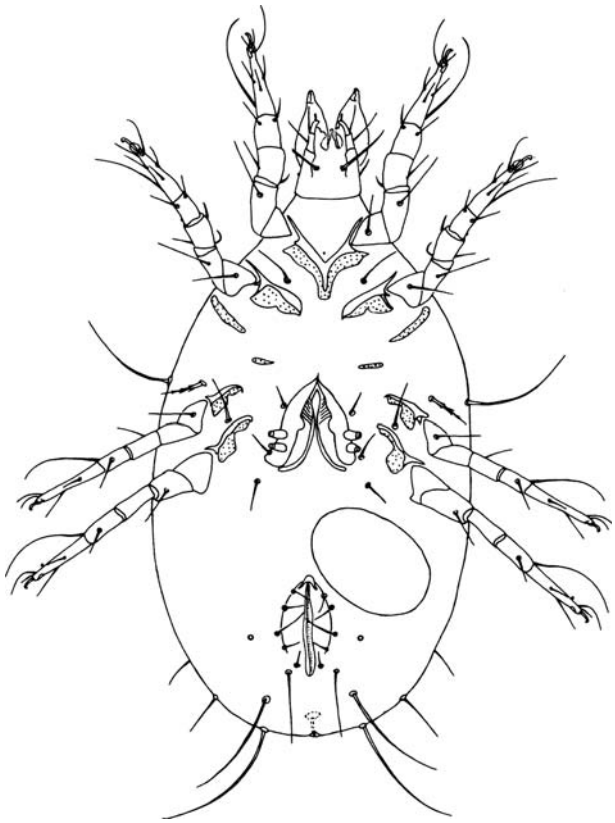


FIGURE 26.14 *Acarus farris* (Acaridae), female, with single egg, ventral view. *Modified from Hughes, 1976.*

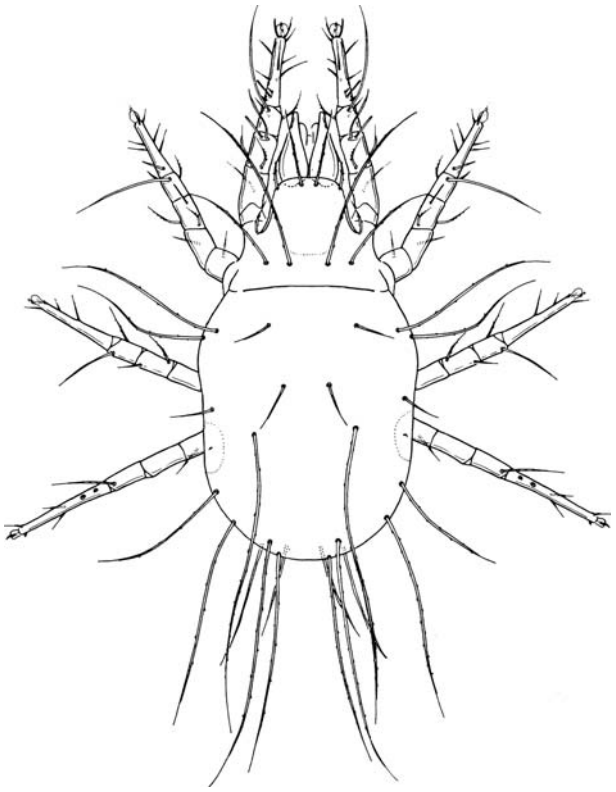


FIGURE 26.15 *Tyrophagus longior* (Acaridae), male, dorsal view. *Modified from Hughes, 1976.*

(Ottoboni et al., 1989). *Tyrophagus putrescentiae* occurs throughout much of the world, where it is found in a wide range of situations, including grasslands, soil, old hay, mushrooms, and the nest of bees and ducks. This mite was reported to cause human dermatitis in a butcher's shop in Austria, where it was breeding in molds growing on bacon in a poorly ventilated room (Czarnecki and Kraus, 1978).

A few other acarid mites are known to cause human dermatitis. One is *Tyrolichus casei*, reported by Henschel (1929). This is a cosmopolitan species commonly found in stored foods, grains, flour, cheeses, dog meal, old honeycombs, and insect collections (Hughes, 1976). Another is *Suidasia nesbitti* which occurs in Europe, Africa, North America, and the West Indies. Although it is particularly associated with wheat pollards and bran in England (Hughes, 1976), it also has been reported in rice, whale meat infested with dermestid beetles, dried bird skins, and milking machinery.

Pyemotidae

Pyemotid mites are external parasitoids of insects that typically attack the larval stage of moths, beetles, and hymenopterans. A few species commonly occur in dried, insect-infested plant products such as hay, straw, and grains. Upon contact with humans and other animals, these mites cause intense itching when they pierce the skin with their stylet-like chelicerae and inject a toxin produced in their salivary glands. It is a potent neurotoxin that they use to immobilize their insect prey, enabling them to paralyze insects 150,000 times their size (Tomalski and Miller, 1991).

The most important species affecting humans is *Pyemotes tritici* (Fig. 26.16). It is variously known as the **straw itch mite**, **hay itch mite**, and **grain itch mite**, depending on the plant material with which it is associated. Exposure to *P. tritici* represents an occupational hazard for agricultural workers, sales and stock personnel in farm supply stores, and other individuals in the arts-and-crafts field who handle wheat, hay, and straw. People handling infested materials usually develop multiple skin lesions in the form of papules or papulovesicles, accompanied by intense itching. Each bite site typically consists of a minute white wheal with a central reddened area where a tiny vesicle forms. During the early stages, the mite often is visible as a tiny white speck where the vesicle is located. Although lesions can occur on any exposed part of the body, they usually appear on the back, abdomen, and forearms, where contact with infested materials typically takes place. Lesions seldom occur on the

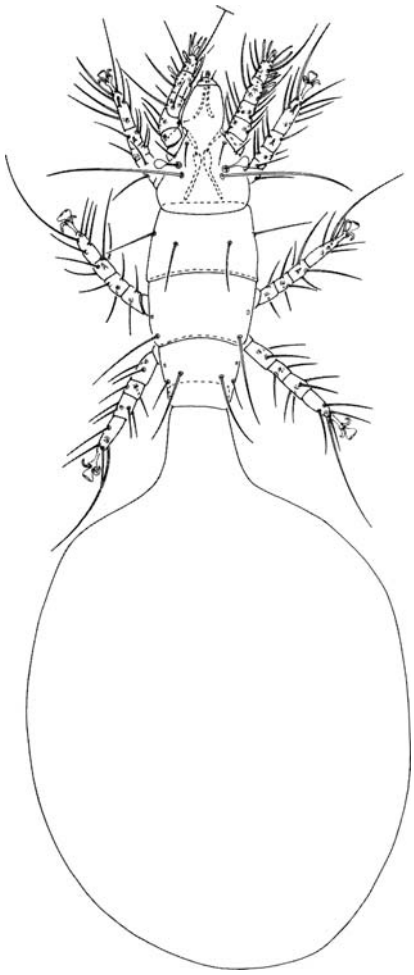


FIGURE 26.16 Straw itch mite, *Pyemotes tritici* (Pyemotidae), gravid female, dorsal view. *From Gorham, 1991; courtesy of the US Department of Agriculture.*

face or hands. Heavily infested or sensitized individuals may experience other symptoms, including headache, fever, nausea, vomiting, diarrhea, and asthma (Southcott, 1984). Less commonly reported are chills, fever, malaise, and anorexia (Betz et al., 1982).

Two other species of *Pyemotes* reportedly cause human dermatitis. Workers in France developed erythematous lesions and reported intensely itchy papules after handling dried everlasting flowers (*Helichrysum angustifolium*) infested with *P. zwoelferi* imported from Yugoslavia (Le Fichoux et al., 1980). People working in a feed-mixing shed of a pig farrowing house developed a papular rash after contact with grain infested by *P. herfsi* in former Czechoslovakia, whereas workers handling commercial cultures of mealworms (*Tenebrio molitor*) infested with *P. herfsi* developed wheals in former West Germany. *Pyemotes herfsi* also has caused outbreaks of dermatitis in the central United States, where the mite is a parasitoid of an oak leaf gall midge, *Contarinia* sp. (Diptera: Cecidomyiidae) (Broce et al., 2006).

Cheyletidae

Cheyletid mites are mostly free-living predators that commonly feed on other mites and small arthropods in stored products. Occasionally they cause pruritic dermatitis in people handling infested grains and other dried plant materials. The most common cheyletid found in stored products is *Cheyletus eruditus*. This cosmopolitan species has been used commercially as a biological control agent to reduce the numbers of grain mites, notably *Acarus siro* and *Lepidoglyphus destructor*, in granaries and agricultural warehouses. Severe pruritus was reported in a worker at a wholesale florist shop who handled fern wreaths imported to the United States from the Philippines (Shelley et al., 1985). The mite involved was apparently *Cheyletus malaccensis*, a species previously shown to cause itching papules in humans (Yashikawa et al., 1983). A second cheyletid mite, *Cheyletomorpha lepidopterorum*, also may have been involved.

Skin-Invading Mites

Representatives of only two families of mites typically invade human skin or associated dermal structures and glands. They are the Demodecidae, or follicle mites, and the Sarcoptidae, or scabies mites. Whereas only a relatively small number of humans infested with follicle mites develop clinical problems, most individuals who become infested with the human scabies mite experience an annoying, often severe dermatitis.

Demodecidae

Members of this family are called **follicle mites**, although species also occur in skin glands, the oral cavity, and reproductive tract of some small mammal hosts. They are extremely tiny, elongate, annulate mites with very short, stout, three-segmented legs (Fig. 26.17). They lack body setae and possess a pair of tiny, needle-like chelicerae that are used to pierce dermal cells on which the mites feed. Their minute size and strong reduction of most of the external features represent adaptations for living in the close confines of hair follicles and associated ducts and glands.

Two species of *Demodex* infest humans. *Demodex folliculorum* (Fig. 26.17) occurs primarily in hair follicles, whereas *Demodex brevis* is generally found in the sebaceous glands (sweat glands) that open via ducts into the hair follicles. Both species may infest the same host, appearing together in samples taken from a given individual. Adults of the two species closely resemble one

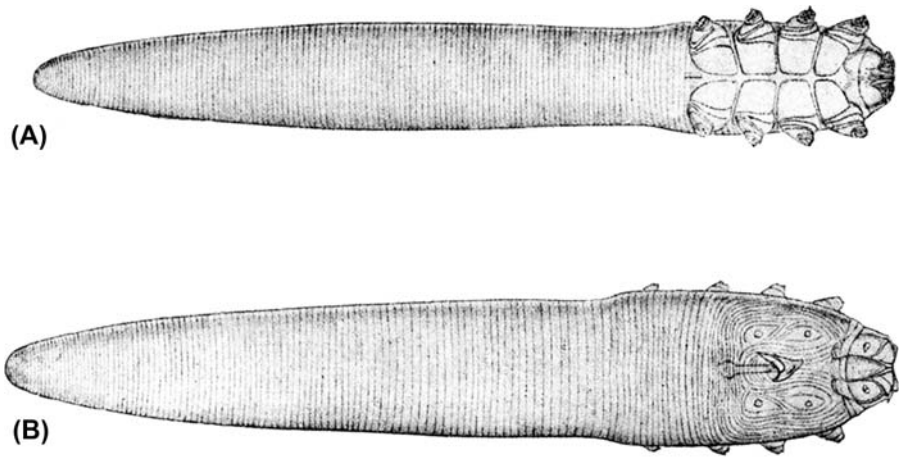


FIGURE 26.17 Human follicle mite, *Demodex folliculorum* (Demodecidae), female. (A) Female, ventral view; (B) male, dorsal view. *From Hirst, 1922.*

another but can be distinguished based on the general body shape and relative size of the males and females. *Demodex folliculorum* females have elongate bodies that are gently tapered from the podosoma to the slender, rounded caudal end. *Demodex brevis* females have bodies that are usually widened posterior to the podosoma and terminate in a more broadly rounded caudum. The eggs are also distinctive; they are spindle-shaped in *D. folliculorum* and oval in *D. brevis*.

The entire life cycle of *D. folliculorum* (Fig. 26.18) and *D. brevis* is spent on their human host. The mites feed by piercing host cells with their styletiform chelicerae and drawing the cell contents into the esophagus with a pumping action of the pharynx. They are highly host-specific and can survive only on humans. Transfer of mites from one individual to another is presumed to occur primarily between mothers and infants during the intimacy associated with facial contact and nursing. Adults of both sexes are readily transferred between hosts at these times. That 90%−100% of all humans apparently harbor follicle mites attests to the success with which such transfers are accomplished.

Human follicle mites tend to occur primarily in the regions of the forehead, eyelids, and nose. They also can occur in the eyebrows, the Meibomian glands of the eyelids, perioral mucosa, ear canal, chest, nipples, and other parts of the body (Nutting et al., 1989). In most cases, they cause no apparent harm and go virtually unnoticed. Only under unusual circumstances, which remain largely unexplained, do they cause clinical problems that warrant medical attention. Such cases involving dermal reactions to *Demodex* mites are called **demodicosis**. It does not appear that any specific pathogens are involved, although secondary bacterial infections can aggravate the condition.

In addition to differences in one's body chemistry and immunological responses, certain hormones affect population levels of *Demodex* mites and the development of demodicosis. Populations tend to build as the host matures, leveling off in the middle-aged groups. Substances such as diethylstilbestrol tend to inhibit mite populations, whereas progesterone and testosterone may promote an increase. The long-term use of topically applied corticosteroids has been correlated with an increased incidence of demodicosis, which suggests a possible link between hormonal levels and the development of inflammatory reactions induced by follicle mites. Cases of human demodicosis can be categorized in five clinical forms: demodex folliculitis, demodex blepharitis, pityriasis folliculorum, demodex granuloma, and human demodectic mange.

Demodex folliculitis occurs most commonly on the face, but also on the forearms and chest. It typically causes rosacea-like skin lesions, initially appearing as red follicular papules and tiny pustules. This is the most difficult *Demodex* infestation to diagnose because it is almost indistinguishable clinically from other skin problems such as acne cosmetica, corticosteroid telangiectasia (dilated blood vessels within the skin that have a tortuous appearance), and rosacea. In some cases, it may complicate or aggravate preexisting skin conditions. Confirmation of demodicosis therefore depends on demonstrating the presence of the mites, all stage of which can be found in the pustule contents.

Demodex blepharitis, also known as ocular demodicosis, is an inflammation of the hair follicles of the eyelids associated with high populations of demodicid mites. The patient's eyelids typically itch or burn and become reddened and are often characterized by accumulations of waxy or gelatinous debris at the base of the eyelashes. The

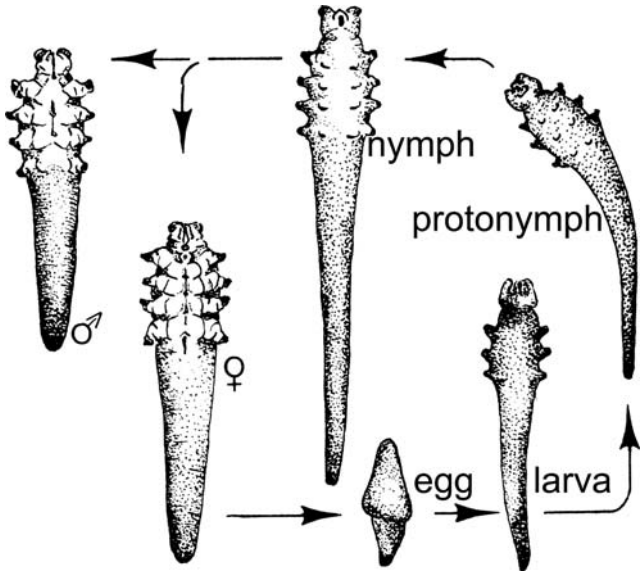


FIGURE 26.18 Life cycle of human follicle mite, *Demodex folliculorum* (Demodecidae). *Modified from Nutting, 1984.*

presence of mites can usually be detected by plucking affected lashes and examining them under a microscope.

Pityriasis folliculorum is an uncommon form of demodicosis that is clinically recognized as dry, scaly skin with brownish or grayish hyperpigmentation and associated pruritus. The condition is often intensified by scratching or shaving, resulting in erythema, excoriation, mottling, and what has been described as a nutmeg-like roughening of the affected skin. It usually occurs on the face and neck in both young and older adults.

Demodex granuloma results when follicle mites rupture out of blocked hair follicles into subcutaneous tissues. There, they can elicit a response by lymphocytes and histiocytes to form granulomas.

Human demodectic mange is the term applied to cases of human demodicosis in which transient infestations of humans by *Demodex* spp. are contracted from other host species. The most common source is dogs and usually involves intimate contact, such as sleeping with a pet. Patches of papules and vesicles develop, accompanied by a burning or itching sensation. Commonly affected areas include the chin, neck, chest, forearms, stomach, and thighs, reflecting the skin surfaces most likely to come in contact when handling household pets.

A positive diagnosis of demodicosis is made by confirming the presence of large numbers of *Demodex* mites directly associated with the affected areas of skin. The mites can be seen by microscopic examination of skin scrapings, pustule contents, cellular debris from hair follicles, and plucking eyelashes in the case of demodex blepharitis. Adhesive cellophane tape, applied to infested areas of the skin, can be used to recover demodicid mites near the follicular orifices or moving on the surface of the skin. A follicular biopsy also can be helpful, in which a quick-setting cyanoacrylate polymer is used to extract the contents of sebaceous follicles (Mills and Kligman, 1983). Various stages of the mites, including eggs, are usually evident.

Cases of human demodicosis can be effectively treated by daily washing of the affected skin with mild, alkaline, or sulfur soap, followed by application of a mild sulfur lotion sold for this purpose. Other compounds such as gamma benzene hexachloride (lindane), metronidazole, and physostigmine ophthalmic ointment in blepharitis cases also are effective. When properly treated, cases often are resolved in 2–3 weeks, they but may take as long as 2 months. This is not to say that the mites are eliminated; their numbers simply are reduced to lower levels that do not cause pathogenesis. Regular daily washing of the face and eyelids with alkaline soap suppresses *Demodex* populations and reduces the risk of developing demodicosis. The use of mascara also seems to retard mite increases. On the other hand, the regular use of medicated creams, skin moisturizers, and topical applications of corticosteroids tends to promote *Demodex* numbers, leading to heavier infestations and increased prospects of related skin problems.

For further information on *Demodex* species and their medical importance, see Desch and Nutting (1972), Nutting (1976a,b,c), English and Nutting (1981), Rufli and Mumculoglu (1981), Franklin and Underwood (1986), and Burns (1992).

Sarcoptidae

The only mites in this family that commonly infest humans are members of the genus *Sarcoptes*, generally referred to as **scabies mites**. They represent a complex of varieties or physiological types of the single species *Sarcoptes scabiei*.

Human Scabies Mite (Sarcoptes scabiei)

The form that typically infests people is called the human scabies mite, or human itch mite, *S. scabiei* var. *hominis* (varietal names simply indicate the host from which *S. scabiei* was recovered). This mite is cosmopolitan in distribution and infests human populations of all races as an obligate parasite that lives in the skin. The adults are small (females 350–450 μm; males 180–240 μm in length) and rounded, with tiny pointed, triangular spines on their dorsal surface that assist them in burrowing (Fig. 26.19). These spines are more numerous and conspicuous in females than in males. The legs are short, with legs I and II of the female and legs I–III of the male each bearing a terminal sucker. The two hind pairs of legs of the

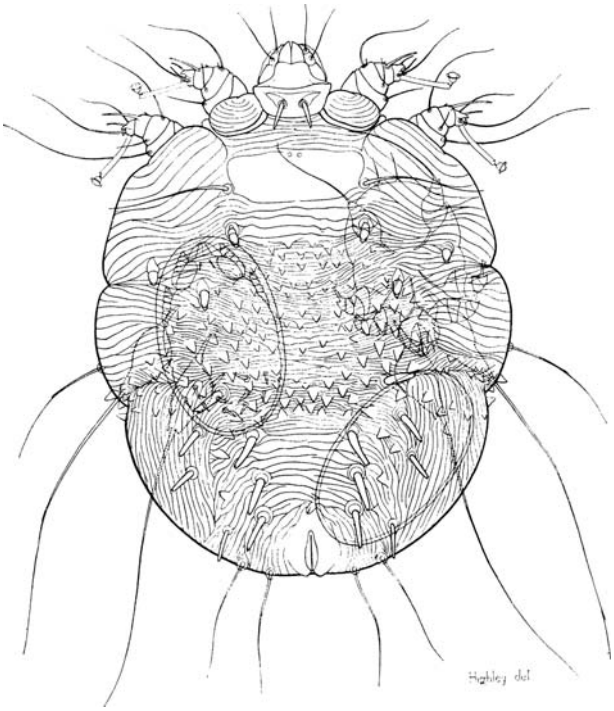


FIGURE 26.19 Human scabies mite, *Sarcoptes scabiei* (Sarcoptidae), female, with two developing eggs, dorsal view. *From Hirst, 1922.*

female and the last pair of legs in the male lack a sucker and instead terminate in long setae or bristles.

The adult mites can crawl rapidly onto the surface of the skin, with females traveling up to 2.5 cm/min. Upon finding a suitable site, the female uses her chelicerae and first two pairs of legs to burrow into the skin, disappearing beneath the surface in about 1 h. There she waits in this temporary pit, or shallow burrow, for a wandering male to find her, after which mating takes place. The fertilized female then emerges on the skin surface and searches for a site in which to excavate a permanent burrow. She penetrates the skin once again and makes her way down through the stratum corneum, or horny layer of the skin, to its lower boundary with the underlying stratum granulosum. There she excavates a horizontal burrow within the stratum corneum, where she will spend the rest of her life, commonly 30 days or more. During this time, she continues to extend the length of her burrow by 0.5 mm/day or more, commonly reaching a total length of 1 cm or more. As viewed from the skin surface, fresh burrows appear as tiny grayish, sinuous lines with the adult female discernible as a whitish speck at the end of the tunnel.

Within a few hours, the female begins laying eggs in the burrow, producing two to three each day thereafter. The eggs hatch in 3–4 days. The resultant larvae often remain in the burrow for about a day before actively crawling out of the burrow onto the surface of the skin. There they excavate shallow burrows in which they moult to nymphs about 3 days later. The nymphs in turn either remain on the skin surface or dig just beneath the surface, where they molt to adults in 3–4 days. The developmental time from egg to adult typically takes about 10 days for males and 14 days for females.

Although the temporary burrows made by the larvae, nymphs, and virgin females may occur on many parts of the body, the more permanent burrows made by fertilized females tend to be in characteristic locations. The most frequent sites are folds of the skin about the wrists and in the sides of the fingers or webbing between them. Other common sites are the elbows, feet and ankles, axillae, buttocks, penis, scrotum, and female breasts. The location of burrows in infants and young children differ somewhat from that of adults, commonly involving the palms, sides and soles of the feet, and areas about the head and neck. In addition to the rash and discomfort directly associated with the burrows, rashes often occur on other parts of the body and do not correspond with the distribution of the adult female mites. These other rashes are believed to be caused in part by the shallow burrowing of the immature stages of *S. scabiei* and temporary burrows made by unfertilized females. Unlike adults, children often develop rashes on the face, chest, and back.

The most common means of transmission is by direct contact between individuals when the mites are crawling on the skin surface. However, transmission also can occur via bed linen, clothing, and other fabrics from infested hosts. The mites are able to survive 2–3 days at room temperatures when the relative humidity is more than 30%. The higher the relative humidity, the higher the survival rate. Larvae of *S. scabiei* can hatch from eggs deposited off the host and infest fomites up to 7 days. However, transmission by fomites generally is not of major importance in temperate regions. *Sarcoptes scabiei* from infested horses reportedly has been transmitted to humans via saddle blankets, harnesses, and grooming utensils.

Human Scabies

Human with scabies usually experience intense itching, especially at night. The itching typically is out of proportion to the visible signs of the infestation and tends to be aggravated by heat, warm baths, and removal of clothing. The pruritus and rash are attributed to antigens associated with the mite bodies, secretions, and fecal material deposited in the burrows. They stimulate the host's cell-mediated immune response, contributing to the development of acquired immunity to subsequent infestations by *S. scabiei* after initial exposure. This suggests the possibility of vaccines being developed to protect human and other hosts against natural infestations of scabies mites (Arlian et al., 1994a,b). The antigenic nature

of the cuticular components of the mites, their secretions, and excretory products explains the persistence of the rash and other clinical signs long after the mites themselves have been killed by acaricidal treatments.

Cases of human scabies occur in a number of clinical forms. The most common type is **papular scabies**, characterized by erythematous papules that erupt as a generalized pruritic rash on various parts of the body. The accompanying itching usually leads to scratching and excoriation of the affected areas, contributing to an eczema-like condition. Vigorous scratched lesions may become secondarily infected with pyogenic, or pus-forming, bacteria, causing an acute, inflammatory, destructive skin condition called pyoderma. In some individuals, tiny vesicles develop in the epidermis in response to burrowing mites. If they become enlarged enough to form macrovesicles, or bullae, they cause what is known as **bullous scabies**. In other cases, the patient may develop **urticarial scabies** in which a histamine-like vascular reaction produces wheals or hives that may be intensely itchy and can obscure the primary cause of the problem.

In a small number of individuals, *S. scabiei* may burrow deeper into the skin, penetrating the dermis and inducing infiltration of lymphocytes. This can lead to the formation of firm, reddish brown, pruritic masses and a condition called **nodular scabies**. The nodules tend to occur most commonly at the elbows, axillary region, groin, and male genitalia, where they may persist for months or even a year or more despite treatment. Mites seldom are recovered from nodules that are more than a month old. Cases ultimately resolve with or without therapy.

One of the more distinctive yet rare clinical types of the disease is **crusted scabies**, also called hyperkeratotic scabies. It is characterized by dry, scaly, or crusted lesions, usually on the hands and feet. Pruritus is typically mild or absent altogether, despite the extremely large numbers of mites, sometimes in the thousands, amidst the overgrowth of keratin tissue in the horny layer of the epidermis. The lack of discomfort and absence of burrows often results in these cases going undiagnosed. This condition is highly contagious and can be spread even upon casual contact owing to the large numbers of mites involved. Patients with this condition thus can serve as silent carriers and are often detected only as a result of clusters of cases of the more common forms of scabies in individuals with whom the source has come in contact, especially in hospitals and other institutional settings. Evidence indicates that the mites even can become airborne along with small scales of skin from the crusted lesions. Crusted scabies is generally associated with immunosuppressed individuals who do not respond normally to infestations of *S. scabiei*, or individuals with nervous disorders that render them insensitive to pain, especially skin sensations. They do not experience the usual itching, and their inclination to scratch is suppressed. Consequently, cases of crusted scabies often are associated with physically or immunologically compromised patients.

Despite the high host specificity of the different varieties of *S. scabiei*, many cases have been reported of humans being temporarily infested with scabies mites from other animals. Such cases are referred to as **animal scabies** and human sarcoptic mange. Although these cases usually involve dogs, particularly puppies, sources include livestock such as horses, cattle, sheep, goats, camels, and pigs. Such infestations typically result in localized erythematous papules and pruritus at contact sites. The mites do not form burrows and rarely survive to reproduce. Infestations are self-limiting and usually self-resolve within a few weeks, provided the source is removed to prevent reinfestation. The absence of burrows and low numbers of mites usually make it difficult to confirm cases by recovering mites from affected individuals. The diagnosis is thus often based on demonstrating *S. scabiei* infesting the suspected animals involved.

A diagnosis of scabies can be confirmed by demonstrating the presence of *S. scabiei*. The presence of eggs, immature stages, adults, or fecal material from the burrows are all diagnostic. The presence of burrows in characteristic locations such as the wrists, fingers, elbows, and feet are considered nearly pathognomic, i.e., by themselves virtually confirm the diagnosis. To help in locating burrows, one or two drops of ink can be applied to suspected areas and then wiped off with alcohol after 10 min. The ink is retained in the burrows, making them more discernible. Several techniques have been developed to recover mites from scabies patients for microscopic examination and identification. Adult females can be removed from the blind end of their burrows by using a sharp-pointed scalpel blade to pierce the skin and gently extract the mite. Alternatively, scrapings can be taken by vigorously scraping the affected skin several times with a sterile scalpel blade. The scraping is then transferred to a glass microscope slide for examination. Even in the absence of adult mites, the oval-shaped eggs (approximately 170 × 190 μm) are often clearly visible, as are the characteristic yellowish brown fecal pellets.

Skin biopsies can be taken and prepared for histological examination. Another method is to place skin scrapings in a small petri dish or other container and examine it after 12−24 h for the presence of mites crawling on the bottom. A centrifuge-flotation method also has been used with some success, especially in cases of crusted scabies or when abundant material from affected areas can be collected. The scrapings are placed in 10% potassium hydroxide or sodium hydroxide and gently heated. The mixture then is added to a saturated sugar solution in a centrifuge tube and spun until any mites or eggs that are

present float to the surface. Drops of the surface fluid can be examined microscopically. Eggs and eggshells have been detected by examining suspected skin scrapings in glycerine preparations using fluorescent microscopy.

The most widely used and effective means of treating scabies cases is the topical application of acaricides to the affected areas of skin. Among the more commonly prescribed acaricides are 1% lindane (gamma benzene hexachloride), crotamiton creams and lotions, sulfur applied directly to the skin or used in baths, 5% flower-of-sulfur suspended in lanolin or petrolatum, benzyl benzoate emulsions in the form of a lotion or ointment, and tetrahydronaphthalene with copper oleate. It is recommended that these materials be applied after taking a warm, soapy bath. The number of follow-up applications and the prescribed intervals vary depending on the particular product used. Overtreatment can complicate conditions and should be avoided.

In addition to treating known patients and individuals with whom they recently have had contact, fomites should be treated to disrupt possible transmission. Acaricide sprays containing pyrethrins or 5% lindane are commercially available for this purpose. Laundering clothes, bedding, towels, and other fabrics using the hot cycle of a washing machine is usually adequate to kill *S. scabiei*. Hot ironing and placing items in a freezer for 1 week also is effective in killing them. Clothing and other fomites that cannot be treated (e.g., rugs, couches) should be set aside, if possible, and not touched for 2 weeks. Any scabies mites that may have been present will have died by then.

For further information on human scabies, see Heilesen (1946), Mellanby (1972), and Orkin et al. (1977).

Human Notoedric Mange

Humans occasionally become infested with *Notoedres cati*, a sarcoptid mite that causes notoedric mange in cats. Cases in humans are called human notoedric mange or human notoedric scabies. After prolonged exposure to infested cats, people can become sensitized to this mite and develop intense pruritus within a few hours of subsequent contact with them. The reaction is induced without the mites actually burrowing. The most common sites of skin lesions are on the hands and legs, reflecting the areas most likely to come in contact with pets. The lesions subside when infested cats are either treated or removed from further contact (Chakrabarti, 1986).

Mite-Induced Allergies

Members of several families of mites can cause allergic responses in humans by direct contact of mites with the skin, inhalation of mites or mite parts, or ingestion of mite-contaminated foodstuffs. The most common sources of allergy-inducing mites are stored products and house dust.

Storage Mites

People who handle mite-infested stored products may become sensitized to the mites on subsequent contact, resulting in an immunological response called **storage-mite allergy**. Although the precise nature of the allergens is unknown, these substances include components of both live and dead mites and material produced in the mite alimentary tract. Sensitized persons may experience either contact dermatitis or respiratory allergy, depending on the type of exposure.

Allergic contact dermatitis results from exposure to mites in grains, dried fruits, flour, and other stored products, causing itching and redness at the contact sites. The families of mites most commonly involved are the Acaridae, Carpoglyphidae, and Glycyphagidae. In addition, what was probably *Dermatophagoides pteronyssus*, but reported as *D. scheremetewski* (Pyroglyphidae), has been associated with cases of feather pillow dermatitis. Contact with this mite infesting feather pillows is known to cause red papular lesions and pruritus about the scalp, eyes, ears, and nostrils (Traver, 1951; Aylesworth and Baldridge, 1983). A similar allergenic response to *D. farinae* associated with buckwheat-husk pillows has been reported in China (Hong et al., 1987).

Inhalational allergy results when airborne mites and associated allergens are drawn into the respiratory tract. The mucosal membranes lining the nasal and bronchial passages become irritated and inflamed, causing allergic rhinitis and asthma. The mucous membranes lining the eyelids also may be affected, causing conjunctivitis. These responses involve a T cell—type reaction and both immediate and delayed hypersensitivity. Such reactions to mites present an occupational hazard, especially among farmers and other agricultural workers who handle mite-infested grains and other stored materials. Among the more common storage mites that cause inhalational allergy are *Aleuroglyphus ovatus* and *Tyrophagus putrescentiae* (Acaridae), *Lepidoglyphus destructor* (Glycyphagidae), and *Blomia tropicalis* (Echimyopodidae). For further information on storage-mite allergy, see Cuthbert (1990).

House-Dust Mites

A major source of human allergens in the home is house dust and its associated mite fauna. Where humidity is sufficiently high, fungi tend to thrive in accumulated dust, providing food for a variety of house-infesting mites that are primarily saprophages or fungivores. Many of these mites are the same species that infest stored products, nests of rodents and birds, and animal litter. When their

populations reach high levels in the home, they can cause acute or chronic allergic reactions commonly known as **house-dust allergy**. The principal allergenic components in house dust are mites and mite feces, rather than the dust material itself.

As many as 10 families and 19 species of mites have been recovered from house dust in a single urban community (Tandon et al., 1988), which reflects the diversity of mites that occur in that microhabitat. The most important taxa that cause human allergy are members of the Pyroglyphidae, notably those belonging to the genera *Dermatophagoides* and *Euroglyphus*. These mites typically comprise 90% or more of the mites found in house dust. The other families of mites commonly associated with house dust are the Acaridae, Glycyphagidae, Cheyletidae, and Echimyopodidae. A member of the latter family, *Blomia tropicalis*, is often the most common house-dust mite in the Neotropics. Many of these same species also infest stored products. Four of the more common storage mites found in house dust are *Acarus siro*, *Tyrophagus putrescentiae*, *Lepidoglyphus destructor*, and *Glycyphagus domesticus* (Fig. 26.20) (Wraith et al., 1979). For a comprehensive and authoritative account of dust mites, see Colloff (2009).

The most widespread pyroglyphid species that causes house-dust allergy is the **European house-dust mite** (*Dermatophagoides pteronyssinus*), which thrives in floor dust and the surface dust of mattresses. It is regarded as the most frequently encountered house-dust mite, occurring especially in humid coastal areas of Western Europe and North America. This was the first mite to be identified as a cause of house-dust allergy, in 1966, shortly after the genus *Dermatophagoides* was first linked to house-dust and bronchial asthma. The **American house-dust mite,** *D. farinae* (Fig. 26.21), tends to be common in drier regions than does *D. pteronyssinus,* such as the more continental-type climates of central Europe and the central United States. It is a frequent inhabitant of dried animal meal (e.g., dog biscuits, poultry feed) and coarsely ground wheat. The common name reflects its more common and widespread occurrence in the United States than in Europe and other parts of the world. The third most common mite known to cause house-dust allergy is *Euroglyphus maynei* (Fig. 26.22). This is a cosmopolitan species frequently implicated in human allergy cases in Europe and Japan. It typically occurs in damper habitats than that of *D. pteronyssinus*, with which it is often associated.

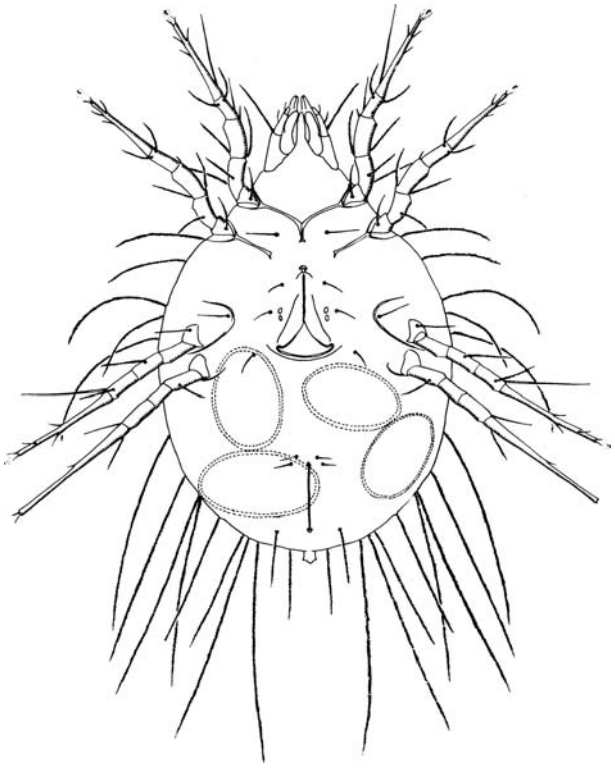


FIGURE 26.20 *Glycyphagus domesticus* (Glycyphagidae), female with four large, ovoid eggs, ventral view. *From Hughes, 1976.*

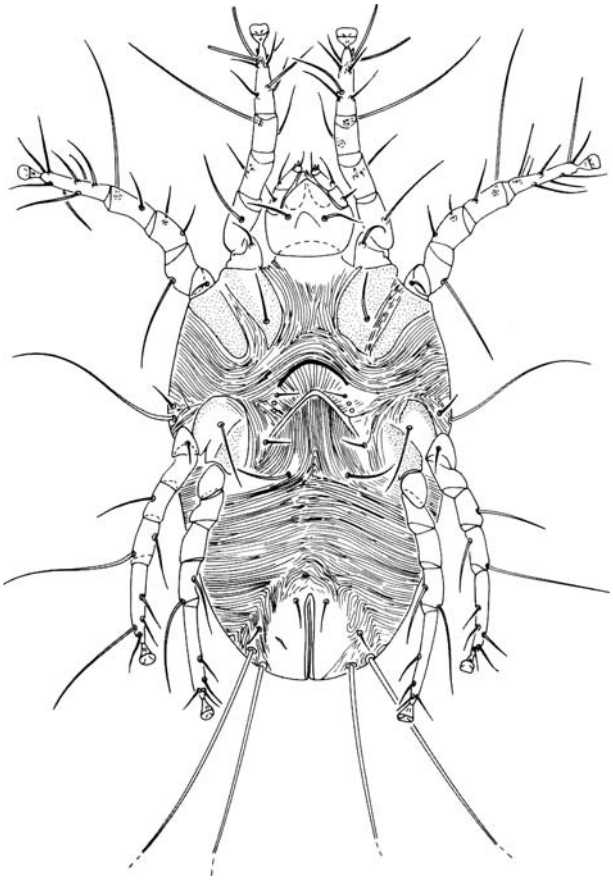


FIGURE 26.21 American house-dust mite, *Dermatophagoides farinae* (Pyroglyphidae), female, ventral view. *From Gorham, 1991; courtesy of the US Department of Agriculture.*

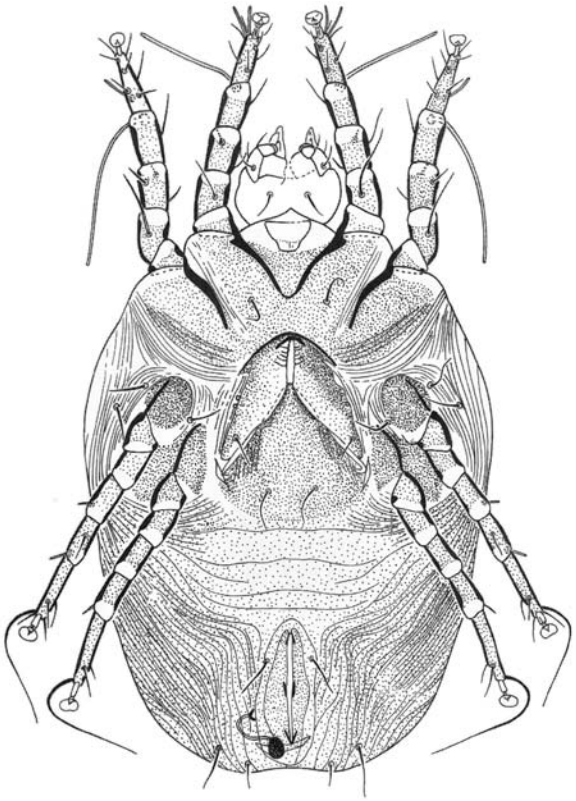


FIGURE 26.22 House-dust mite, *Euroglyphus maynei* (Pyroglyphidae), female, ventral view. *From Gorham, 1991; courtesy of the US Department of Agriculture.*

House-dust mites thrive in environments with relative humidities above 65%−70%. These mites depend on water vapor as the primary source of water that they extract from the air. They cannot actively survive more than 6−11 days at relative humidities below 50%. However, they can endure prolonged dry periods by forming desiccation-resistant protonymphs, which can survive for months below the critical humidity for the active stages. Their feeding activity, reproductive rate, and amount of fecal material generated are all directly related to humidity levels (Arlian and Hart, 1992). Populations tend to increase beginning in early summer, reach their highest levels in early fall, and remain relatively constant during the winter months, reflecting indoor humidity.

The developmental times of *Dermatophagoides* and *Euroglyphus* species vary with temperature and humidity. Under favorable conditions at room temperature and a relative humidity of 75%, they typically complete a generation in about 30 days. Females do not lay eggs unless fertilized and commonly experience multiple matings. They lay one to two eggs per day during their adult life, usually lasting 30 or more days. Half or more of the mites in dust samples may be represented by eggs; if overlooked, this often leads to underestimates of population sizes when only the nymphs and adults are counted (Colloff and Hart, 1992).

Dermatophagoides and *Euroglyphus* species are saprophages, which in the home feed primarily on skin scales shed by humans and indoor pets. Although fungi are commonly found in the alimentary tract of house-dust mites, they presumably are ingested only secondarily and are not a significant nutritional component of their diet. In fact, some fungi, such as *Aspergillus penicillioides*, actually may be detrimental to mite growth and reproduction (Hay et al., 1993). Mattresses, and sleeping quarters in general, provide particularly favorable sites for mites to develop. Human semen has been shown to be a dietary supplement for house-dust mites and can significantly increase the number of eggs that a female produces (Colloff et al., 1989).

House-dust mites occur in greatest numbers in the more humid living quarters of homes frequented by the occupants, notably wherever dust accumulates in bedrooms and living rooms. Mattresses are especially suitable, apparently owing to the accumulation of human squamous cells and other skin debris. Under optimal conditions, as many as 5000 mites have been recovered per gram of mattress dust. The type of floor can influence the species and number of mites. In damp houses, carpeted floors contribute to high populations, whereas in drier homes there may be little difference in the numbers of mites in carpeted and uncarpeted flooring. When humidity levels are high, even floors covered with linoleum and other plastic materials will support relatively high numbers of house-dust mites. In general, however, drier floors and carpets support higher populations of *D. farinae* than either *D. pteronysinnus* pteronyssinus or *E. maynei*. *Euroglyphus maynei* has the highest humidity requirements; it occurs primarily in mattresses and bedding and is the least likely to be found in carpets. Although some studies have shown that wool carpets have higher numbers of mites than do carpets made from synthetic fibers (e.g., nylon), other studies have shown no significant differences between them.

House-dust mites also may occur in fairly large numbers in other situations in the home. In a survey of household fabrics in Germany, 18% of mites recovered were found in clothing (e.g., suits) hanging in closets (Elixmann et al., 1991). The same mites may infest improperly stored food products. In one such case in Alabama (United States), an individual experienced sneezing, intense ocular pruritus, and facial edema within minutes of inhaling a puff of dry pizza dough mix heavily infested with *D. farinae* (Skoda-Smith et al., 1996).

House-dust mites are recognized as the source of 20 or more allergens that cause reactions to house dust, especially in children and young adults. The most common clinical manifestation is bronchial asthma, characterized by

difficulty breathing, inflammation of the nasal passages, and conjunctivitis. This may be accompanied by atopic eczema in some sensitized individuals. Asthmatic attacks tend to occur at night, especially in poorly ventilated bedrooms with old bedclothes and accumulated mattress and floor dust. Occurrence of symptoms is usually seasonal, reflecting the size of the mite populations. The acuteness of the allergic attacks is directly correlated with the number of mites present.

Dermatophagoides pteronyssinus generally produces the most potent house-dust allergens. However, a portion of individuals of other species, including *D. farinae*, *E. maynei*, and certain storage mites, can elicit allergic responses as great as that of *D. pteronyssinus*. Each species of mite appears to have its own species-specific antigens and allergens; differences between those are associated with the mite body and feces (scybala). There is significant cross-reactivity among the antigens of different species. This makes it difficult to determine which mite is involved in individual cases, which complicates clinical diagnosis and treatment. Diagnostic tests for mite-induced house-dust allergy include skin tests and bronchoprovocation using commercial extracts of individual mite species. Enzyme-linked immunosorbent assays and radioallergosorbent tests have been developed to help in diagnosing house-dust mite allergy cases. However, they tend to be less effective than the traditional skin-prick test in identifying people who are only mildly sensitized to mite allergens in house dust.

For further information on dust mites, the allergens they produce, their ecology, and geographic distributions, see Arlian (2002), Arlian et al. (2002), and Colloff (2009).

Several sampling techniques have been developed to determine whether house-dust mites are present in the home, and if so, what species they are and their relative numbers. Most of the techniques entail collecting samples of mattress and floor dust with a vacuum device and examining the samples microscopically for the presence of mites. Various flotation and staining methods can be used to facilitate the process. Another approach is to use a guanine test as an indirect means of determining the number of mites present. Guanine is excreted in mite feces and serves as a quantitative index of mite numbers, irrespective of the species. The amount of guanine can be measured using high-performance liquid chromatography, which provides a simple, rapid method for determining the amount of mite activity in different parts of the home (Quoix et al., 1993).

Oral Mite Anaphylaxis

A syndrome characterized by severe allergic responses after ingestion of mite-infested foods has been termed oral mite anaphylaxis (OMA), or "pancake syndrome." The latter name reflects the most commonly reported source, pancakes made with mite-infested wheat flour (Sánchez-Borges et al., 2005). This syndrome has been most commonly reported from tropical and subtropical countries, although more reports are emerging from temperate regions. Countries with the most reported cases are Spain, Japan, and Venezuela, which probably reflects the attention given this condition in those countries (reviewed in Sánchez-Borges et al., 2013).

Clinically, most reports involve adolescents and young adults, although small children and older adults can also experience symptoms. There seems to be no predominance based on gender. In all reported cases, patients had a history of atopic disease, typically rhinitis or asthma. The most commonly reported symptoms after ingestion of contaminated food are breathlessness, angioedema, and wheezing, with acute respiratory failure occurring in 4 h, occasionally leading to death (Sánchez-Borges et al., 2013). An association between OMA and cutaneous hypersensitivity to aspirin and other nonsteroidal antiinflammatory drugs has also been reported (Sánchez-Borges et al., 2012). Typically, the flour used to make the food (pancakes, beignets, and Japanese *okonomi-yaki*) is heavily mite infested (>500 mites/g). Mite species associated with this syndrome are all common storage or house-dust mites, notably *Dermatophagoides pteronyssinus* and *D. farinae*, *Tyrophagus putrescentiae*, *Thyreophagus entomophagus*, *Aleuroglyphus ovatus*, *Suidasia* spp., and *Blomia* spp.

Allergens associated with this condition have not yet been characterized, but it has been demonstrated that unlike many allergens, these are not thermolabile. Thus, cooking food does not render the allergens harmless.

Internal Acariasis

Under certain conditions of exposure, mites may enter natural body orifices, which leads to cases of temporary internal acariasis. These cases commonly involve the ingestion of mites with food and inhalation of airborne mites or mite-contaminated dust via oral or nasal routes. Mites that are swallowed or inhaled can lead to acariasis involving the alimentary tract, whereas mites that are inhaled can also invade the respiratory tract. Cases of mites infesting the urinary tract are rare. For a general discussion of the mites involved, see Ma and Wang (1992).

Pulmonary acariasis, in which mites invade the lungs, occurs most frequently among individuals exposed to mite-infested stored grains and dried herbs. This reportedly can be a serious problem among workers in grain-storage facilities and medicinal-herb warehouses in China (Chen et al., 1990; Li and Li, 1990). Clinical signs and symptoms include cough, expectorated phlegm and blood, difficulty breathing, chest pain, low-grade fever, restlessness, and marked eosinophilia. Pulmonary lesions have been

documented on x-ray film as shadows and nodular opacities in lung tissues. The following five families and nine species of mites have been recovered from sputum of affected individuals: Acaridae (*Acarus siro*, *Tyrophagus putrescentiae*, *Aleuroglyphus ovatus*, *Sancassania berlesei* (reported as *Caloglyphus berlesei*), and what was probably *Sancassania mycophaga* (reported as *Carpoglyphus mycophagus*); Pyroglyphidae (*Dermatophagoides farinae*, *D. pteronyssinus*); Tarsonemidae (*Tarsonemus granarius*); and Cheyletidae (*Cheyletus eruditus*). It is not clear which of these mites causes the more serious problems. Some species, such as *S. berlesei*, can thrive in exceptionally damp food stores covered with a film of water and may be able to survive for some time in the lungs.

A *Carpoglyphus* species (believed to be *C. lactis*) was associated with a case of pulmonary acariasis in Spain (Toboada, 1954), whereas a *Tyrophagus* species and other unidentified mites were recovered from sputum, bronchial washings, and needle-aspirated lung specimens in routine examinations of patients with respiratory ailments (Farley et al., 1989). Mite eggs, larvae, and adults were found in cytology specimens in the latter study, with evidence of their being surrounded by acute inflammatory cells in several cases.

Human cases of **enteric acariasis** occasionally are reported in which mites are found in excreta, which suggests their presence in the digestive tract. In most cases, they are acarid mites in the genera *Acarus*, *Suidasia*, or *Tyrophagus*. *Suidasia pontifica* (reported as *S. medanensis*) was recovered from the feces of a woman and two infants in Mexico (Martinez Maranon and Hoffman, 1976), whereas various stages of an *Acarus* or *Tyrophagus* species, together with eggs, were recovered from bile of a Romanian patient with chronic cholecystitis (Pitariu et al., 1978). It was concluded that the woman probably ingested the mites with her food and that the mites simply aggravated preexisting cholecystitis by causing inflammation of the digestive tract until the mites were eliminated with the bile. Other cases of enteric acariasis have been reported in children with chronic digestive disorders in Russia (Prisich et al., 1986). With the recognition of mites in foodstuffs causing oral mite anaphylaxis, it seems likely that some reports of enteric acariasis are the result of ingestion of mite-contaminated food rather than actual mite infestations.

A few cases of **urinary acariasis** have been reported, primarily involving acarid mites in the genus *Tyrophagus*. Two species allegedly recovered from the human urinary tract are *T. putrescentiae* and *T. longior* (Harwood and James, 1979). Many, if not most of these cases appear to be misleading and probably involve contamination of containers in which urine was collected or examined. A possible exception was the recovery of unidentified mites in urinary samples from several patients in Romania during acute attacks involving inflammation of the kidneys and urinary bladder (Pitariu et al., 1979). Numerous acarid mites and their eggs were observed in the urinary sediments; others were dead and encrusted with salts. Whether contamination of samples can be ruled out in these cases is unclear. Other cases of urinary acariasis have been reported in Japan (Harada and Sadaji, 1925) and China (Chen et al., 1992; Ma, 1992).

Tyrophagus longior (Fig. 26.15) occurs primarily in cool temperate regions of Europe, where it infests stored grains, hay, and straw; haystacks in open fields; cucumber plants, tomatoes, and beets; and poultry litter in broiler houses. Cases of digestive and urinary acariasis in humans involving *T. longior* have been reported (Harwood and James, 1979).

Stored-products mites in the genus *Tarsonemus* (family Tarsonemidae) have been reported to be associated with human dermatitis and other skin disorders (Hewitt et al., 1973; Krantz, 1978; Oehlschlaegel et al., 1983) and to invade various organs and body fluids of humans and other animals (Dahl, 1910). The most commonly implicated species is *T. hominis*. It is generally believed that these reports represent the contamination of glass slides and other materials used to prepare tissues for microscopic examinations (Hewitt et al., 1973; Samšinák et al., 1976).

An unusual case of large numbers of a mite (Histiostomatidae) infesting the external ear canal of a human, actively feeding and reproducing there, has been documented (Al-Arfaj et al., 2007).

MITE-BORNE DISEASES OF HUMANS

Excluding tick-borne diseases, there are only two significant diseases of humans for which mites serve as the principal vectors: rickettsialpox and tsutsugamushi disease.

Rickettsialpox

Rickettsialpox was first recognized in 1946 during an outbreak in New York City (Huebner et al., 1946). Sporadic cases had been reported as early as 1909 in Washington, DC and other cities along the northeastern seaboard of the United States. Rickettsialpox is a relatively uncommon illness; only 800–900 cases have been reported in the United States. Cases occur primarily in urban areas in crowded living quarters infested with the house mouse (*Mus musculus*), which serves as the major reservoir. The pathogen is transmitted to humans by the bite of the house mouse mite, *Liponyssoides sanguineus* (Dermanyssidae) (Fig. 26.5). Other countries in which cases of rickettsialpox have been reported are Russia, Korea, and parts of equatorial and central Africa.

The causative agent of rickettsialpox is *Rickettsia akari*. It is a spotted fever group (SFG) rickettsia and is

morphologically indistinguishable from *R. rickettsii*, the causative agent of Rocky Mountain spotted fever. The intracellular site in which it replicates in human hosts remains unknown.

Rickettsialpox is usually a mild, nonfatal illness that typically begins with the appearance of a nonpruritic, erythematous papule at each infectious bite site, usually within 24–48 h of contact with *L. sanguineus*. Soon thereafter, a small vesicle forms at the center of the papule, initially filling with a clear, then cloudy fluid. The vesicle dries, producing first a crusty lesion and then a brown or black scab, or eschar, in the center of a larger, indurated area 0.5–3.0 cm in diameter. These lesions can occur on any part of the body, but usually on the face, trunk, and extremities. They may occur on the palms and soles and on mucous membranes about the mouth. The latter include the palate and less commonly the general mouth cavity, tongue, and pharynx. Although there usually are only a few, as many as 100 discrete lesions have been reported in some cases.

Systemic symptoms appear about the time that eschars form, 9–14 days after the initial bites. Fever (usually peaking at 38–40°C), headache, and malaise are characteristic and may be accompanied by muscle aches, especially backaches, drenching sweats, and shaking chills. Less common symptoms are cough, running nose, sore throat, nausea, vomiting, enlarged and tender regional lymph nodes, and abdominal pain. Most cases resolve in 6–10 days without treatment. In some cases, however, headache and lassitude may persist for another 1–2 weeks. Treatment with antibiotics generally alleviates the fever and other symptoms within 48 h.

Diseases that should be included in the differential diagnosis of rickettsialpox are other members of the SFG rickettsiae, notably Boutonneuse fever, tsutsugamushi disease, Siberian tick typhus, and Queensland tick typhus. They can be distinguished, however, by their geographic occurrence and the clinical nature of the associated skin lesions. The nonrickettsial disease with which rickettsialpox is most commonly confused is chickenpox, which is caused by a virus. In chickenpox cases, however, the vesicles are not raised on papules, eschars are not formed, and the lesions are much more numerous. The clinical syndrome and a rise in titer of SFG-specific antibodies are generally sufficient to confirm a diagnosis of rickettsialpox. Immunity appears to be complete, perhaps lifelong, after recovery from infection. For additional information on the clinical and diagnostic aspects of this disease, see Brettman et al. (1981) and Kass et al. (1994).

Liponyssoides sanguineus, the vector of the rickettsialpox agent, is primarily a parasite on the house mouse. The mite is also found on rats (*Rattus* spp.) and voles, although the role of these and other wild rodents in the ecology of this disease is uncertain. Whereas *L. sanguineus* nymphs generally take a single blood meal, adults move onto and off the host to take several blood meals. Most of the time is spent off the host in nests and runways of mouse-infested areas. Where it occurs in human dwellings, the mite seeks the warmth of furnace rooms and incinerators of old buildings, where they may occur in large numbers on the walls and ceilings. Human bites are believed to occur primarily when house mice in apartment buildings become less attractive as hosts, inducing the mites to seek alternative hosts. The occurrence of lymphocytic choriomeningitis in house mice during outbreaks of rickettsialpox in humans has been suggested as a possible factor; such infections cause changes in a mouse's body temperature, perhaps inducing the mites to abandon their natural host (Krinsky, 1983). Starved adults can live 7–8 weeks, whereas blood-fed adults can live 9 weeks or longer.

The only other mite reported as a possible vector of *R. akari* is *Ornithonyssus bacoti*, based on experimental transmission studies using laboratory white mice (Philip and Hughes, 1948; Lackman, 1963).

Tsutsugamushi Disease

Tsutsugamushi disease is a mite-borne rickettsiosis of humans that is endemic in eastern and southern Asia, the western Pacific region, along the northern coast of Australia (Queensland and Northern Territory), and the Indian subcontinent. Cases may occur as far west as Afghanistan, Pakistan, and neighboring areas of the former Soviet Union. It is also known as **scrub typhus** and chigger-borne rickettsiosis. The causative agent is *Orientia tsutsugamushi* (formerly *Rickettsia tsutsugamushi*) transmitted by the bite of trombiculid larvae, or **chiggers** (Figs. 26.10 and 26.23).

Tsutsugamushi disease was recognized as early as the 4th century CE, when it was described in clinical manuals as an illness associated with mites. It was not until 1930, however, that Japanese workers first isolated and identified the pathogen as a rickettsia. This disease first caught the attention of the western world during World War II, when the Allied Forces were severely affected during operations in the Pacific Theater. The number of cases of tsutsugamushi disease exceeded that of direct wartime casualties among the military forces in that region. Fatality rates as high as 27%–35% occurred among troops on the islands of Goodenough and Finschhafen in New Guinea (Philip and Kohls, 1945; Philip, 1948). With the advent of effective antibiotics for treatment, the incidence of tsutsugamushi disease decreased dramatically in the region during the late 1940s and 1950s. However, sudden increases have occurred since that time in Japan

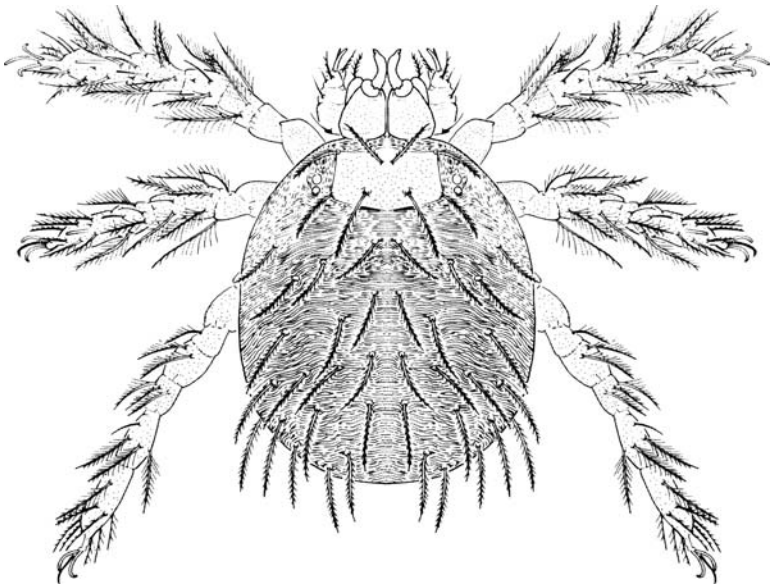


FIGURE 26.23 *Leptotrombidium akamushi* (Trombiculidae), chigger vector of *Orientia tsutsugamushi*, causative agent of tsutsugamushi disease in Japan. *Modified from Baker et al., 1956.*

(approximately 1975), Korea (approximately 1985), and other areas. For comprehensive reviews on this important mite-borne disease, see Traub and Wisseman (1974) and Kawamura et al. (1995).

The causative agent of tsutsugamushi disease is considered to be distinct enough from related rickettsial organisms to be placed in its own genus, *Orientia* (Tamura et al., 1995). Like *Rickettsia* species, it is an obligate intracellular parasite that multiplies in the cytoplasm of host cells. The clinical picture is complicated, however, by a multitude of antigenic variants, or strains, that exhibit various degrees of pathogenicity to humans. Among the better characterized strains are Gillian, Karp, Kato, Kawasaki, Kuroki, and Shimokoski. The relationships among the different strains and their mite vectors remain largely unknown.

The classic form of tsutsugamushi disease varies from a mild to severe illness. It begins with the development of a small papule at the bite site of an infected chigger. The skin reaction varies from hardly noticeable or mildly itchy to painful. The latter discomfort is characteristic of bites of the mite *Leptotrombidium akamushi* (Fig. 26.23) and has been likened to a tiny thorn that has penetrated the skin and induces pain when it is rubbed. This sensation, called *ira* in endemic areas of Japan, usually appears about 10–20 h after the bite and is believed to be caused by an inflammatory eruption associated with formation of a feeding tube (stylostome) by the attached mite. Bites occur most frequently in the folds of soft skin of the axillary region, upper legs, and abdomen. Other common sites include webs between the fingers, skin behind the knees, genitalia, under breasts, and skin constricted by clothing. The bites become ulcerated and form hard black scabs (eschars), typically accompanied by fever and a maculopapular rash.

After an incubation period of about 10 days (range, 5–20 days), symptoms generally include loss of appetite, fever, headache, muscle aches, and general malaise; regional or generalized lymphadenopathy is also common. The more virulent strains of *O. tsutsugamushi* can cause hemorrhaging, intravascular coagulation, and other blood disorders as the rickettsiae multiply in epithelial cells of the vascular system. This can lead to microthrombi in the kidneys, lungs, and heart, contributing to fatalities. Mortality varies widely from 3% to 60%, depending on the strain and geographic region. Cases of tsutsugamushi disease can be treated effectively with antibiotics. However, in severe cases with hemorrhagic complications, heparin therapy and platelet transfusion may be necessary. Immunity after recovery is not lasting, such that reinfections are common in endemic areas.

Although cases of tsutsugamushi disease occur throughout the year, they often are seasonal in certain areas, reflecting the activity of local mite vectors. In some parts of Japan, for example, cases known as **Japanese river fever** tend to occur during the warm summer months along river terraces where larvae of *L. akamushi* are present. Cases in Japan during the autumn and winter months, which may extend into late spring or early summer, are usually associated with two other chigger species, *L. pallidum* and *L. scutellare*. These seasonal differences are reflected in local Japanese names such as **Umayado disease** for the summer form of tsutsugamushi disease in Kagawa Prefecture and **Shichito fever** for the autumn–

winter form in the Hachijo Islands of Izu Shichito. The non-summer types of tsutsugamushi disease are usually characterized by relatively mild symptoms and low fatality rates. In other, subtropical and tropical regions of Southeast Asia and the southwestern Pacific, cases occur independent of seasons and are correlated with the presence of chigger vectors that are present year-round (e.g., *L. arenicola*, *L. deliense*, *L. fletcheri*).

More than 40 species of chiggers (13 genera) are known or suspected to be vectors of *O. tsutsugamushi*. The major chigger vectors of this pathogen and their geographic occurrence are shown in Table 26.3. The most important genus is *Leptotrombidium*, represented by approximately 25 vector species, most of which belong to the subgenus *Leptotrombidium*. Two or more vector species are known in each of the following four genera: *Neotrombicula* (6 spp.), *Ascoschoengastia* (2 spp.), *Euschoengastia* (2 spp), and *Walchia* (family Walchiidae) (2 spp.). Other genera that have a role in transmitting *O. tsutsugamushi* to humans are *Eutrombicula*, *Mackiena*, *Neoschoengastia*, and *Shunsennia* (family Trombiculidae), *Acomatacarus, Leeuwenhoekia*, and *Odontacarus* (family Leeuwenhoekiidae) and *Gahrliepia* (family Walchiidae).

Chiggers that serve as vectors of tsutsugamushi disease are primarily parasites of wild rodents such as field mice (*Apodemus* spp.), voles (*Microtus* spp.), and rats (*Leopoldamys*, *Maxomys*, *Rattus*, and other genera). They also occur on a wide range of birds (including pheasants, pigeons, and chickens) that are susceptible to infection and can develop at least transient rickettsemia. Although rodents serve as a source of infection of *O. tsutsugamushi* for chiggers that feed on them, it is generally believed that they have only a minor role as reservoirs and as a source of infection for the mites. Instead, the mites themselves serve as the principal natural reservoirs of the disease agent. Certain strains of trombiculid mites of a given species effectively transmit the rickettsia transovarially and/or transstadially; thus, the pathogen is transmitted from the adult female to her larval offspring and to each developmental stage that follows. Consequently, *O. tsutsugamushi* is passed from generation to generation of mite and is maintained within local mite populations in endemic areas. Were this not the case, this mite-borne rickettsia could not persist in nature. Because trombiculid mites feed on only a single host, they do not have the opportunity to transmit an acquired pathogen at a subsequent feeding. For further information on the ecology and medical aspects of tsutsugamushi disease, see Kawamura et al. (1995).

Intermediate Hosts of Human Parasites

No metazoan parasites of major health importance to humans involve mites as intermediate hosts. However, mites are hosts for a few tapeworms that occasionally infest people. Oribatid mites are intermediate hosts for two *Bertiella* species of anoplocephalan tapeworms. *Bertiella studeri* parasitizes the small intestine of a wide range of Old World primates, including rhesus and cynomolgus monkeys, Japanese macaque, baboons, mandrills, gibbons, orangutan, chimpanzees, and occasionally humans in Asia and Africa. Although several European species of oribatid mites have been shown to support the development of *B. studeri* experimentally (Stunkard, 1940; Denegri, 1985), the oribatid species involved in the natural transmission cycle remain unknown. *Bertiella mucronata*, which parasitizes monkeys in South America, develops in the oribatid mites *Dometorina suramerica*, *Scheloribates atahualpensis*, and other species of the genera *Achipteria*, *Galumna*, *Scheloribates*, and *Scutovertex* (Sengbusch, 1977; Denegri, 1985). In the case of both of these tapeworms, wild primates are the primary vertebrate hosts, with human cases occurring where infested primates live in close association with people. They cause no apparent lesions or other harm to their hosts.

Occasionally, humans may be parasitized by tapeworms of the genus *Mesocestoides* (family Mesocestoididae) that use mammalian carnivores and charadriiform birds as hosts in North America, Europe, Asia, and Africa. Oribatid mites are believed to have a role as intermediate hosts in the relatively complex life cycles of these cestodes.

Delusory Acariasis and Acarophobia

Because of their tiny size and the general lack of knowledge about mites by the general public, mites are often mistakenly blamed as the cause of skin problems or bite-like sensations when the underlying cause is unknown. The term for this is **delusory acariasis**, the imagined notion that mites are biting or infesting the skin when in fact they are not. A rational discussion is unlikely to convince individuals involved otherwise. This is a specific type of the more general phenomenon of **delusory parasitosis**. A typical example is attributing various skin conditions among office workers to "paper mites." Although there are no such creatures, it is difficult to dispel the misconception that such mites are involved. Other imaginary mites are "telephone mites" and "cable mites" blamed as the cause of skin irritation among telephone users and computer operators.

The term **acarophobia** refers to an undue fear of mites that can cause psychological stress. This may develop as the result of an actual experience with mites, or more likely as a consequence of one or more episodes of delusory acariasis.

VETERINARY IMPORTANCE

Mites have been successful in exploiting all groups of vertebrate hosts except fish. Many are ectoparasites of skin,

scales, feathers, or fur, whereas others are endoparasites that have invaded body cavities, respiratory passages, and internal tissues and organs. Some mites are vectors of disease agents of domestic and wild animals, whereas still others serve as intermediate hosts for animal parasites, notably tapeworms. Occasionally, mites are the cause of allergic reactions of pets and other animals. For overviews of mites of veterinary importance, see Hirst (1922), Baker et al. (1956), Strandtmann and Wharton (1958), Sweatman (1971, 1984), Yunker (1973), Georgi (1980), Whitaker (1982), Nutting (1984), and Pence (1984). For works of a more regional nature, see Domrow (1988, 1991, 1992) for Australia; Mulla and Medina (1980) for South America; and Cosorabã (1994) for Eurasia.

Mite-Induced Dermatitis

Four families of ectoparasitic mites commonly cause irritation when they bite host animals to feed on blood, lymph, or skin tissues. Three families are mesostigmatid mites: Dermanyssidae, Macronyssidae, and Laelapidae. The fourth is the prostigmatid family Trombiculidae (chiggers).

Dermanyssidae

Most dermanyssid mites cause relatively little harm even to heavily infested hosts. *Dermanyssus hirudinis*, for example, has been observed to cause little adverse effects on the survival, growth, or health of house wrens (*Troglodytes aedon*) infested with hundreds or thousands of mites per nestling (Johnson and Albrecht, 1993). Similarly, *D. americanus* and *D. gallinae* seldom cause problems when infesting wild avian hosts. *Dermanyssus gallinae*, however, commonly causes dermatitis in atypical avian hosts and domestic mammals and can cause severe infestations and economic losses in domestic chickens.

Chicken Mite (Dermanyssus gallinae)

This mite (Fig. 26.4), also known as the **red poultry mite** and **pigeon mite**, is an obligate parasite of wild and domestic birds worldwide, including chickens, pigeons, canaries, parakeets, house sparrows, and starlings. Occasionally, it infests dogs, cats, horses, cattle, rodents, rabbits, and other mammals. Cases involving domestic animals usually occur in association with poultry houses or infested bird nests.

The chicken mite is especially a problem in poultry operations. It hides by day in crevices and nesting materials, moving onto the birds to feed at night. Skin lesions in chickens are usually inapparent but may occur as erythematous papules on any part of the body. Chronic or heavy infestations can be debilitating and result in skin irritation, loss of vigor, stunted growth, reduced egg production, anemia, and death owing to exsanguination. Newly hatched chicks are particularly vulnerable. Setting hens may be driven from their nests and susceptibility to disease agents may be significantly increased.

Dogs exposed at night to *D. gallinae* around poultry houses may react adversely to their bites. In addition to developing intense pruritus, they remain awake or howl at night, become less active during the day, and may show signs of depression. Skin lesions include erythematous papules, hyperpigmentation, and scaling, accompanied in some cases by partial loss of hair and slightly enlarged lymph nodes. Removal of an animal from the source of mites usually results in prompt alleviation of symptoms.

Dermanyssus gallinae can develop from egg to adult in as few as 5 days, completing its life cycle in 9–10 days. Mating and oviposition occur off the host. The eggs typically are deposited in groups of four to seven eggs, with a given female producing a total of 20–24 in her lifetime. The mites usually engorge to repletion at one feeding and lay their eggs at approximately 3-day intervals. The eggs hatch in 1–2 days to produce larvae that do not feed. Like adults, the protonymphs and deutonymphs feed on blood. The adults can endure starvation for several months, which enables them to survive extended periods in abandoned bird nests and unoccupied poultry houses.

Macronyssidae

Most members of the Macronyssidae are obligate parasites of vertebrates. As a group they appear to have evolved on bats, from which they have secondarily transferred to other mammals, reptiles, and birds. Although most macronyssid mites cause little or no apparent harm to their bat hosts, *Chiroptonyssus robustipes* has been known to cause the death of heavily infested, captive Brazilian free-tailed bats (*Tadarida brasiliensis*). This mite attaches primarily to the wings, where its feeding can result in increased vascularity and edema at the bite sites, enlargement of lymphatic vessels, hyperkeratosis, and excoriation of the stratum corneum (Sweatman, 1971).

A few macronyssid mites have invaded the oral mucosa of pollen-feeding and fruit-feeding bats (Phyllostomidae). All are members of the genus *Radfordiella* (Fig. 26.24). In heavily infested bats, they can cause bone damage to the hard palate and destruction of gingival tissues, resulting in loss of teeth. The damage is caused by protonymphs. The adult mites presumably are nidicolous and move onto the host only intermittently to feed on parts of the body other than the mouth (Phillips et al., 1969).

Tropical Rat Mite (Ornithonyssus bacoti)

The tropical rat mite (Fig. 26.6) occurs throughout the world, where it parasitizes primarily rodents. It

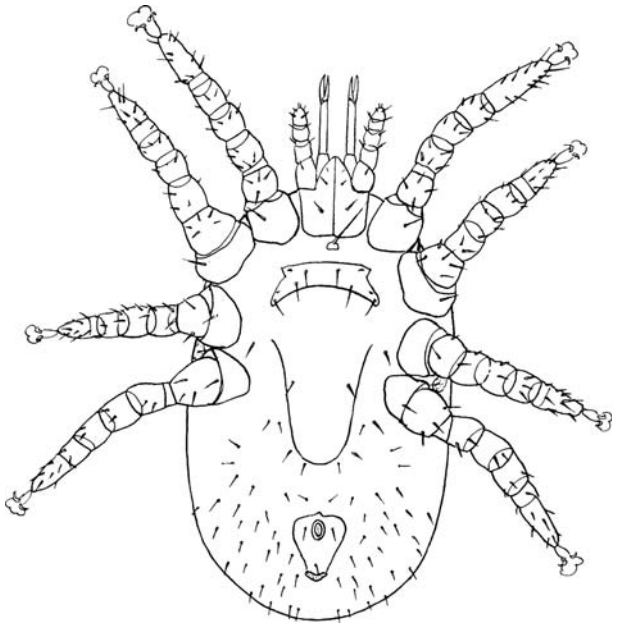


FIGURE 26.24 *Radfordiella oudemansi* (Macronyssidae), female, ventral view. *From Strandtmann and Wharton, 1958.*

occasionally infests cats, wild carnivores, chickens, and other birds, as well as humans. Most veterinary problems involving this mite occur in laboratory mice, rats, and hamsters. Heavily infested animals may become debilitated and anemic or experience reduced reproductivity; death can occur in some cases. Infestations usually are recognized by the presence of blood-engorged protonymphs and adults in the animal bedding, cages, and corners or crevices of cage racks. The tropical rate mite is a vector of *Litomosoides carinii*, a filarial nematode in cotton rats (see the section "Mite-Borne Diseases").

Blood-fed females lay their eggs in bedding or nest debris of their hosts and in cracks and crevices. The eggs hatch in 1–4 days, producing larvae that molt to protonymphs about 1 day later without feeding. The protonymphs feed on blood, molting to deutonymphs in 1–2 weeks. Deutonymphs, which are morphologically regressive, molt to adults after 1–2 days without feeding. Development from egg to adult can be completed in 11–16 days under favorable conditions. Adults usually mate within 1–2 days after emergence and can survive several days to a few weeks without a blood meal. The tropical rat mite is found primarily in rodent nests, moving onto the host animals only to feed.

Tropical Fowl Mite (Ornithonyssus bursa)

The tropical fowl mite (Fig. 26.7) is a common ectoparasite of wild and domestic birds throughout the warmer regions of the world. Domestic or peridomestic birds that may be infested include chickens, ducks, pigeons, starlings, house sparrows, and canaries. Most infestations originate from contact with wild birds or infested nest materials. Heavy infestations in chickens and other domestic fowl can result in anemia, decreased weight gain and egg production, and occasionally death. Newly hatched chicks and young birds are especially vulnerable. Blood feeding by this mite causes skin irritation that may be intense enough to induce setting hens to leave their nests. Inspection of the plumage will reveal the mites in the down feathers, particularly around or just below the vent. Infested feathers are soiled or dirty in appearance owing to the accumulation of mites, exuviae, eggs, and excreta. In the case of young birds, the mite commonly occurs around the eyes and beak.

Ornithonyssus bursa may cause more problems for wild birds than previously suspected. In Denmark, for example, nest infestations of the barn swallow (*Hirundo rustica*) have been shown to decrease reproductive success by reducing clutch sizes, nesting periods, and number of fledglings; and lengthening the time between clutches and the incubation period (Möller, 1990). Barn swallows regularly reuse old nests but tend to avoid nests infested with this mite.

The tropical fowl mite lays its eggs on the host or in nesting materials, where they hatch in 2–3 days. The larvae do not feed, whereas the protonymphs and adults feed intermittently on host blood. Although relatively few details about its life history have been reported, this mite is believed to develop from egg to adult in 6–8 days. In the absence of a host, it can survive about 10 days.

Northern Fowl Mite (Ornithonyssus sylviarum)

The northern fowl mite (Fig. 26.8) is a major pest of chickens and other domestic fowl, particularly in temperate regions of North America and Eurasia. It also is an economic pest of domestic fowl in Australia, New Zealand, and other parts of the world where it has been introduced. In addition to infesting chickens, it is commonly found in the nests of pigeons and various wild birds and as an incidental pest biting rodents such as hamsters, and humans.

The greatest economic impact occurs in chicken houses. Initial infestations usually occur via wild birds or newly acquired chickens already infested with the mite. They can then spread throughout even the largest houses within a few weeks. The northern fowl mite causes problems similar to those caused by *O. bursa*. These include skin irritation without apparent lesions at the bite sites; matted and grayish feathers, especially around the vent where the mites, their exuviae, and feces are concentrated (Fig. 26.25); scaly, scabby or thickened skin; and general loss of thriftiness. Individual caged birds may have 10,000 or more mites. Heavily infested birds often become anemic, experience decreased weight gains and egg production, and

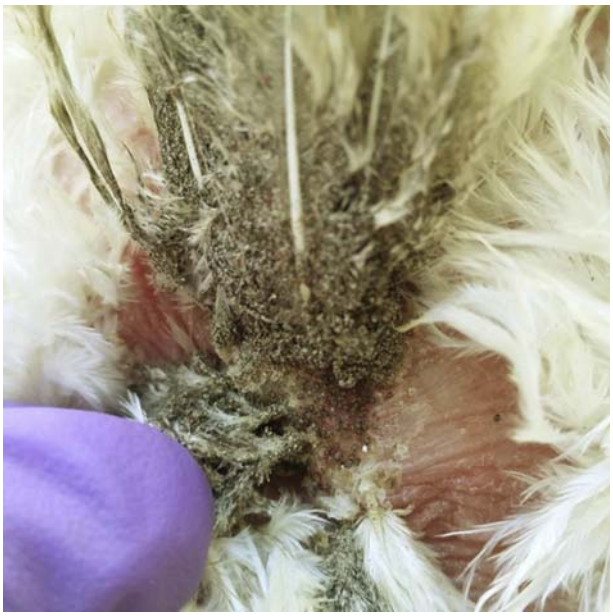


FIGURE 26.25 Northern fowl mite, *Ornithonyssus sylviarum* (Macronyssidae); heavy infestation of chicken in vent area. *Photograph by Amy C. Murillo.*

sometimes die. Egg shells may become significantly thickened, and egg production may drop 5%–15% compared with uninfested birds. The greatest effect on body weight and efficiency of feed conversion generally occurs when hens are infested with mites before they reach full egg production. Although pathogens of poultry, such as viruses that cause Newcastle disease and fowl pox, have been recovered from *O. sylviarum* after feeding on infected chickens, there is little or no evidence that the mite transmits these agents when it bites.

Northern fowl mites spend most of their time on the host. However, they also occur in nesting materials and nest debris, roosting areas, and cracks and crevices in the floors and walls of chicken houses, where they can be found during the day or night. Oviposition usually occurs on the host about 2 days after complete engorgement with blood. Eggs typically are laid two to three at a time, but there may be as many as five. The eggs hatch in about 24 h. The protonymph, the only immature stage that feeds on blood, requires at least two feedings before it molts to produce the deutonymph. The complete life cycle is typically 5–7 days, enabling populations to build rapidly. Survival time without a host is usually 3–4 days but may be as long as 2–3 weeks.

Snake Mite (Ophionyssus natricis)

This mite (Fig. 26.9) is a common parasite of captive snakes, on which it is usually found around the eyes and under chin scales. It also may be found on captive lizards and other reptiles. The sources of infestation are usually caged snakes in pet stores, zoos, and biological laboratories. Although *O. natricis* occasionally is found in low numbers (<100/host) on wild snakes, its populations on captive snakes may reach hundreds or thousands per host. Heavy infestations cause listless behavior, loss of appetite, anemia, skin irritation, loss of scales, and in some cases, death. Several hundred mites are sufficient to cause severe anemia and other symptoms.

Ophionyssus natricis is a vector of the bacterium *Aeromonas hydrophila*, which causes hemorrhagic septicemia in snakes. Infected snakes hemorrhage internally and often die 3–4 days after infection.

The snake mite lays its eggs in crevices and debris and on rough surfaces of cages, where they hatch in 2–4 days. The larvae and deutonymphs do not feed, whereas the protonymphs and adults are obligate parasites that feed exclusively on blood. The life cycle usually is completed in 2–3 weeks at room temperature, with adult females living 5–6 weeks. Females typically feed two or three times, depositing about 20–25 eggs after each blood meal. See Camin (1953) for further details on the biology and behavior of this mite.

Laelapidae

Laelapid mites are commonly associated with rodents, other nest-building mammals, and bird nests. Those of medical or veterinary concern belong to the subfamilies Laelapinae, Hemogamasinae, and Hirstionyssinae, the latter two of which are sometimes accorded family rank. The first two groups include both facultative and obligate parasites that generally cause little or no apparent harm to their hosts, whereas all Hirstionyssinae are obligate parasites. The genera most commonly encountered by veterinarians are *Androlaelaps*, *Haemogamasus*, *Laelaps*, and *Echinonyssus*. *Haemogamasus liponyssoides* is an opportunistic blood feeder that inhabits nests of rodents and shrews. Its slender, chelate chelicerae are capable of piercing skin to feed on blood, enabling this mite to feed on laboratory mice if they become infested.

Spiny Rat Mite (Laelaps echidninus)

The spiny rate mite occurs throughout most of the world as an ectoparasite associated primarily with the black rat and Norway rat. Occasionally it is found on *Sigmodon* and other *Rattus* species, the house mouse, and various domestic and wild rodents. It is rarely found on laboratory animals. This mite is generally easy to recognize by its large body size (about 1 mm long), heavy sclerotization, and long, stout body setae that give it a spiny appearance. It lives primarily in host bedding or nesting materials, moving onto the host at night to feed. Its chelicerae are not capable of piercing intact skin but instead assist the mite in feeding on lachrymal secretions and blood or serous exudates from abraded skin. Rarely does its feeding cause discernible lesions, although injury to the footpads of suckling mice has

been reported. The spiny rat mite is a natural vector of *Hepatozoon muris*, a blood protozoan of rats.

Regular blood meals are required for *L. echidninus* to survive and reproduce. Blood-fed females give birth to live larvae, which do not feed. The protonymphs and deutonymphs both apparently feed similarly to adults, completing their development to adults in 1−3 weeks. The length of their life cycle is variable, requiring at least 16 days. The females can live 2−3 months; without food, however, they survive only about a week.

A smaller species, *Laelaps nuttalli*, is also commonly found on commensal and wild *Rattus* species. The life cycle is presumably similar to that of *L. echidninus*.

House Mouse Echinonyssus (Echinonyssus butantanensis)

This species is associated with the house mouse, *Mus musculus*, in many parts of the world. It has also been referred to as *Hirstionyssus latiscutatus*, *H. musculi*, and *H. orcadensis* (Herrin, 1974). The species is an obligate hematophage in its protonymphal, deutonymphal, and adult stages. It is a common pest of laboratory mice and has been observed to colonize other rodent species, especially in captivity. It can be a serious pest in rodent houses in zoos, where house mouse infestations occur.

Trombiculidae, Leeuwenhoekiidae, Walchiidae

Although not widely recognized as a problem, larvae of three families of trombiculoid mites (**chiggers**) commonly infest domestic animals. They are likely to be brought to the attention of veterinarians only in cases of heavy infestation or sensitivity reactions. As in humans, the resultant dermatitis is a response to chiggers that are normally parasitic on other host animals. They attach to atypical hosts such as cats, dogs, sheep, other livestock, and occasionally to domestic or pet birds only incidentally. Most cases involve mild pruritus and are likely to go unnoticed. Cases of heavy infestations, however, can result in severe itching with formation of vesicles and crusty or scabby skin lesions, usually about the head and neck. Large numbers of engorged chiggers may be visible as orange patches associated with the lesions. The chiggers typically remain attached only up to 2−3 days. Treating the lesions with acaricides and preventing secondary infection usually resolves the problem if the animal is not reinfested.

Some chiggers enter the skin via large hair follicles, crawling down the shaft of the hair, sometimes well beneath the skin surface. An extension of the stylostome, or feeding tube, may extend backward around the mite to form a hyaline capsule. Usually these capsule-forming chiggers are completely intradermal and may cause localized inflammation and edema. In some cases, they induce formation of cysts at the base of the hair follicles that can lead to secondary infections and slow-healing lesions. Intradermal chiggers include members of the genera *Cheldonta*, *Euschoengastia*, *Guntherana*, *Intercutestrix*, and *Schoutedenichia* (family Trombiculidae), *Apollonia* (Family Leeuwenhoekiidae), and *Gahrliepia* (family Walchiidae) (Sweatman, 1971). These mites occur primarily in Southeast Asia, the South Pacific Islands, Australia, Africa, and other parts of the Old World, although some are known from the New World as well. Rodents, shrews, and bandicoots are some of their more common natural hosts.

A few species of chiggers have been identified as the cause of trombiculosis in domestic cats. In the United States, *Ripiaspichia (=Walchia) americana* (family Walchiidae) is known to cause papules on the face, ears, and thoracic areas of cats, in addition to thickening and crusting of the skin on the abdomen and legs. This is accompanied by hyperkeratosis, eosinophilia, and infiltration of mast cells, as evidenced in skin biopsies at the lesion site (Lowenstine et al., 1979). Large numbers of *Eutrombicula alfreddugesi* have been observed as distinct orange patches on the head and ears of a cat in North Carolina (United States), causing inapparent dermatitis (Hardison, 1977). Other chiggers known to infest cats are *Leeuwenhoekia adelaidiae*, *L. australiensis*, *Schoengastia philippinensis*, and *S. westraliensis* in Australia. Natural hosts for these mites include wallabies, gray kangaroos, and wild pigs (Wilson−Hanson and Prescott, 1985). Other cases in cats involving unidentified chigger species have been reported in Australia. Lesions occurred as pinpoint erythemas and orange crusts on the ears (pinnae), pruritus, papules, and orange crusty lesions about the eyes and face; conjunctivitis, and ocular discharges. In one case, swelling and irritation of the perineal region with concomitant inability to pass urine was attributed to trombiculid mites (Wilson-Hanson and Prescott, 1985).

Dogs appear to be less commonly bothered by chiggers. Bite reactions are similar to other host animals, with localized redness, pruritus, and development of papules or vesicles at the bite sites. Cases involving heavy infestations may warrant veterinary attention. In Europe, the harvest mite (*Neotrombicula autumnalis*) reportedly caused nervous symptoms in dogs, including partial paralysis of the limbs and lameness (Prosl et al., 1985).

Virtually all species of livestock are subject to chigger infestations while grazing, walking paths to and from barns, being held in enclosures, or by contact with recently harvested hay or grains infested by these mites. Skin lesions in the form of papules or crusty eruptions can be irritating and lead to self-inflicted skin damage as the host animal rubs and abrades the affected areas. Lesions occur primarily on the lips, muzzle, face, feet, and belly. Pigs have developed

a generalized pruritus after feeding on fresh chigger-infested grains from automatic feeders. Sheep, goats, and cattle are particularly prone to infestations with *Neotrombicula autumnalis* in Europe during the harvest season, causing pruritus, scabs, and loss of hair, particularly about the head and neck. In Australia, sheep experienced severe dermatitis on the legs and feet caused by infestations by *Eutrombicula sarcina*, a chigger that normally parasitizes kangaroos. An orf-like condition in sheep caused by a *Guntheria* species has been reported during the summer months in South Africa (Otto and Jordaan, 1992).

Domestic birds such as chickens may become parasitized by chiggers (e.g., *Neoschoengastia americana*), leading to itching and dermatitis. In most cases, the mites can be found under the wings or around the vent. Reports of anemia in chickens attributed to heavy infestations of chiggers should be treated with skepticism; chiggers feed on dermal tissues and not on blood. Occasionally, other captive birds may be affected. A chronic infestation of canaries by an unidentified trombiculid mite has been reported in Australia. Canaries in a commercial aviary developed nonpruritic subcutaneous swellings of the legs and ventral trunk, with acute inflammation and skin necrosis at the sites of mite attachment (Pass and Sue, 1983).

Wild animals generally do not show adverse reactions to chiggers even when heavily infested. Occasionally, however, they react severely to bites of certain species that normally parasitize other hosts. Reactions include formation of vesicular or crusty lesions, slow-healing eschars, localized skin discoloration, and some loss of hair. Examinations often reveal orange or red clusters of mites about the head, ears, neck, axillae, or groin. Sometimes infestations of chiggers about the eyes cause ocular lesions in the form of pruritic eyelids and conjunctivitis. Snakes, lizards, skinks, and other reptiles are parasitized by chiggers, most commonly noticed as orange or red clusters of mites on the head and neck. Many lizards have specialized "mite pockets," invaginations of the skin in the axillae or nuchal region, which harbor colonies of chiggers. The host seldom shows apparent harm, even in cases of individual snakes infested with several thousand mites.

Fur Mites

Certain families of mites are categorized as fur mites because they are specially adapted for living in the hair coat of mammalian hosts. They often exhibit striking modifications of the palps, legs, and other body structures for grasping or clinging to hair. The six groups of particular veterinary interest are the cheyletoid families Cheyletidae (including the former family Cheyletiellidae) and Myobiidae, and the astigmatid families Listrophoridae, Atopomelidae, Chirodiscidae, and Myocoptidae.

Cheyletidae

Although most cheyletids are predatory, parasitic cheyletid mites occur on domestic cats, dogs, and rabbits, as well as many wild mammals and birds. They are nonburrowing mites that live in the pelage of their hosts and feed on lymph and other tissue fluids by piercing the epidermis with their stylet-like chelicerae. The enlarged gnathosoma and pair of large, terminal palpal claws give cheyletid mites a characteristic appearance. These structures are used to secure the mites to their hosts and assist them in inserting their chelicerae. Members of the genus *Cheyletiella* can cause problems that warrant veterinary attention. Although most cases of cheyletiellosis go unnoticed, infestations of these mites can cause eczema-like skin conditions, or **cheyletid mange**, with associated pruritus and hair loss. Three *Cheyletiella* species of veterinary importance are *C. blakei* of cats (Fig. 26.26), *C. yasguri* of dogs, and *C. parasitivorax* of rabbits (Fig. 26.27). All developmental stages of these mites occur on the host animal. The eggs are glued to hairs but can be dislodged with loose hairs by host grooming. They also can be ingested and passed in the feces. The presence of *Cheyletiella* eggs in cat and dog feces thus serves as evidence of mite infestations even in asymptomatic cases (Fox and Hewes, 1976; McGarry, 1993). Transmission is usually by direct contact with infested animals, including maternal transfer while nursing. Because *Cheyletiella* species can survive up to 10 days or more off a host, animal bedding, household furniture, blankets, and carpets frequented by pets can serve as other sources of these mites. *Cheyletiella* mites are commonly phoretic on cat and dog fleas (*Ctenocephalides* spp.) and also may be transmitted via these ectoparasites.

Cheyletiella blakei usually infests the facial area of cats. Heavy infestations can result in the formation of small, crusty, erythematous papules and loss of hair, accompanied by itching and scratching. Long-haired cats tend to be more commonly infested than short-haired cats and are more likely to be involved in human cases of cheyletiellosis. *Cheyletiella yasguri* parasitizes domestic dogs, particularly in Europe and North America. It is generally less common than *C. blakei* and only occasionally causes problems warranting veterinary attention. Signs of an infestation are scratching and a mealy or powdery dandruff in the affected areas, commonly the lower back. Heavy infestations can cause scaling, hyperkeratosis and thickening of the skin, erythema, pruritus, and hair loss. Puppies tend to have a higher incidence of *C. yasguri* than do adult dogs and are more likely to exhibit pruritus. This mite can cause dermatitis in humans upon close contact with infested dogs, especially puppies (Fig. 26.28). Acaricidal treatments of dogs and their surroundings are effective in controlling *C. yasguri*. This is

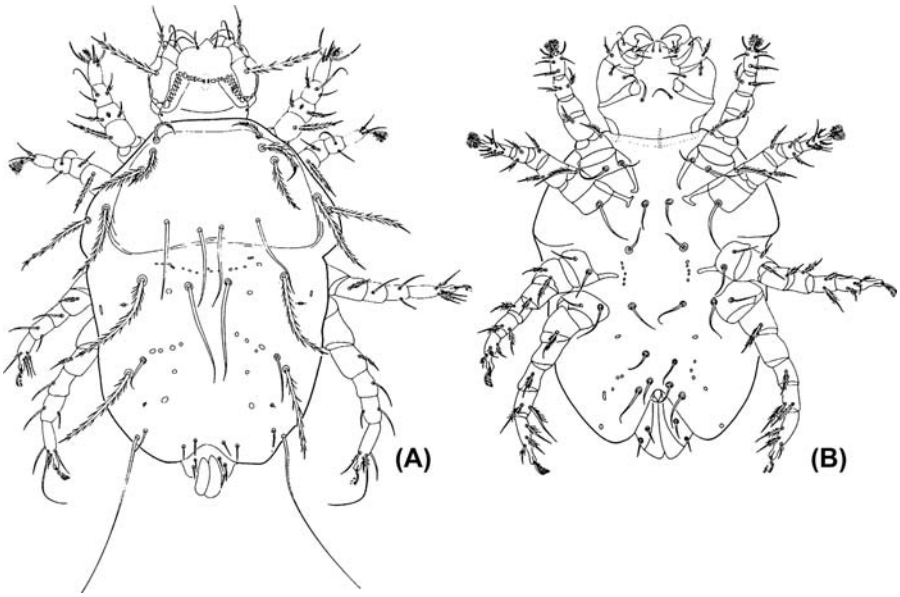


FIGURE 26.26 *Cheyletiella blakei* (Cheyletiellidae), female. (A) Dorsal view; (B) ventral view. *Modified from Domrow, 1991.*

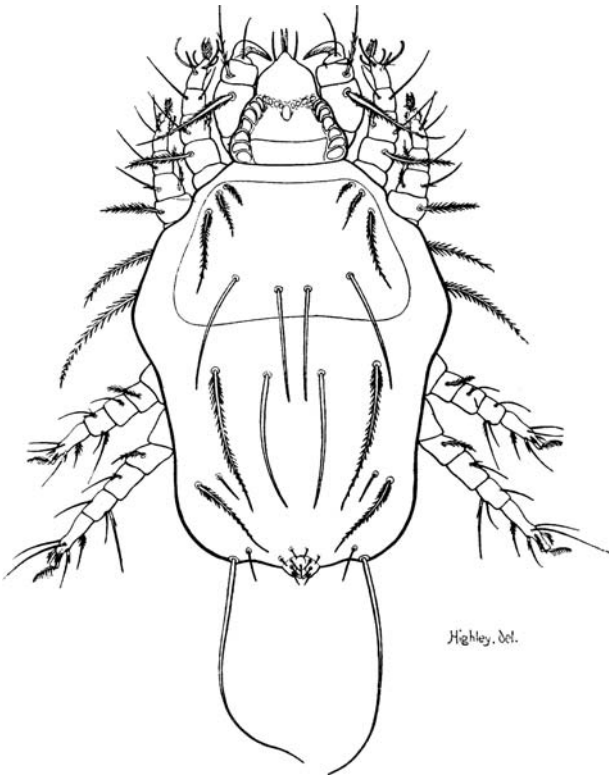


FIGURE 26.27 Rabbit fur mite, *Cheyletiella parasitivorax* (Cheyletidae), female, dorsal view. *From Hirst, 1922.*

especially important in kennels, which serve as a common source of mite infestations.

Cheyletiella parasitivorax, the **rabbit fur mite**, is a common parasite of the European rabbit (*Oryctolagus cuniculus*) in North America, Europe, Asia, Australia, and New Zealand. It occurs most frequently in the posterior-back region of infested hosts, but also may occur on the face, frontal area, and other parts of the body. High mite populations induce the accumulation of epidermal scales and a scurfy appearance, leading in untreated animals to varying degrees of dermatitis, erythema, thickening of the skin, and hair loss. Severe cases may involve serous exudates and hairless patches in which the mites can be found in the disrupted keratin layer amidst epidermal debris. Infestations of *C. parasitivorax* are particularly a problem in commercial rabbit colonies and laboratories where rabbits are closely confined. Wild rabbits seldom exhibit cheyletiellid mange. *Cheyletiella parasitivorax* is capable of transmitting myxomatosis virus among European rabbits in Australia.

For additional information on *Cheyletiella* species and their veterinary importance, see Smiley (1970) and van Bronswijk et al. (1976).

Myobiidae

Members of the family Myobiidae are obligate parasites of rodents, bats, insectivores, and certain marsupials. They typically grasp the hairs of their host with their forelegs, which are typically highly modified for this purpose. The mites move up and down the hair shaft and remain clinging to the hairs as they feed on epidermal fluids. Their chelicerae are long and stylet-like and adapted for puncturing thin epidermal tissues to feed on extracellular fluids. A few species, however, are known to feed on blood (e.g., *Blarinobia simplex* on shrews and *Eadiea brevihamata* on the shrew-mole). Most species cause little apparent discomfort or harm to their hosts,

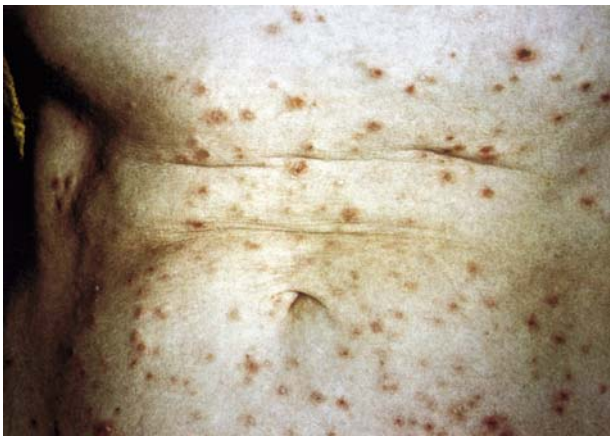


FIGURE 26.28 Multiple lesions from bites of *Cheyletiella yasguri* (Cheyletidae) on abdomen of woman after contact with infested puppy. *From Southcott, 1976.*

even when mite populations are high. Exceptions of veterinary interest are a few *Myobia* and *Radfordia* species that commonly infest rats and mice, often causing mild to severe dermatitis and scurfiness in laboratory rodents.

The most widely recognized myobiid is the **mouse fur mite**, *Myobia murismusculi* (Fig. 26.29), often incorrectly referred to as *Myobia musculi*. This is a cosmopolitan, ubiquitous species that infests the pelage of both wild and captive house mice (*Mus musculus*). Most of what is known about the development and biology of myobiids is based on this species. Females deposit their eggs singly, gluing them to the bases of hair shafts. The developmental time from egg hatch to adult is about 23 days. All stages, including the larvae, feed on dermal tissue fluids. The host response varies greatly, depending on the strain, sex, age, and sensitivity differences of individual mice. Lightly infested hosts are often asymptomatic or exhibit little adverse reaction. In highly sensitive hosts, however, even a few mites can elicit allergic reactions and severe pathologic responses. Heavy infestations can lead to severe dermatitis, with intense pruritus, hair loss, self-inflicted trauma from scratching, and in some cases, death. This can be a particular problem in laboratory mice colonies where infestations are likely to involve virtually all individuals.

Two *Radfordia* species occur worldwide, infesting the fur of wild and laboratory rodents. The more common is *R. ensifera* parasitic on rats (Fig. 26.30); the other is *R. affinis* on the house mouse. They closely resemble *Myobia murismusculi*, from which they are distinguished by a pair of tarsal claws (rather than one claw) on the second leg. Although they generally cause little pathologic effect, dermatitis and self-inflicted trauma have been associated with heavy infestations of *R. ensifera* on laboratory rats.

Listrophoridae

Listrophorid mites (Fig. 26.31) are obligate parasites of rodents, lagomorphs, carnivores, and other mammalian

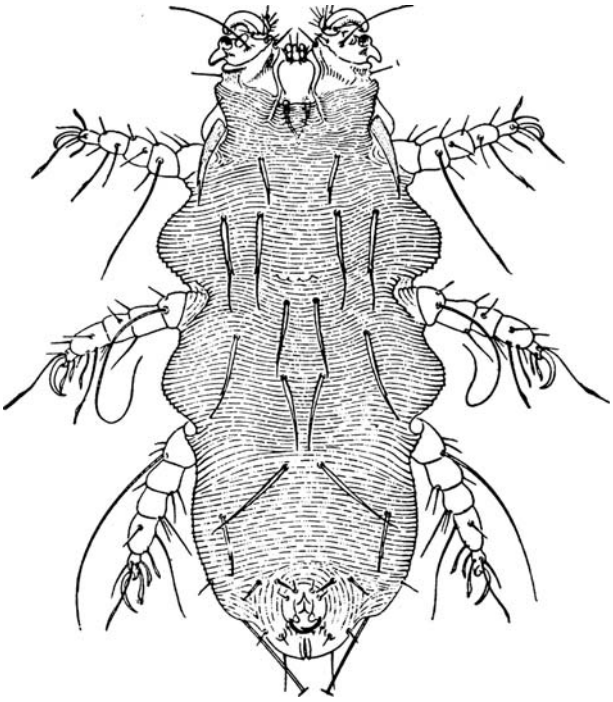


FIGURE 26.29 Mouse fur mite, *Myobia murismusculi* (Myobiidae), female, dorsal view. *From Baker et al., 1956.*

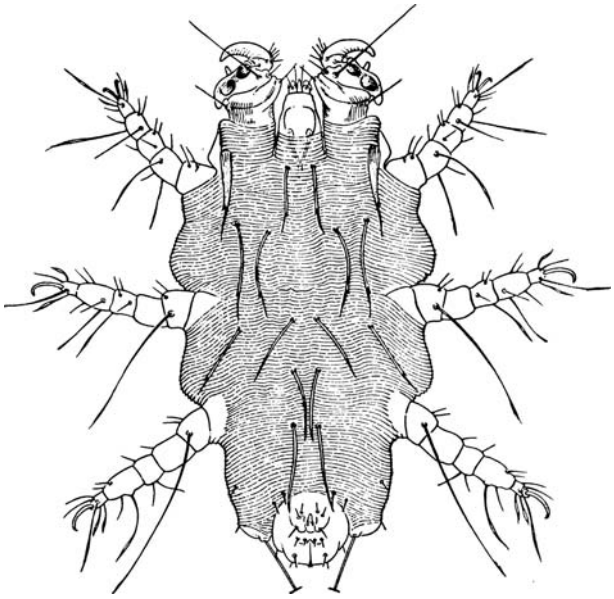


FIGURE 26.30 *Radfordia ensifera* (Myobiidae), female, dorsal view. *From Baker et al., 1956.*

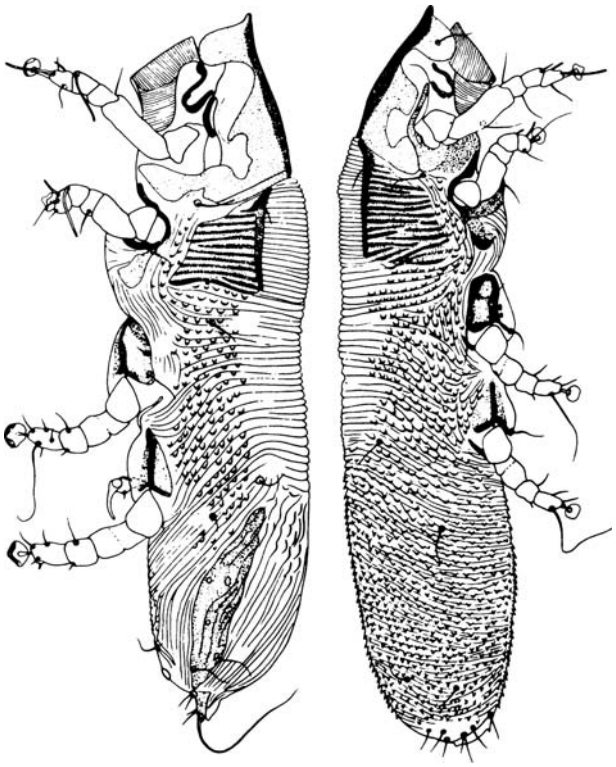


FIGURE 26.31 *Listrophorus synaptomys* (Listrophoridae), male (left), female (right), lateral views. *From Whitaker, 1982; after Fain et al., 1974.*

hosts in the Old and New Worlds, although none are native to Australia or Madagascar. They are well-adapted for clasping securely to hair shafts, with appendages modified for this purpose. They attach so firmly that they are difficult to remove and can damage the hairs by bending or crimping them. They feed primarily on sebaceous secretions, usually causing little or no apparent harm even when mite numbers are high. Exceptions occur, however, in rodents and rabbits, especially under conditions of confinement or crowding. Heavy infestations in such cases can result in dermatitis, scratching, and hair loss.

Members of the family are generally cylindrical to laterally flattened and are distinguished by the anterior coxal fields that are expanded and flattened, with grooved surfaces; they serve as attachment organs, along with the anterior legs, for grasping host hairs. Rabbits infested with *Leporacarus gibbus* may experience pruritus, hair loss, skin abrasion caused by scratching, and occasionally damage to the fur as a result of nibbling. Wild populations of *Rattus* and *Mus* harbor species of *Afrolistrophorus* in tropical regions, but these have not been reported from laboratory populations. *Lynxacarus radovskyi*, which infests domestic cats, has been reported to cause patches of mange and a scurfy appearance of cats in Hawaii (Tenorio, 1974).

Atopomelidae

Atopomelid fur mites are parasites of marsupials, rodents, insectivores, and primates, primarily in the Southern Hemisphere. They attach to hair shafts with their anterior two pairs of legs. They are distinguished from the Listrophoridae by the lack of a projecting tegmen above the gnathosoma, and the striated forecoxal fields do not project around the host's hair.

Guinea pigs are commonly infested with *Chirodiscoides caviae*, particularly in laboratory colonies. This mite usually attaches to hairs on the back but may occur on any part of the body. Attachment to hairs is facilitated by their striated sternal area and legs I and II, which are flattened and curved as clasping structures. Although most infestations in guinea pigs go unnoticed, hair loss and severe pruritus can occur; however, these are more likely the result of *Trixacarus caviae* (Sarcoptidae).

Listrophoroides cucullatus is widely distributed on commensal rats (*Rattus rattus*, *R. norvegicus*), primarily in tropical and subtropical areas. This species is rarely observed in laboratory colonies and is not known to cause damage.

Chirodiscidae

Chirodiscid fur mites are distinguished by the extreme fusion of segments on legs I and II and flattening of the fused segments to form structures used for attachment to host hairs. Most Chirodiscidae parasitize bats, but a few are found on shrews, primates, and carnivores. Most unusual is the genus *Schizocarpus*, species of which parasitize beavers (Bochkov and Saveljev, 2014). The genus is hyperspeciose, with 48 species being restricted to populations of the North American (*Castor canadensis*) and Eurasian (*C. fiber*) beavers. These mites presumably feed like other fur mites and do not damage the pelage. They can occur in large numbers and are the most common ectoparasites on these fur-bearing animals.

Myocoptidae

This family is superficially similar to the Listrophoridae, although most species are flattened rather than cylindrical. All stages occur in the pelage of their rodent and marsupial hosts, where they attach to hairs with their modified legs 3 and 4. In the female, the tibia and tarsus of both pairs of legs fold against the striated inner surfaces of the genu and femur to provide efficient clasping organs. The most common member of this family is *Myocoptes musculinus* (Fig. 26.32), which infests wild and captive house mice throughout the world. It attaches its eggs singly to the lower part of the hair shaft and requires about 14 days

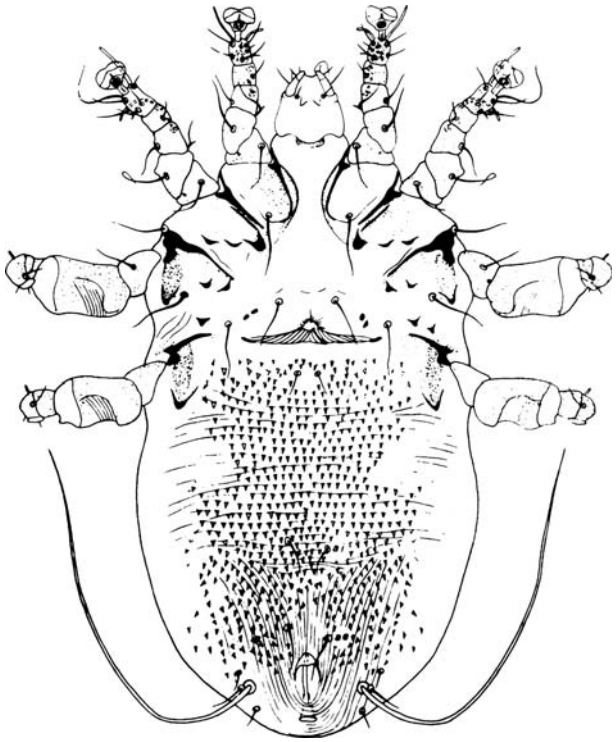


FIGURE 26.32 *Myocoptes musculinus* (Myocoptidae), female, ventral view. *Modified from Whitaker, 1982; after Fain et al., 1970.*

to complete its life cycle. These mites feed on superficial epidermal tissues; infestations usually go unnoticed. In laboratory mice, however, conditions often contribute to a buildup of mite numbers that can cause **myocoptic mange**. This is characterized by pruritus, erythema, development of a dull coat, and thinning of the hair owing to physical damage by attached mites and scratching. Signs usually are first noticed on the neck and from there spread to the shoulders, back, and other parts of the body. Myocoptic mange generally is more severe in older mice or those with lower resistance.

A few other myocoptid mites occasionally cause mild dermatitis in rodent hosts. These include *Trichoecius romboutsi*, which is known to infest laboratory mice; *T. tenax* which infests voles (*Microtus* spp.); and *Sciurocoptes sciurinus,* which infests squirrels.

Feather Mites

Mites representing 33 families and approximately 2000 species live on or in the feathers of birds (Gaud and Atyeo, 1996; Proctor, 2003). Their diversity as a group is reflected in their morphological adaptations for exploiting the many microhabitats that feathers provide. These include different types of feathers (e.g., primary and secondary flight feathers, wing coverts, contour feathers) and their location on the feathers. Mites that live on exposed feather surfaces tend to be more sclerotized, with a reduced setation and prominent terminal body setae. Those that live in more protected sites (e.g., contour feathers and calmus) are generally less sclerotized, often with distinctly modified body forms and pretarsal structures. Most feather mites live on the surface of the feathers, where they feed primarily as saprophages on skin scales, feather debris, and oily secretions. Diatoms, fungal spores, and other organic materials also serve as food. Among the more common examples of such feather mites are members of the families Analgidae (Fig. 26.33), Proctophyllodidae (Fig. 26.34), and Pterolichidae (Fig. 26.35). The analgid mites *Meginia cubitalis* and *M. ginglymura* and the pterolichid mite *Pterolichus obtusus* occasionally cause dermatitis and reduced thriftiness in chickens. Other mites called **quill mites** live inside the base of feathers (calmus), where they feed on feather tissues or pierce the calmus wall to feed on host fluids. Two such families are the Syringobiidae (Fig. 26.36) and Syringophilidae (Fig. 26.37). Like most feather mites, quill mites rarely cause apparent harm to their hosts and generally are not considered to be economically important. Occasionally, however, heavy infestations of *Syringophilus bipectinatus* cause feather loss in chickens.

Mange Mites

The following families include species that cause mange in animals, including livestock, poultry, companion animals, and laboratory colonies: Epidermoptidae, Dermationidae, Knemidokoptidae, Laminosioptidae, Demodecidae, Psorergatidae, Sarcoptidae, Psoroptidae, Harpirhynchidae, and Hypoderatidae.

Epidermoptidae and Dermationidae

Several species of the Epidermoptidae have been reported to cause discomfort and injury to infested birds. Although commonly referred to as feather mites, they are more appropriately called **avian skin mites**, as reflected in the family name. They generally live on the skin surface or in feather follicles, where their feeding can lead to itching, pityriasis (scaly or scabby dermatitis), and various other types of superficial skin lesions. *Epidermoptes bilobatus* (Fig. 26.38) infests galliform birds and occasionally causes pityriasis in chickens, whereas *E. odontophori* has been reported to cause mange in African birds. *Microlichus avus* and *M. americanus* are known to produce crateriform skin lesions and severe mange in several avian species. *Hemimyialges* species (e.g., *H. macdonaldi*) may invade the outermost skin layers to produce pityriasis and mange, sometimes severe enough to cause feather loss. Heavy infestations of *Rivoltasia bifurcata* (Dermationidae) on

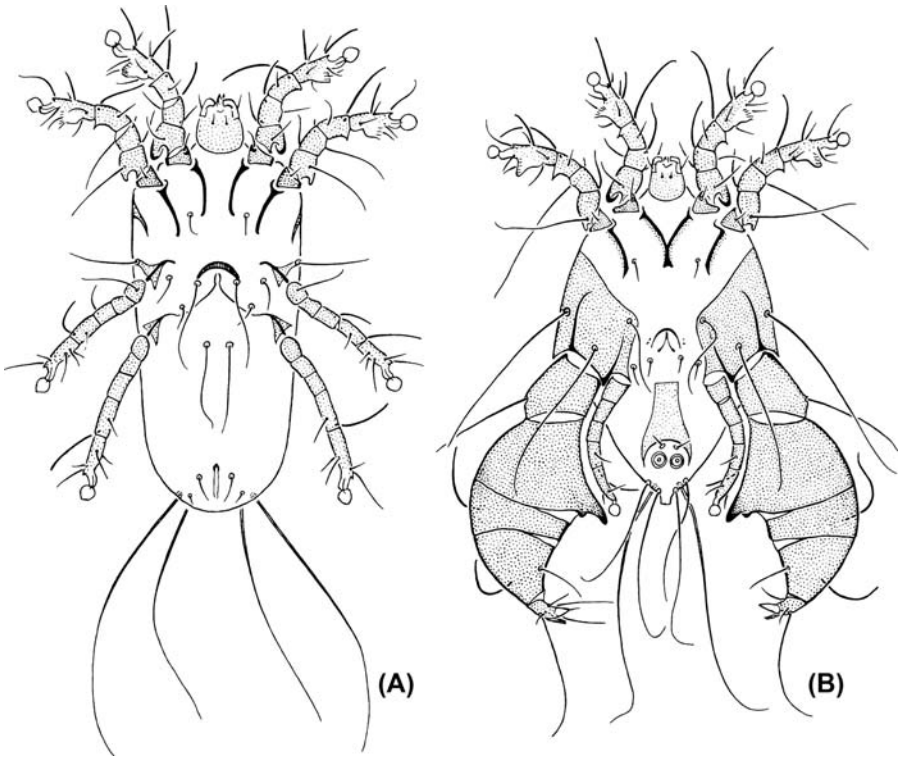


FIGURE 26.33 *Analges chelopus* (Analgidae), ventral views. (A) Female; (B) male. *From Gaud and Atyeo, 1996.*

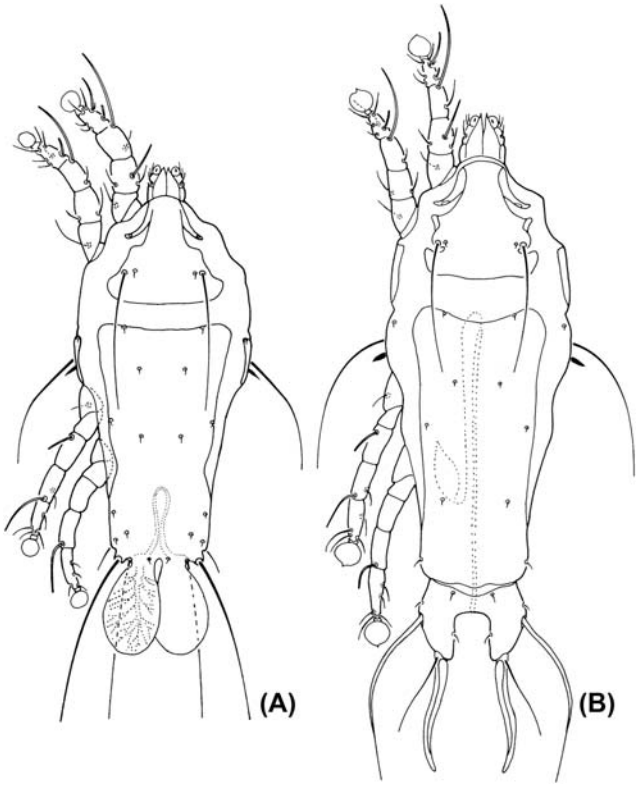


FIGURE 26.34 *Proctophyllodes glandarinus* (Proctophyllodidae), dorsal views. (A) Male; (B) female. *From Gaud and Atyeo, 1996.*

chickens can result in intense itching and pityriasis, especially involving the head. For further information on these and other epidermoptid genera of veterinary interest, see Fain (1965) and Krantz (1978).

Knemidokoptidae

Knemidokoptid mites superficially resemble sarcoptids, from which they differ by having short legs without pretarsi or long setae and lacking dorsal triangular spines. They invade the feather follicles and skin of wild and domestic birds worldwide, causing **knemidokoptic mange** in some species. Their life cycle is similar to that of *Sarcoptes scabiei*. All stages of these mites occur on the host and transmission is by direct contact with infested birds. There are several species of veterinary importance: *Knemidokoptes mutans* and *Neocnemidocoptes gallinae* infest poultry, *K. pilae* infest parakeets, and *K. jamaicensis* infest passerine birds, including canaries. Based on phylogenetic analyses, some workers have suggested that the Knemidokoptidae be included within the Epidermoptidae or Dermationidae.

Scaly-Leg Mite (Knemidokoptes mutans)

This mite (Fig. 26.39) is a pest of poultry, especially chickens, in North America, Europe, and Africa, and

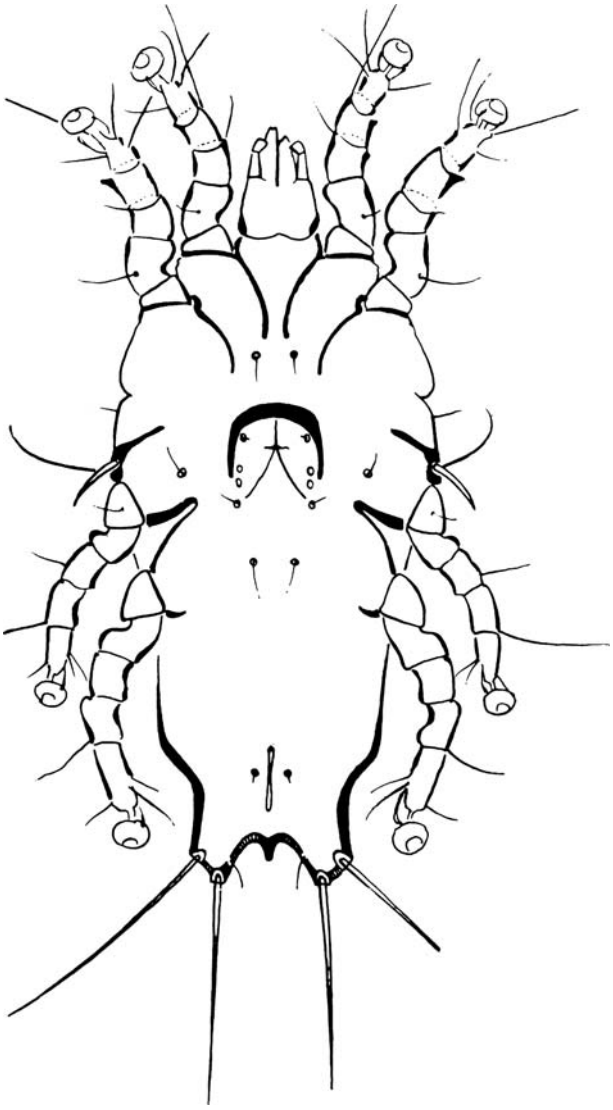


FIGURE 26.35 *Musophagobius cystodorus* (Pterolichidae), female, ventral view. *From Gaud and Atyeo, 1996.*

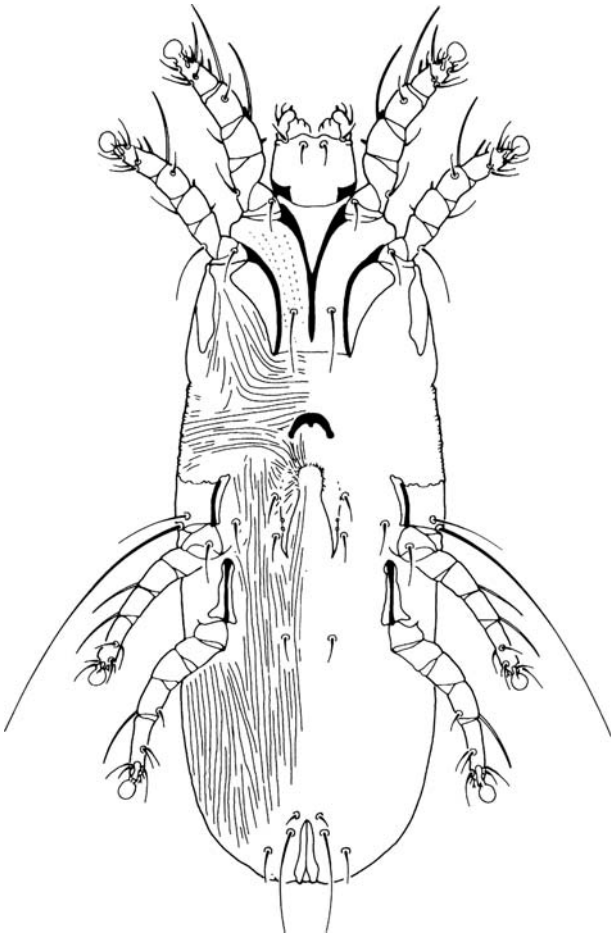


FIGURE 26.36 *Dabertia tricalcarata* (Syringobiidae), female, ventral view. *From Gaud and Atyeo, 1996.*

probably occurs worldwide. It burrows beneath the epidermal scales of the legs and feet, causing irritation, inflammation, hyperkeratization, formation of vesicles, and encrustations (Fig. 26.40). The crusts may cover entire limbs, hence the term **scaly leg**, a condition most commonly seen in older birds. In chronic cases, infestations can lead to lameness, deformed legs and feet, and occasionally the loss of digits. The skin of the comb and wattle also may be involved. *Knemidokoptes jamaicensis* causes similar symptoms in wild and cage-reared passerine birds.

Scaly-Face Mite (Knemidokoptes pilae)

This cosmopolitan mite infests captive parakeets, causing crusty lesions primarily about the face, head, and legs. Lesions usually appear initially in the cere (at base of the upper beak) and at the corners of the beak, where the mites invade the feather follicles and folds of skin. There they form pouch-like cavities or pits and a honeycomb pattern that is discernible upon close examination. Early signs include whitish excrescences that may spread to the eyes, forehead, and other parts of the body. These form gray-white to yellow encrustations in chronically infested birds. Lesions of the legs and feet, especially in the early stages, can closely resemble those of *K. mutans*. Advanced cases may involve distortions of the beak as the keratinized tissues become overgrown and friable. Even in such cases, there appears to be little or no pruritus, rubbing, or scratching of the beak.

Depluming Itch Mite (Neocnemidocoptes gallinae)

Unlike the previous two mites, the depluming itch mite infests the feathered areas of chickens, burrowing into the epidermis at the base of feathers or into the feather shafts.

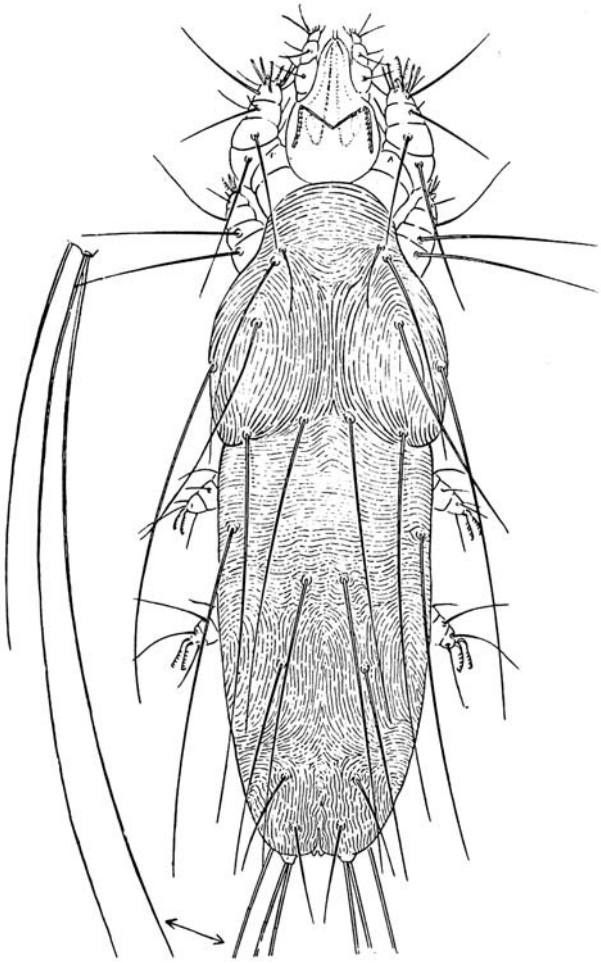


FIGURE 26.37 *Syringophilus bipectinalis* (Syringophilidae), female, dorsal view. *From Baker et al., 1956.*

The parts of the body most commonly affected are the head, neck, back, abdomen, and upper legs. The wings and tail are not usually involved. The mites tend to be confined to the stratum corneum, causing hyperkeratosis, thickening and wrinkling of the skin, and sloughing of keratinous layers. Feathers in the affected areas often break off or fall out, or may be plucked by the bird, which accounts for the mite's common name. Severely infested birds may become emaciated and die. A similar species, *Picicnemidocoptes laevis*, may cause similar symptoms in pigeons and doves.

Laminosioptidae

The only significant laminosioptid mite of veterinary importance is the fowl cyst mite.

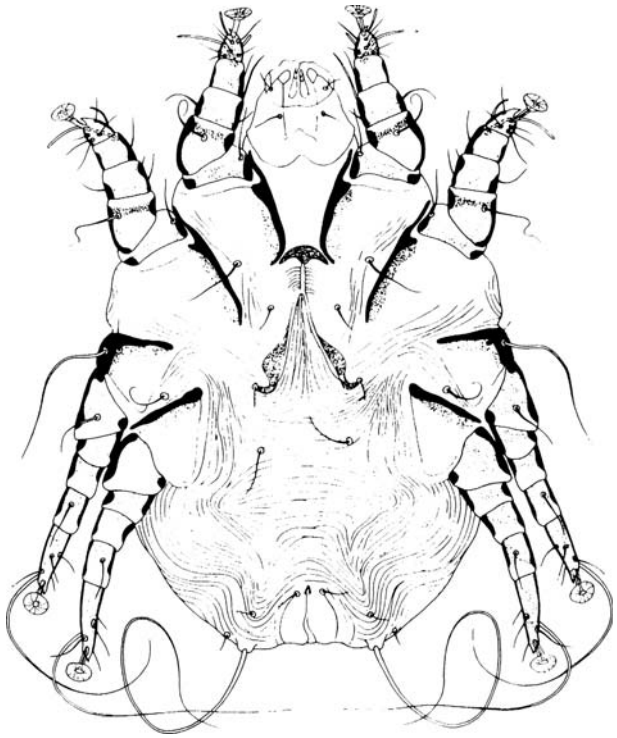


FIGURE 26.38 *Epidermoptes bilobatus* (Epidermoptidae), female, ventral view. *From Gaud and Atyeo, 1996; original by Fain, 1965.*

Fowl Cyst Mite (Laminosioptes cysticola)

This mite (Fig. 26.41) occurs worldwide as a parasite of chickens, pheasants, turkeys, geese, pigeons, and other birds. It invades the skin of its avian hosts to form small, yellowish, subcutaneous nodules or cysts up to several millimeters in size. The nodules are formed by calcareous deposits produced around the mites after they have died. They are most easily seen in living birds by wetting and parting the breast feathers and sliding the skin back and forth with the fingertips. Occasionally, *L. cysticola* causes heavy infestations that can be fatal. A case was reported in West Virginia (United States) involving a wild turkey with severe neurologic disease (Smith et al., 1997). The affected turkey held its head bent over its back when at rest and exhibited circling behavior and falling to one side when it attempted to walk. Histopathologic examination revealed *L. cysticola* mites in enlargements of the wing nerves, inflammation of the brain, and numerous mites in the esophagus, small intestine, and other internal tissues. The fowl cyst mite also has been reported to cause granulomatous pneumonia in dogs (Shaddock and Pakes, 1978). Little is known about the life history or transmission of this mite.

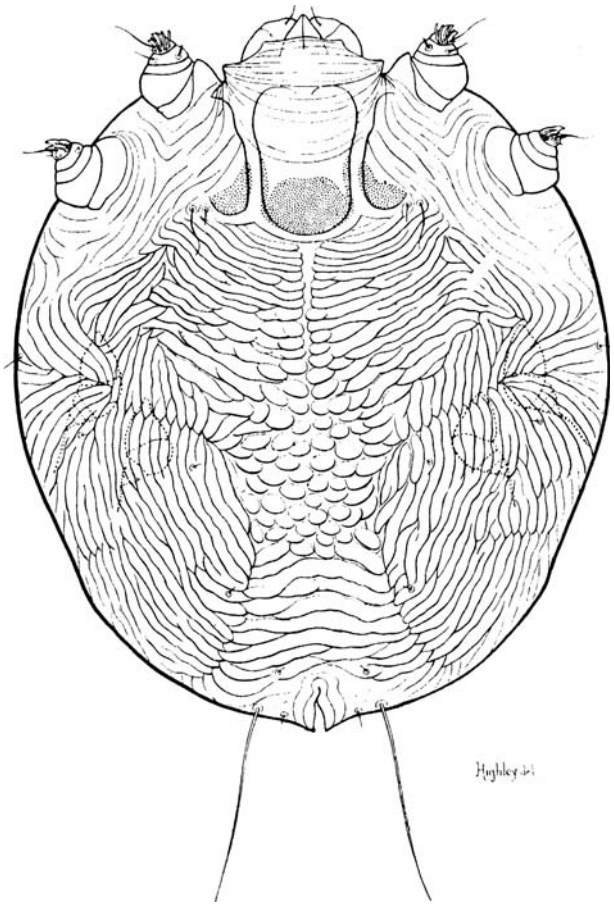


FIGURE 26.39 Scaly-leg mite, *Knemidokoptes mutans* (Knemidokoptidae), female, dorsal view. *From Hirst, 1922.*

FIGURE 26.40 Chicken with crusty, raised scales on feet caused by infestation with scaly-leg mite (*Knemidokoptes mutans*). *Photograph by Jerry F. Butler.*

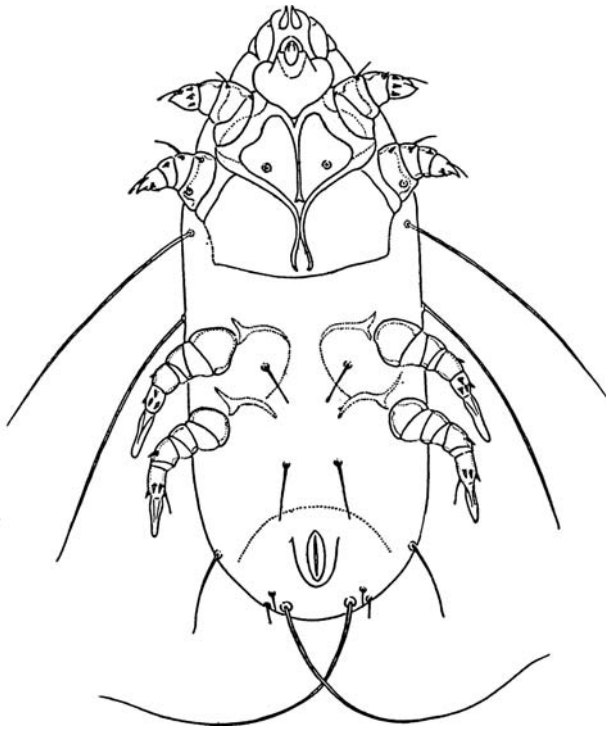


FIGURE 26.41 *Laminosioptes cysticola* (Laminosioptidae), female, ventral view. *From Hirst, 1922.*

Demodecidae

Mites in this family are highly specialized skin parasites that live in the hair follicles and associated glands of domestic and wild mammals. An infestation of demodecid mites is called **demodecosis**, whereas cases with clinical signs are called **demodectic mange**. The mites are host-specific and typically occur either in hair follicles or dermal glands. These sites include sebaceous glands, modified sebaceous glands (e.g., meibomian, caudal, preputial, and vulval glands), modified sweat glands (e.g., ceruminous glands and submaxillary skin papillae), and mixed sebaceous-sweat glands (e.g., perianal glands). *Opthalmodex* infests lachrymal ducts. A few species burrow into the skin to form epidermal pits, as in *Demodex criceti* of hamsters. Other demodecid species invade oral tissues of their host, infesting the oral epithelium, tongue, and esophagus of the grasshopper mouse (*Onychomys leucogaster*) in western North America and the oral cavities of bats and lemurs in Europe and Africa. In most cases, demodecid mites cause little or no apparent harm to their hosts. In other cases, infestations can lead to varying degrees of dermatitis and other skin problems.

Demodectic mange is common in dogs; livestock such as cattle, goats, sheep, and swine; wild animals such as foxes, other canids, and rabbits; and occasionally

laboratory animals such as hamsters, gerbils, guinea pigs, rats, and mice. It is relatively uncommon in cats and horses.

Injury to the host occurs as the mites puncture with their stylet-like chelicerae the epithelial cells lining the hair follicles and glands to feed on the cell contents. In most cases, the host response is only mild to moderate hypertrophy of the affected epithelia. In other cases, marked hypertrophy and cell destruction may occur. The openings of hair follicles or the ducts of glands may become blocked, leading to the formation of dermal papules and nodules. Damage to the follicles can lead to hair loss, whereas secondary bacterial infections may cause inflammation, pruritus, and the formation of pustules. Lesions often occur first on the face and head, spreading from there to other parts of the body.

Two major clinical forms of demodicosis are recognized, squamous and papulonodular. **Squamous demodicosis** is the more common form, characterized by a dry, scaly dermatitis with itching and loss of hair in the affected areas. Secondary infections often result in the rupture of follicular cells, severe inflammation, and purulent exudates. This can be either a localized or generalized skin condition and occurs in all host groups. **Papulonodular demodicosis** occurs when the hair follicles or gland ducts become obstructed and produce palpable, cyst-like or nodular swellings in the skin, trapping the mites within. The development of demodectic papules and nodules is most commonly seen in cattle, goats, and pigs. These lesions continue to enlarge as the mites multiply, sometimes reaching several thousand mites per lesion, along with accumulated cellular debris and glandular secretions. These nodules may rupture externally leading to secondary infections and abscesses. In other cases, they may rupture within the skin, introducing the mites to the circulatory and lymphatic systems. There, they can cause thromboses and internal infestations.

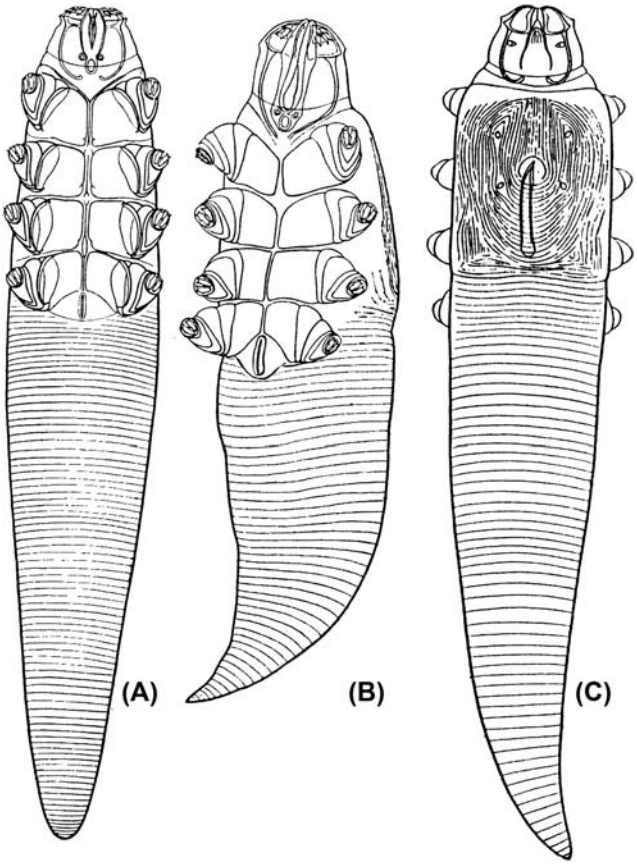


FIGURE 26.42 Follicle mites (Demodecidae) of domestic animals. (A) *Demodex canis;* (B) *Demodex phylloides*; (C) *Demodex cati*. *From Hirst, 1922.*

Dog Follicle Mites (Demodex canis, D. injai, and D. cornei)

Demodex canis (Fig. 26.42) inhabits the hair follicles and sebaceous glands of dogs throughout the world. It completes its life cycle in 3–4 weeks; the eggs and all developmental stages are found in the follicles or glands. Clinical signs are most common in dogs less than a year old, presumably reflecting an immunodeficient state in young animals. Canine demodectic mange (Fig. 26.43) usually appears as mildly erythematous patches about the eyes and corners of the mouth, typically associated with hair loss. From there, the infestation may spread to the forelegs and trunk as a typical squamous form of demodicosis. Most cases resolve without treatment. In genetically predisposed or immunodepressed animals and cases of secondary infections, the condition can develop into chronically severe, moist, purulent dermatitis known as **pustular demodicosis**. This is often accompanied by an unpleasant odor variously described as rancid or mousy. If this becomes generalized with intense redness and tenderness of the skin and easily bleeds, it is called **red mange**. Severe cases of red mange also occur in foxes and other wild canids, sometimes leading to death in heavily infested animals. Diagnosis of *Demodex* infestations is confirmed by demonstrating the presence of mites in material expressed from hair follicles and in skin scrapings or biopsies of skin lesions. Although most dogs recover from localized infestations even without treatment, other cases may require extended acaricide applications; some are never resolved despite every treatment effort.

Most dogs apparently become infested with *D. canis* as newborn pups, either while nursing or in other intimate contact with their mother or another infested dog. There is little or no evidence to substantiate reports of prenatal transmission of the mite. Nor is the transfer of *D. canis* between mature animals likely; apparently it occurs only under unusual circumstances.

Two other species of *Demodex* are reported from dogs, *D. injai* and *D. cornei*. *Demodex injai* occurs mainly in the sebaceous glands and ducts, whereas *D. cornei* occurs in the keratinized layer of the epidermis. Both are capable of

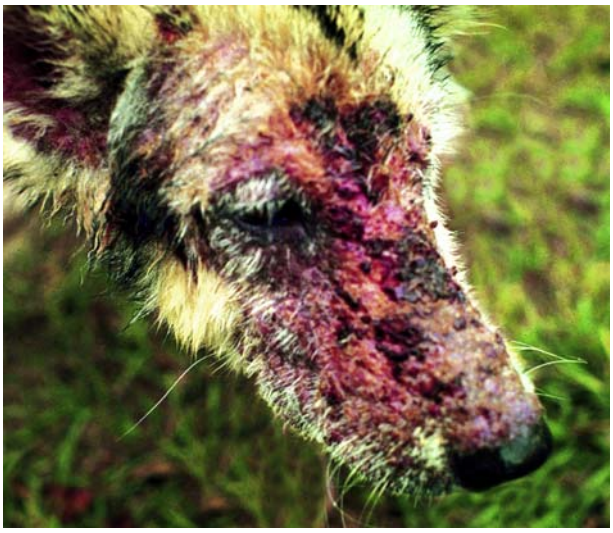


FIGURE 26.43 Demodectic mange in dog caused by *Demodex canis* (Demodecidae). *Photograph by Jerry F. Butler.*

causing pathology similar to that of *D. canis* when occurring in large numbers (Izdebska, 2010).

Cat Follicle Mites (Demodex cati and D. gatoi)

Occasionally, cats develop lesions attributed to *Demodex cati* and *D. gatoi*. They usually are the squamous form of demodicosis and occur on the head or as a generalized condition with varying degrees of pruritus. Cases of **feline demodicosis** are believed to be associated with underlying immunosuppressive diseases (e.g., feline leukemia, diabetes mellitus).

Cattle Follicle Mites (Demodex bovis and D. tauri)

Cattle infested with *Demodex bovis* commonly develop a papulonodular form of demodicosis. Adult female mites deposit their eggs in hair follicles, where their populations may build to hundreds or thousands of mites per follicle as the follicles become dilated to form dermal papules or cysts. As they enlarge, they can be felt beneath the skin even though they may be difficult to see. Some female mites exit the follicular cysts to invade other hair follicles, thereby spreading the infestation. It is presumably at this time when they also are transferred to other animals by intimate contact. It is postulated that transfer between cattle can occur during copulation.

The lesions tend to be concentrated on the anterior parts of cows, notably the neck, shoulders, and axillary region, but also may occur on the udder. Papular cysts enlarge to form granulomatous nodules when the follicular opening becomes blocked by mite bodies, keratin, and other debris. The occurrence of papulonodular demodicosis in cattle is commonly associated with cows that are stressed by pregnancy or lactation. Individual nodules typically form over a month and then gradually disappear only to be replaced by other developing nodules. Both natural and acquired immunity have a role in reducing mite numbers and associated clinical signs in infested cattle.

The dilated follicles vary in size from that of a pinhead to a chicken egg. Commonly, the larger nodules rupture to produce suppurative sores. The pus-like exudate containing large numbers of *D. bovis* has the consistency of toothpaste and can serve as a means of transfer of mites to other animals. Skin damage resulting from these ruptured nodules can cause defects in raw leather and significant economic losses to the tanning industry in the form of diminished quality of processed cowhides.

A second species of demodecid mite of bovids, *Demodex tauri*, has been reported from hair follicles and ducts of sebaceous glands in the eyelids of cattle in the former Czechoslovakia (Bukva, 1986).

Goat Follicle Mite (Demodex caprae)

When infested with *D. caprae*, goats develop dermal papules and nodules similar to those in cattle. Cases of caprine demodicosis occur most commonly in young animals, pregnant does, and dairy goats, the latter presumably reflecting the stress of lactation. Papules usually appear on the face, neck, axillary region, or udder, with a few to several hundred lesions per animal. They are easily palpable in the skin, enlarging to form nodules up to 4 cm in diameter as the mites multiply within. Ruptured nodules tend to suppurate, contributing to transmission of the mites via exudates to other animals. As in dogs, goats have a high incidence of generalized demodicosis that can involve almost any part of the body. If the nodules rupture internally, granulomas develop while phagocytic giant cells of the goat host engulf and destroy the mites. As individual nodules disappear, others are formed. Transmission of *D. caprae* to newborn goats typically occurs within the first day after birth. Other possible means of transfer are parental licking and intimate contact of animals during copulation. Certain breeds of goats (e.g., Saanen) tend to be more sensitive to demodicosis than are others.

Psorergatidae

Psorergatid mites are obligate parasites of mammals that live in superficial layers of the skin of their hosts. Their life histories are similar to those of the Demodecidae. Whereas most species cause no apparent harm to their hosts, a few species cause psorergatid mange in sheep, rodents, and certain primates. Transmission between animals is by direct contact and transfer of adult females that enter hair follicles,

where they deposit their eggs. All developmental stages occur within the follicles, where the mites feed by puncturing follicular cells. Mange-inducing *Psorergates* species cause inflammation and enlargement of the hair follicles to form dermal pouches, or pockets, beneath the stratum corneum. The lesions appear as small white nodules (up to 2 mm in diameter) on the inner skin surface. The nodules contain all stages of the mite and necrotic debris from the destroyed follicles. In rodents, large lesions may form, with mites lining the inner surface of the lesions.

Sheep Itch Mite (Psorobia ovis)

The psorergatid mite of most veterinary importance is the sheep itch mite, formerly named *Psorergates ovis*. It infests all breeds of domestic sheep, but especially Merinos. Clinical signs include dry scurfy skin, loss of hair, and sometimes erythema accompanied by intense pruritus. Infested animals often are restless and bite or rub the affected areas, damaging the skin and wool. Lesions occur most frequently on the neck and shoulders, gradually spreading to the face, flanks, thighs, and other parts of the body. The spread of *P. ovis* through a flock is slow and is most evident during the winter months.

Mouse Itch Mite (Psorergates simplex) and Related Species

The mouse itch mite (Fig. 26.44) infests wild and laboratory mice in Europe and North America. Its development and life history are similar to those of *P. ovis*. The dermal pouches and resultant white nodules occur most frequently on the head of infested mice, but also may involve the neck, legs, and other parts of the body. Chronic infestations, especially in laboratory mice, can lead to crusty or ulcerous nodules, hypertrophy of skin cells, dermatitis, and hair loss.

Psorergates mites are sometimes associated with cases of murine ear mange, evidenced by pale yellow crusts on the inner and outer ear surfaces. Such cases in the house mouse (*Mus musculus*) typically involve *P. muricola*. A third species, *P. hispanicus*, forms lesions on the legs of *M. musculus*.

Other *Psorergates* species that cause lesions in rodents include *P. apodemi*, *P. dissimilis*, and *P. microti*. Cattle are hosts for *Psorobia bos*, which seldom causes apparent lesions or harm to its bovine hosts. Occasionally, however, it is reported to cause psorergatic mange, as in an infested herd of Bonsmara bulls in South Africa (Oberem and Malan, 1984). *Psorobia cercopitheci* reportedly causes mild dermatitis in mangabey monkeys (Sheldon, 1966) and vervet monkeys (Seier, 1985), whereas unspecified psorergatid species are known to cause dermal cysts and crusty skin in patas monkeys (Raulston, 1972) and macaques (Lee et al., 1981).

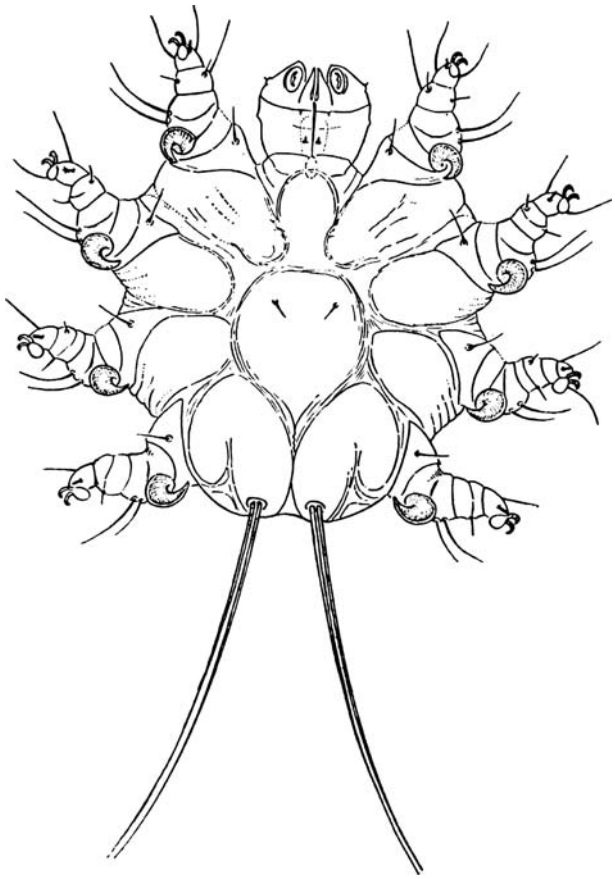


FIGURE 26.44 Mouse itch mite, *Psorergates simplex* (Psorergatidae), female, ventral view. *Adapted from Baker et al., 1956.*

Sarcoptidae

The family Sarcoptidae includes three important genera that infest domestic and wild animals: *Sarcoptes*, *Notoedres*, and *Trixacarus*. The adult females burrow into the epidermis of their hosts causing varying degrees of dermatitis with accompanying erythema, pruritus, hair loss, scaling, and dermal encrustations characteristic of **sarcoptic mange** (Fig. 26.45). Lesions may occur on any part of the body but often begin in typical locations, depending on the mite species involved.

For additional information on sarcoptid taxonomy, phylogenetic relationships, and host associations, see Klompen (1992).

Scabies Mite (Sarcoptes scabiei)

Sarcoptes mites that cause mange in animals are morphologically indistinguishable from the mite that causes human scabies, *S. scabiei*, and are considered conspecific. Their life histories are similar, with all developmental stages (larva, protonymph, tritonymph, and adult) living in burrows formed by the adult females in the stratum corneum,

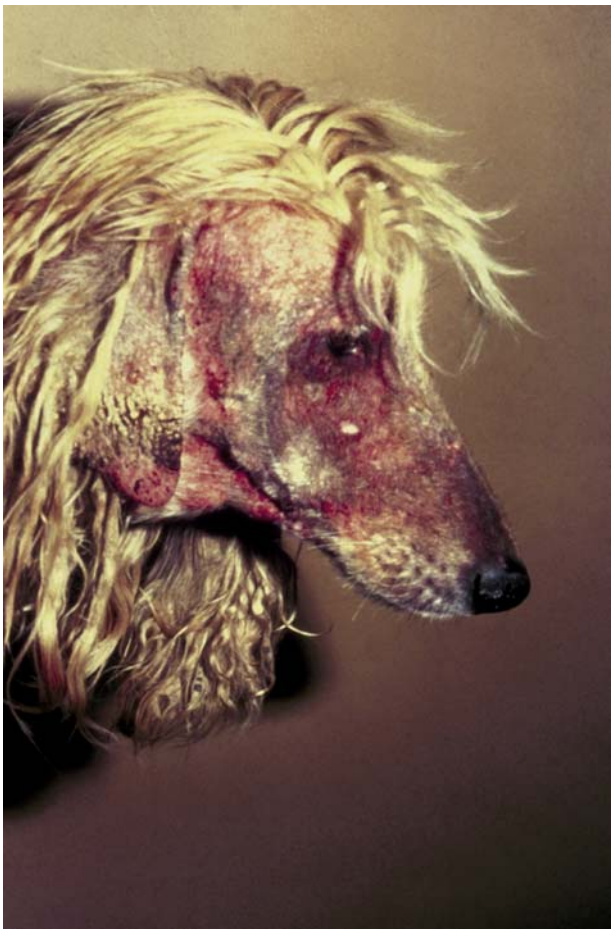


FIGURE 26.45 Severe case of sarcoptic mange in dog caused by *Sarcoptes scabiei* var. *canis* (Sarcoptidae). *Courtesy of Department of Pathobiology, Auburn University College of Veterinary Medicine.*

stratum lucidum, and upper Malpighian layer of the skin. The female lays up to three eggs per day in the burrow over 2–3 weeks, after which she dies. Development from egg to adult takes 2–3 weeks and occurs largely within the burrows. Male protonymphs leave the burrow to establish new epidermal tracts in which they molt to adults. The adult males then mate with females either on the skin surface or in shallow dermal pits.

Host reactions occur primarily in response to the mites and their fecal deposits in the burrows. This usually occurs 3 weeks or more after the initial infestation, with the reaction time becoming much shorter (e.g., a few days) after subsequent exposures. Initial lesions can occur anywhere on the body but are usually localized where the hair tends to be thin, most commonly on the head. From there, the infestation can spread quickly to cause a more generalized mange. Infestations generally appear as papular eruptions with erythema, pruritus, and hair loss. As it progresses, skin in the affected areas often becomes thickened, crusted with exudates, and secondarily infected after excoriation of the skin owing to scratching and rubbing by the host. Scaly areas around the periphery of infested patches often indicate the spread of mites. In extreme cases, severely sensitized animals may experience weight loss, difficulty eating, impaired hearing, blindness, exhaustion, and death.

Burrows seldom are detectable in nonhuman animals, which makes it difficult to recover mites to confirm their identification. Therefore, negative skin scrapings are inconclusive. As a result, the diagnosis of scabies in animals is often presumptive, based on clinical signs and positive responses to acaricide treatments.

The various races of *S. scabiei* tend to be relatively host specific, infesting a range of domestic and wild mammals. Transfer of mites occurs among conspecific hosts by direct contact. When it does occur, transfer between different host species often results in only temporary infestations usually limited to a transient, mild dermatitis. In rare cases, however, it can cause severe reactions, sometimes requiring hospitalization. Such a reaction in humans (e.g., from *S. scabiei* of dogs or goats) is called **animal scabies**.

Virtually all domestic animals except cats and guinea pigs are subject to infestations of *S. scabiei*. Dogs are the most commonly affected (Fig. 26.45). Initial lesions tend to occur on thinly haired parts of the body such as the ear margins, belly, axillary and inguinal regions, elbows, and hocks. If untreated, the infestations generally spread to the head and other parts of the body. In particularly severe cases, dogs may develop thickened, pigmented skin with almost complete hair loss in affected areas of the neck, shoulders, back, trunk, and extremities. The situation often is complicated by secondary infections, self-inflicted trauma in efforts to relieve the itching, emaciation, and sometimes death. Conditions which may be mistaken for canine scabies include seborrhea, eczema, allergic dermatitis, ringworm dermatophytosis, and infestations of other mange mites (notably *Demodex*, *Notoedres*, and *Otodectes* spp.). Detection of mites requires deep skin scrapings, with best results being obtained from the tips of the ears, even in the absence of ear lesions.

Initial lesions in farm animals are usually localized in specific body regions. In sheep, goats, and horses, this is typically on the face, head, and neck. In cows, lesions usually first appear on the underside of the neck, inner thigh, brisket, and tail head. In pigs, the lesions tend to be more generalized (Fig. 26.46) but are most commonly evident as encrustations and scabs in the ears of chronically infested sows. The highest incidences of *S. scabiei* infestations in farm animals usually occur in the winter months and are attributed to crowding of animals and loss of condition at that time of the year.

In addition to direct transfer between mature animals, *S. scabiei* is transmitted from mother to offspring at birth.

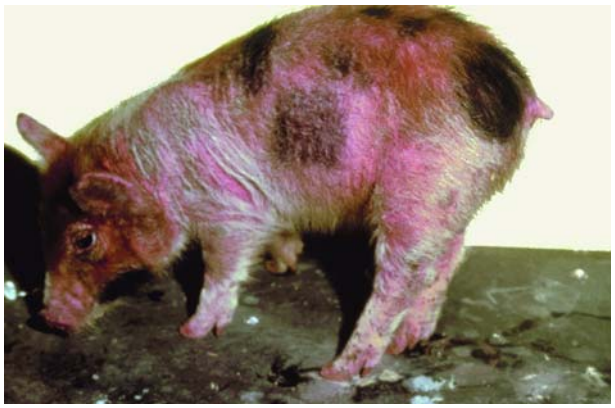


FIGURE 26.46 Sarcoptic mange in pig caused by *Sarcoptes scabiei* var. *suis* (Sarcoptidae). Note inflammation of skin and hair loss. *Courtesy of Department of Pathobiology, Auburn University College of Veterinary Medicine.*

Pruritus has been reported in 4-day-old piglets born to infested sows. Maternal antibodies to *S. scabiei* are detectable in neonatal pigs within 6 h, reaching their maximum levels 24–48 h after birth (Bornstein and Zakrisson, 1993).

Although cases of sarcoptic mange in goats often resolve without developing severe signs, heavily infested goats may exhibit crusty lesions and extensive hair loss around the muzzle, eyes, and ears; lesions on the inner thighs extending to the hocks, brisket, ventral abdomen, and axillary region; dermal thickening and wrinkling of the scrotum and ears; and dry, scaly skin on all parts of the body, especially in areas of hair loss (Kambarage, 1992). Sarcoptic mange also can cause significant problems in camels (Kumar et al., 1992).

Sarcoptes scabiei may also infest laboratory animals. Canine scabies is common in laboratory dogs obtained from commercial suppliers and animal shelters, with the highest incidence in young dogs and short-haired breeds. Laboratory rabbits usually develop initial lesions on the head, ears, and legs, followed by a more generalized dermatitis with associated erythema, pruritus, scaling of skin, and loss of fur. The situation can be complicated by scratching and self-inflicted injuries within the confines of a cage. Other laboratory animals are infrequently or rarely subject to sarcoptic mange. These include mice, hamsters, and guinea pigs.

In addition to human hosts, *Sarcoptes scabiei* has been reported to infest captive cynomolgus monkeys and simian primates (orangutan, gibbons, and chimpanzees). Lesions are characterized by thickening of the skin of the neck, shoulders, and back and sometimes the lower trunk and extremities. This may be associated with other signs such as skin scales, pruritus, hair loss, and emaciation. For further information on sarcoptic mange in laboratory animals, see Yunker (1973).

Several groups of wild mammals are susceptible to sarcoptic mange by *S. scabiei*. Members of the Canidae, Procyonidae, and Cervidae are the more commonly infested. Canid hosts are the most severely affected carnivores in North America, notably the red fox (*Vulpes fulva*), gray fox (*Urocyon cinereoargenteus*), wolf (*Canis lupus*), and coyote (*Canis latrans*). The most severe cases have been reported in red foxes during the winter months. In the northeastern United States, eastern Canada, and Russia, epizootic sarcoptic mange has had an economic impact on the pelt industry in some years. Early signs of mite infestations are dry, flaky skin, followed in succeeding weeks and months by crusty lesions, hair loss, eyelid scaling, emaciation, scratching, biting, and even death. Affected areas include the muzzle, neck, shoulders, back, and hind quarters. Successful transfer of *S. scabiei* from red foxes to domestic dogs, gray foxes, and feral dog–coyote hybrids have been demonstrated experimentally (Stone et al., 1972). Sarcoptic mange in raccoons (*Procyon lotor*) is anecdotally reported as a mortality factor in these populations.

Cervid species that are known to develop sarcoptic mange include roe deer (*Capreolus capreolus*) and red deer (*Cervus elaphus*) in Europe, and wapiti (*C. wapiti*) in North America. Lesions in deer occur as encrustations of the outer ear, whereas in wapiti they may appear as moist scabs in the dorsal and lateral thoracic regions. Some cases involve head lesions, impaired vision, blindness, and general debilitation.

Other animals that are known to develop sarcoptic mange are the fisher (*Martes pennanti*), and ferrets (*Mustela* spp.) in North America, Europe, and Asia; llamas (*Lama* spp.) in Central and South America; Thompson's gazelle (*Gazella thompsonii*), and wildebeests (*Connochaetes taurinus*) in Africa; and chamois (*Rupicapra rupicapra*) in Europe and the Caucasus. There is evidence in the case of chamois that mineral imbalances may contribute to the severity of mange cases. Providing mineral blocks or salt licks has been shown to reduce mange significantly in infested animals (Onderscheka et al., 1968). Sarcoptic mange is also a serious disease in Australian wombats; 35% of the population was reportedly affected in one study (Hartley and English, 2005). Zoo animals also become infested with *S. scabiei* as reported in the capybara (*Hydrochoerus hydrochoeris*), tapir (*Tapirus* sp.), and camel (*Camelus* sp.) in Poland (Zuchowska, 1991).

Wild primates may develop sarcoptic mange, sometimes in epizootic form. Such epizootics among chimpanzees have resulted in noticeable hair loss. Severe cases in white-headed capuchins attributed to *S. scabiei* have been characterized by abscesses, localized hemorrhages, stratified crusts, weight loss, extreme debilitation, epileptic excitations, and sometimes death (Sweatman, 1971). It is

probable, however, that another sarcoptid mite was involved.

Other Sarcoptid Genera

Sarcoptid mites in genera other than *Sarcoptes* also naturally infest primates. These species are similar morphologically and in the damage they cause. Typical signs are scabby, encrusted papular lesions, intense pruritus, and hair loss. *Prosarcoptes talapoini* and *P. pitheci* cause mange in guenons (*Cercopithecus* spp.) and baboons (*Papio* spp.), and *Prosarcoptes scanloni* causes mange in macaques (*Macaca* spp.). *Kutzerocoptes gruenbergi* causes a similar condition in capuchin monkeys (*Cebus capucinus*). For further information on sarcoptid mites infesting primates, see Fain (1968), Smiley and OConnor (1980), and Klompen (1992).

The following genera of sarcoptid mites are restricted to bats: *Chirophagoides* (New Zealand), *Chirnyssoides* (Neotropics), *Nycteridocoptes* (Europe, Asia, and Africa), *Cynopterocoptes* (Asia), *Rousettocoptes* (Asia), *Tychosarcoptes* (Asia), *Teinocoptes* (Africa, Asia, and Australia), and *Chirobia* (Africa and Asia). Most species of *Notoedres* are also restricted to bat hosts.

Whereas some bat hosts exhibit little adverse reaction, others develop pustules, scabby lesions, or small, cornified pouches, nodules, or cysts containing the mites. These lesions can occur on any part of the body including the ears and wing membranes. In some cases, the mites burrow into the skin, particularly the anterior part of the body, or excavate shallow epidermal pits in the stratum corneum, where the mites can be found. For further details on sarcoptid mites of bats, see Sweatman (1971).

Notoedres Species

The genus *Notoedres* is grossly similar to *Sarcoptes*. These mites differ from *Sarcoptes*, however, by having the anal opening located dorsally and lacking dorsal spines, although rounded scales may be present. They are skin parasites that typically burrow in the stratum corneum, causing inflammation, crusty lesions, and hair loss known as **notoedric mange**. Common hosts include cats, rats, squirrels, rabbits, and bats. *Notoedres* species have infrequently been reported to parasitize dogs and foxes, civets, lorises (primates), koalas and bandicoots (marsupials), hamsters, and hedgehogs. Most infestations begin on the head, causing a condition called **head mange**, commonly observed in cats and rabbits. From there, the mites spread to other parts of the body. Severe cases can lead to dehydration, emaciation, and death of the host. Transmission between animals is by direct contact; only rarely are humans affected. Diagnoses are based on the clinical pattern of lesions and identification of the mites in skin scrapings. *Notoedres* mites are more readily recovered in skin scrapings than are *Sarcoptes* mites.

Notoedric Cat Mite (*Notoedres cati*)

This is the common *Notoedres* mite (Fig. 26.47) of domestic cats in North America, Europe, and Africa, although it probably occurs worldwide. It also infests wild cats, laboratory rabbits, and rarely dogs, foxes, other canids, and civets. As the adult female burrows in the skin, she deposits eggs that hatch in 3–4 days. Development from egg to adult requires 6–10 days. Although *N. cati* typically burrows in the stratum corneum and stratum germativum, it occasionally invades hair follicles and

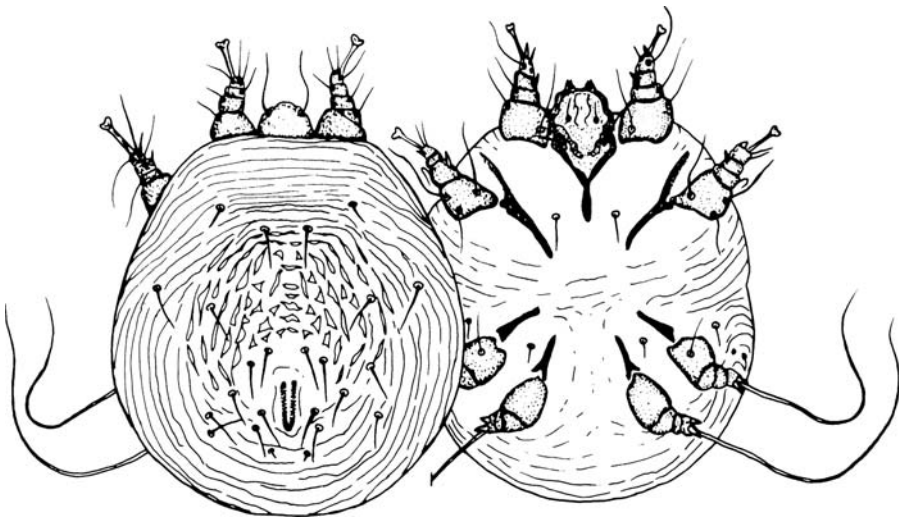


FIGURE 26.47 *Notoedres cati* (Sarcoptidae), female; dorsal (left) and ventral (right) views. *From Nutting, 1984.*

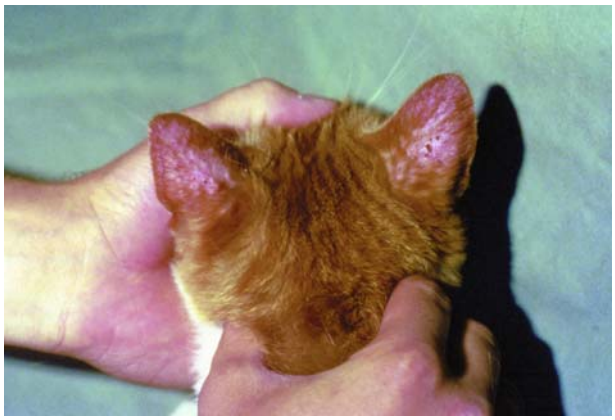


FIGURE 26.48 Notoedric mange in cat caused by *Notoedres cati* (Sarcoptidae), showing characteristic skin lesions on outer ears. *Courtesy of Department of Pathobiology, Auburn University College of Veterinary Medicine.*

sebaceous glands causing hyperkeratosis and thickening of the epidermis. Lesions usually appear first on the ears (Fig. 26.48), neck, face, and shoulders, but sometimes on the ventral abdomen, legs, and genital area, especially in younger animals. The feet and perineum may become involved owing to the cat's sleeping position and grooming behavior. Typical signs are intense pruritus, erythema, skin scaling, grayish-yellow crusts, and loss of hair. As infestations progress, the affected skin becomes thickened, folded, and wrinkled. Scratching to alleviate the itching aggravates the condition by excoriating the skin and causing inflammation. Severe chronic cases can lead to systemic debilitation and death. To distinguish cases from other possible skin problems or mite species, the identity of *N. cati* should be confirmed by examining skin scrapings, usually best taken from the ears.

Wild cats known to become infested with *N. cati* include the Siberian tiger (*Felis tigris*) and North American bobcat (*Lynx rufus*). A fatal case of notoedric mange in an adult bobcat was reported in Texas (United States). The cat was emaciated and extremely weak, with hair loss about the head, neck, and shoulders and with associated greatly thickened skin and gray encrustations. Bobcat kittens in the same area exhibited similar skin lesions confirmed as notoedric mange (Pence et al., 1982).

Notoedric Squirrel Mite (Notoedres centrifera)

This mite, formerly known as *N. douglasi*, causes notoedric mange in North American squirrels (*Sciurus* spp.) and porcupines (*Erethizon dorsatum)*. Until the first report of infestations in porcupines (Snyder et al., 1991), it was thought to infest only sciurids. Cases in the United States have been reported in the eastern gray squirrel (*S. carolinensis*) in Massachusetts, California gray squirrel (*S. griseus griseus*) in California, and fox squirrel (*S. niger*) in Indiana, Michigan, and West Virginia. It may also occur in chipmunks (*Tamias* spp.). Lesions are similar to those of other *Notoedres* species, with thickened and wrinkled skin, scurfy yellowish crusting, and hair loss usually about the head and neck. Other affected areas include the back, torso, limbs, and base of the tail. In some cases, multifocal hyperpigmentation, pinpoint nodules, and microabscesses have been reported, in addition to extensive hair loss, dehydration, emaciation, and death. Significant mortality has occurred in epizootics of notoedric mange among California gray squirrels attributed in part to impaired vision and disruption of food-seeking ability of severely affected animals. For further information on *N. centrifera*, see Carlson et al. (1982) and Kazacos et al. (1983).

Notoedric Rat Mite (Notoderes muris)

This mite infests rodents and is the cause of notoedric ear mange commonly seen in laboratory rats. It is known to parasitize the Norway rat (*Rattus norvegicus*), black rat (*Rattus rattus*), multimammate mouse (*Mastomys natalensis*), and certain wild rodents, marsupials, and hedgehogs. Its distribution is apparently cosmopolitan, albeit sporadic. The mite burrows into the stratum corneum where the eggs are deposited; they hatch in 4–5 days. The entire life cycle is completed in about 3 weeks. Only occasionally does *N. muris* penetrate the deeper skin tissues. The female lives 2–3 weeks, laying up to three eggs per day. Although the larvae and nymphs may develop in the parent burrow, they also may move onto the skin surface to excavate pits in the stratum corneum, which in turn become the entrances to new burrows as they continue to develop (Sweatman, 1971). Mating takes place in the burrow, and transmission between hosts typically involves direct transfer of the active immature stages.

Lesions usually appear several weeks after the initial infestation, in the form of wartlike, horny excrescences and yellowish encrustations on the ears, nose, neck, and tail, and sometimes the limbs and genitalia. The involvement of the ears and tail are often diagnostic. The skin becomes thickened and hairs appear to become shortened and displaced owing to proliferation and cornification of epidermal cells in the affected areas. Severe cases can develop erythema, vesicular or papular lesions, serous exudates, and other complications as a result of secondary bacterial infections.

Related species (*N. musculi*, *N. oudemansi*, and *N. pseudomuris*) are known from *Rattus* and *Mus* species, as well as from wild rodents and insectivores. These and other *Notoedres* species of rodents show more restricted geographic ranges. The three mentioned are all reported to cause mange similar to that caused by *N. muris*.

Trixacarus Species

Two sarcoptid mites in the genus *Trixacarus* can cause severe skin problems called **trixacaric mange** in guinea pigs and laboratory rats. Like those of *Sarcoptes* and *Notoedres* species, *Trixacarus* females burrow in the upper layers of cornified epithelium, where the eggs are deposited and the larvae and nymphs are found. The duration of each developmental stage and the time required to complete the life cycle are unknown. *Trixacarus* species are much smaller than *S. scabiei* (females are 140−180 µm versus 400 µm in *S. scabiei*) and can be distinguished from adults of the latter by the propodosomal shield reduced to a small circular plate; elongate, sclerotized striae on the dorsal and ventral anterior idiosoma; large, weakly sclerotized denticles on the dorsum; long, spine-like dorsal setae; and the absence of an ambulacral sucker on leg IV in the male.

Trixacarus caviae

This mite (Fig. 26.49) was first described from a guinea pig colony in England in the early 1970s. Subsequently it has been reported to cause mange in laboratory and pet guinea pigs in other parts of Europe and in North America. Lesions begin as an erythematous rash that progresses to a pruritic dermatitis, with thickening and wrinkling of the skin and induced scratching. Lesions vary from dry and scaly to moist and crusty, with associated hair loss. Affected areas include the head and neck, shoulders, back, sides, lower abdomen, axillary region, and inner thighs. The intense pruritus can lead to self-inflicted trauma, such as frantic running about in cages and blindly striking objects, loss of condition, lethargy, and grand mal seizures. Death commonly occurs within a few weeks or months in heavily infested, untreated animals.

Trixacarus caviae is readily transferred upon contact with other guinea pigs, including mothers to neonates, causing rapid spread through colonies. Owners of pet guinea pigs can develop papulovesicular lesions and pruritus by direct contact with infested animals held against the skin. This also is occasionally reported as a problem in people working in animal facilities. For further information on *T. caviae*, see Kummel et al. (1980) and Zenoble and Greve (1980).

Trixacarus diversus

This mite is reported to cause severe infestations in the Norway rat, white mice, and hamsters in animal facilities in Europe and in wild field mice (*Calomys musculinus*) in Argentina (Klompen, 1992). Lesions are similar to those described for *T. caviae*, first appearing between the shoulders and from there spreading to the back and sides. Young or weakened animals may die 2−3 weeks after the appearance of skin problems, whereas untreated adult rats are more likely to die after 5−6 weeks (Sweatman, 1971).

Rhyncoptidae

Rhyncoptid mites are similar to sarcoptid mites but are generally more elongate and are typically restricted to hair follicles. Species of *Audycoptes*, *Saimirioptes*, and most *Rhyncoptes* species occur in monkeys, with other *Rhyncoptes* species in African porcupines, *Caenolestocoptes* in South American marsupials, and *Ursicoptes* species in

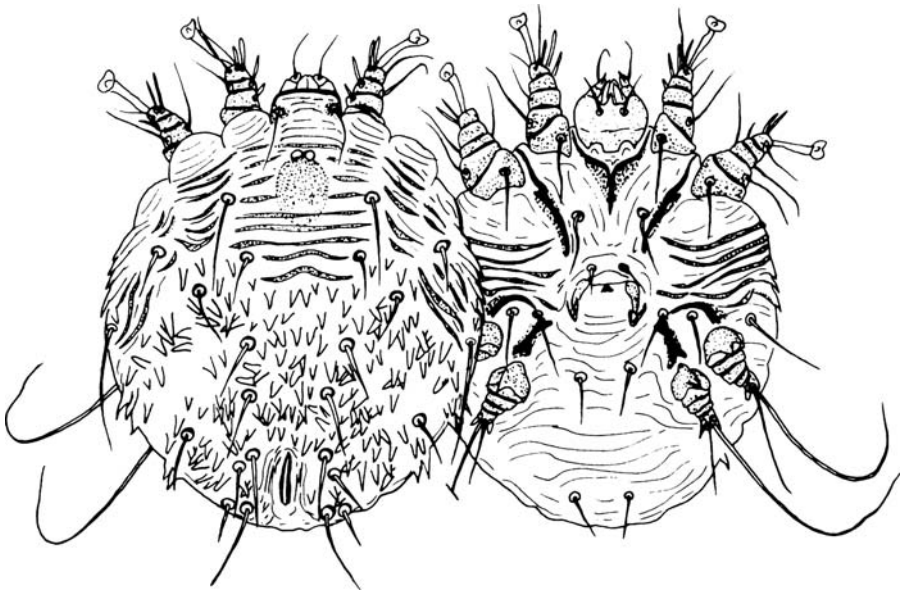


FIGURE 26.49 *Trixacarus caviae* (Sarcoptidae), female; dorsal (left) and ventral (right) views. *From Nutting, 1984.*

bears and raccoons. The primate parasites such as *Rhyncoptes grabberi* from rhesus macaques (Klompen, 1989) are generally innocuous, but mange conditions have been reported associated with *Ursicoptes americanus* in black bears in North America (Yunker et al., 1980) and with *U. procyonis* in North American raccoons.

Psoroptidae

Mites in the family Psoroptidae are mammalian ectoparasites called **scab mites**. All developmental stages occur on the host. They do not burrow into the epidermis but instead live on the surface of the skin. Some pierce the skin with their chelicerae to feed on lymph, blood, and serous exudates. Others have chelicerae adapted for feeding on sloughed skin scales and other epidermal debris. Feeding injury commonly results in inflammation, pruritus, hair loss, crusting, and scab formation. The host hair often becomes matted and, together with the skin, may be severely damaged as a result of biting and rubbing by the host against fence posts and other objects. This can result in extensive loss of hair and generalized debilitation, with death occurring in some cases.

Psoroptid mites of veterinary interest parasitize primarily the Artiodactyla, Perissodactyla, and Carnivora. They are also found on certain edentates, marsupials, insectivores, and primates. Members of four genera cause problems for domestic and laboratory animals. *Psoroptes* mites infest cattle, sheep, goats, horses, and rabbits; *Chorioptes* mites are primarily a problem on cattle; *Otodectes* mites cause problems for cats and dogs; whereas *Caparinia* mites infest wild and captive hedgehogs.

Psoroptic Scab Mites

For many years, it was generally accepted that there were five *Psoroptes* species (*P. cervinus*, *P. cuniculi*, *P. equi*, *P. ovis*, and *P. natalensis*) based on host associations, the location on the host, and the length of the outer opisthosomal setae (L_4) of the adult males (Sweatman 1958b). A sixth species, *P. pienaari* from the African buffalo, was described by Fain (1970a). More recently, morphological and molecular evidence has supported the treatment of four of these (*P. cervinus*, *P. cuniculi*, *P. equi*, and *P. ovis*) as conspecific (Pegler et al. 2005; Wall and Kolbe 2006), with the correct name for the species being *P. ovis* (OConnor and Klimov 2015). Bochkov (2010) regarded *Psoroptes* as including the three species, *P. ovis*, *P. natalensis*, and *P. pienaari*.

Mites in the genus *Psoroptes* (Fig. 26.50) cause **psoroptic mange**, a highly contagious form of mange that can spread rapidly by direct transfer of mites between animals

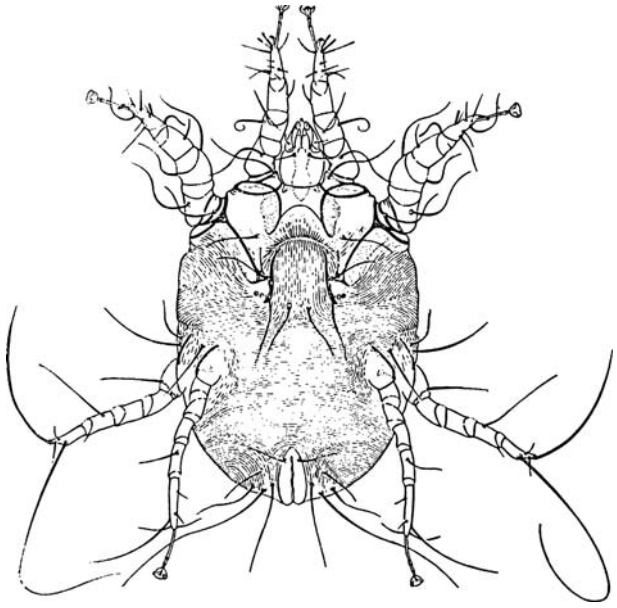


FIGURE 26.50 *Psoroptes ovis* (Psoroptidae), female, ventral view. *From Baker et al., 1956.*

or indirectly by rubbing against fence posts and other objects. Infestations tend to be more prevalent during the winter months, usually subsiding or disappearing during the warmer seasons. *Psoroptes* mites cause more severe problems than any of the other psoroptid genera, commonly resulting in economic losses in cattle, sheep, and goat operations.

The mouthparts of *Psoroptes* species are adapted for feeding on the surface of the skin rather than piercing the epidermis. The mites abrade the stratum corneum with their chelicerae, ingesting lipids and other dermal substances. The host responds antigenically to the mites by developing localized perivascular dermatitis and edema. Hemoglobin and other blood components are ingested by the mites only as a result of small hemorrhages at the skin surface of abraded sites. Their food primarily consists of digested cells of the stratum corneum and skin exudates.

The species of the most veterinary importance is *P. ovis* of sheep, goats, cattle, horses, deer, and rabbits. *Psoroptes natalensis* causes mange in cattle, zebu, Indian water buffalo, and horses, whereas *P. pienaari* can cause mange in African buffalo. *Psoroptes natalensis* occurs in South Africa, South America (Brazil and Uruguay), New Zealand, and possibly France (Sweatman, 1958b), whereas *P. pienaari* is known only from southern Africa. *Psoroptes* infestations of wild animals are rare and seldom cause apparent problems. Exceptions are cases of mange in South American primates such as monkeys, marmosets, and tamarins, especially when held in captivity (Sweatman, 1971).

Livestock Scab Mite (Psoroptes ovis)

This mite causes psoroptic mange in sheep and cattle known as **sheep scab** and **cattle scab**. On different hosts, it may be found to attack the general body; thus, it is also called the **psoroptic body mite**. Otherwise it tends to be largely restricted to the ears. Clinical cases of *P. ovis* are misleadingly called *scabies*, a term that is best reserved for mange caused by *Sarcoptes scabiei*. Although more common in domestic stock, *P. ovis* can be transferred from domestic sheep to wild sheep, causing severe infestations and die-offs in bighorn sheep (*Ovis canadensis*) in the western United States. *Psoroptes ovis* tends to occur in the more densely haired or wooly parts of sheep, with initial lesions usually appearing on the back and sides. Heavy crusting and scab formations with associated inflammation, hair damage, and depilation are typical in animals that become antigenically sensitized to this mite (Fig. 26.51). Heavily infested lambs have been found to have thicker than normal pelts, with matted wool and enlarged lymph glands attributed to *P. ovis* (Cochrane, 1994). Pruritus often induces self-inflicted trauma, with extensive wool loss and skin injury owing to licking, biting, and rubbing. Mites move to the periphery of the affected areas, spreading over the body surface. In untreated animals, lesions may develop over major portions of the body, causing extensive loss of wool and reduced weight gains as high as 30% (Kirkwood, 1980). In extremely severe cases, sheep may die. In other cases, sheep heavily infested with *P. ovis* may show no gross evidence of skin damage yet exhibit adverse effects on wool quality and general body condition.

Transfer of *P. ovis* among sheep primarily involves mites less than 2 weeks old. Older mites apparently fail to establish infestations even upon successful transfer to a new host. When *P. ovis* is transferred from infested sheep to calves and goats, the mites survive only about a week on the recipient host and do not induce clinical signs, at least not in naive, i.e., previously unexposed, animals (O'Brien et al., 1994).

Infestations of *P. ovis* in cattle cause exudative dermatitis and hair loss similar to those in sheep (Fig. 26.52). The severity varies from mild cases to those that can involve virtually the entire body surface. Systemic effects can include mild anemia with hematologic changes such as marked reductions in lymphocytes, neutrophils, and total white blood cells; increases in plasma proteins and fibrinogen; and bone marrow effects (Stromberg et al., 1986; Stromberg and Guillot, 1987a,b). The degree of these responses is directly correlated to the severity of the associated dermatitis. Infested animals also may experience reduced weight gain, reduced energy conversion rates, and higher maintenance energy requirements (Cole and Guillot, 1987).

Significant differences occur between cattle that have not previously been exposed to *P. ovis* and those that have. In naive animals, skin lesions are slower to appear but progress rapidly. The associated mite populations grow much more quickly, reaching densities 100−1000 times those in previously infested animals. The lower growth rates and lower fecundity in previously exposed cattle result from both cellular and humoral immune responses involving the development of antibody activity to live *P. ovis* mites infesting the skin. Mites that infest animals with this acquired immunity have much lower ovipositional rates, reflecting decreases in the number of ovigerous females,

FIGURE 26.51 Psoroptic mange, or sheep scab, in sheep caused by *Psoroptes ovis* (Psoroptidae). Note extensive hair loss. *Courtesy of US Department of Agriculture/Animal Research Service, Kerrville, TX, M&M 8165.*

FIGURE 26.52 Psoroptic mange in calves, caused by *Psoroptes ovis* (Psoroptidae). *Courtesy of the US Department of Agriculture/Animal Research Service, K7268-14.*

rather than detrimental effects on egg development (Guillot and Stromberg, 1987). Cattle can become hypersensitive to *P. ovis* antigens, developing severe clinical disease even when infested by relatively modest numbers of mites. This explains why cattle in areas endemic for *P. ovis* generally experience more severe lesions than do cattle in nonendemic areas. The severity also is exacerbated by stress caused by stanchioning animals and by extremely cold weather that can contribute to hypothermia and the death of infested cattle.

Psoroptic mange also occurs in bighorn sheep, mule deer, elk, and wapiti in the western United States. The mite agent has been called either *P. ovis* or *P. cervinus*, which are now regarded as conspecific. Often these mites are simply referred to as *Psoroptes* sp. because of this taxonomic uncertainty. *Psoroptes* mites collected from bighorn sheep apparently do not establish lasting infestations when transferred to domestic sheep and can be established only with difficulty on cattle (Wright et al., 1981). Comparative studies based on antigenic characterization of *Psoroptes* mites from various hosts further suggest that the mite that infests bighorn sheep and mule deer is different from *P. ovis* on cattle (Boyce and Brown, 1991).

Lesions of psoroptic mange in bighorn sheep occur primarily in the ears or on the face and other parts of the head. The affected areas are characterized by yellowish-white scabs of dried serous exudates and crusty, exfoliated epidermal tissue overlying a reddened and raw epidermis. Other clinical signs are hair loss on the head, neck and back; droopy ears; and blockage of the outer ear canal with cerumen and exudates. Lesions on other parts of the body generally are less extensive or severe, with mites being recovered primarily from the head and ears.

Psoroptes infestations can be especially severe in desert bighorn sheep (*Ovis canadensis mexicanus*) in the San Andres Mountains of New Mexico (United States). High mortality attributed to psoroptic mange reduced the population of desert bighorn sheep in the San Andres National Wildlife Refuge from more than 200 to about 25 individuals during the 12-year period of 1978–89 (Hoban, 1990). Serologic surveys using immunologic tests for detection of antibodies to *Psoroptes* mites have shown widespread prevalence of these mites in desert bighorn sheep populations in California (United States), where lesions tend to be mild and confined to the ears (Mazet et al., 1992).

Psoroptes ovis also parasitizes elk (*Cervus elaphus*) and wapiti (*C. canadensis*) in the western United States, where it is called the **elk scab mite**. Infested elk may develop moist, thick scabs with associated dermatitis and hair loss, especially at the base of the neck and on the dorsal and lateral thorax. Particularly affected are the cows, young males, and calves (Samuel et al., 1991). In wapiti, body lesions occur primarily in the winter months and may involve large areas of the neck, trunk, and upper legs. A wet eczema with overlying scabs and extensive hair loss is similar to that observed in desert bighorn sheep and can be fatal.

Chorioptic Scab Mites

Psoroptic mites in the genus *Chorioptes* cause **chorioptic mange** in domestic ungulates, notably cattle, sheep, goats, and horses. The species of greatest importance is *C. bovis,* which infests each of these hosts. Another species, *C. texanus,* infests the ear canals of reindeer in Canada, whereas *C. sweatmani* occurs on moose in northern Europe. As a group, chorioptic mites are primarily parasites of herbivores (Artiodactyla, Perissodactyla, and Lagomorpha), including llama, guanaco, alpaca, and rabbits. They feed on sloughed epidermal tissues, sometimes causing irritation and crusty, pruritic lesions that warrant treatment. For further information on *Chorioptes* species and their host associations, see Sweatman (1957, 1958c) and Bochkov et al. (2014).

Chorioptes bovis

This mite (Fig. 26.53) occurs primarily on the legs and feet of its hosts, where all of the developmental stages are likely to be found. Eggs are deposited singly at the rate of one egg per day and are attached with a sticky substance to the host skin. Adult females usually live for 2 weeks or more, producing about 14–20 eggs during this time. The eggs are often clustered as multiple females oviposit in common sites, or females return on successive days to deposit their eggs. The eggs hatch in 4 days. The larval and protonymphal stages last 3–5 days each, whereas the tritonymphal stage takes 7–8 days, with 1 day of quiescence between each developmental stage. The cycle is completed in about 3 weeks. Optimum conditions for development are about 35°C and a relative humidity of 80%.

Most animals infested with *C. bovis* do not exhibit noticeable lesions or unusual discomfort owing to this mite, even at relatively high mite densities. As a result, infested sheep and cattle often remain asymptomatic, serving as silent carriers and a source of infestation for other animals. Host reactions are induced only when numbers increase to thousands of mites per host, occasionally causing extensive mange and pruritus. Most of the mites are found on the feet, notably the pasterns, regardless of where lesions appear elsewhere on the body. The irritation in sensitized animals can lead to stamping of feet, rubbing and chewing of legs, and other self-inflicted injury. Body lesions in severe cases are characterized by dermal crusting, erythema, and hair loss.

In cattle, *C. bovis* is found more commonly on the hind feet than on the forefeet, and particularly on the pasterns

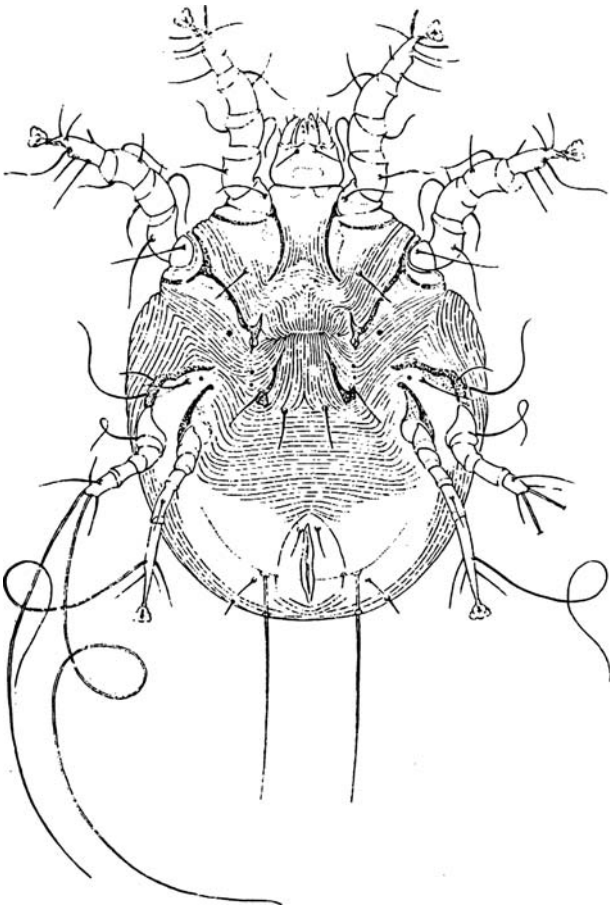


FIGURE 26.53 *Chorioptes bovis* (Psoroptidae), female, ventral view. *From Baker et al., 1956.*

(between the fetlock and hoof). The mites move from the feet to other parts of the body to cause mange of the escutcheon, base of the tail, buttocks, and perineum. This has given rise to names such as **foot mange**, **leg mange**, and **itchy heel** in referring to chorioptic mange of cattle and sheep. Mite populations are highest during the winter and are especially a problem in housed animals. In the spring, their numbers drop sharply and lesions generally disappear when cattle are turned out to pastures.

Although sheep are commonly parasitized by *C. bovis*, the small crusty lesions are hidden beneath the coat and usually go unnoticed. When clinical cases occur, they are typically in the form of foot mange, affecting the forefeet. The mites occur about the accessory digits and along the coronary border of the outer claws, often in clusters, causing crusting primarily below the accessary digits and in the interdigital spaces. Infestation rates of 30%–60% in sheep have been reported in the United States, Europe, Australia, and New Zealand. Prevalence of chorioptic mange tends to be highest in rams and generally low in ewes and lambs.

Chorioptes bovis may spread by direct contact of the feet with other parts of the body, notably the upper parts of the hind legs and scrotal area of rams, causing an exudative dermatitis called **scrotal mange**. The scrotal skin develops thick, yellowish, crusty layers as much as 4 cm deep. In severe cases, elevated scrotal temperatures attributed to allergic responses can cause degeneration of the seminiferous tubules, reduced sperm quality, and complete spermatogenic arrest. Testicular weights may become significantly reduced. The effects are reversible, with seminal regeneration and restored sperm production occurring after treatment for mites or spontaneous recovery of infested rams. Prevalence of leg and scrotal mange is usually highest in the fall and winter months and declines in the spring.

Infestation rates of *C. bovis* tend to be higher in goats than in sheep; up to 80%–90% of goats in individual herds are parasitized (Cremers, 1985). As in sheep, the mites occur most commonly on the forefeet of goats, where the largest numbers of mites and lesions are usually associated with the accessory digits and claws. However, they also may occur on the pastern or higher on the foot. Lesions are generally mild and seldom draw attention.

Chorioptic mange due to *C. bovis* is occasionally observed in horses; Belgian and Frisian breeds are among the more commonly infested. The mites are largely restricted to the pasterns and are most likely to cause foot mange in these horse breeds with long-haired feet (Cremers, 1985). Signs of *C. bovis* infestations in horses include stamping of feet and rubbing one foot against the opposite leg or against some object.

Caparinic Scab Mites

Psoroptid mites of the genus *Caparinia* infest hedgehogs and a few other Old World mammals, causing **caparinic mange**. All active stages of these mites feed on sloughed skin cells and epidermal debris, similar to *Chorioptes* species. Although most infestations tend to go unnoticed, high mite populations can cause severely debilitating conditions and even death of the host. Two species that draw particular attention are *Caparinia tripilis* and *C. erinacei*, both of which parasitize wild and captive hedgehogs.

Caparinia tripilis, the **Eurasian hedgehog mange mite**, infests the Eurasian hedgehog (*Erinaceus europaeus*). It was first recognized in Great Britain in the late 1880s, where it is still occasionally reported and has caused the death of at least one captive hedgehog. This mite was introduced to New Zealand on hedgehogs in the 19th century but did not attract attention there until 1955, after hedgehog populations increased dramatically (Brockie, 1974). More recently, *C. tripilis* has been introduced to the United States via breeding colonies of African hedgehogs for sale as pets. An infested colony has

been reported in New Mexico (Staley et al., 1994), and the death of a pet hedgehog attributed to *C. tripilis* has been documented in Alabama (Mullen, unpublished). The mites tend to gather in clusters on their host and invade the skin of the head and ears, flanks, and inner surfaces of the legs. The affected skin becomes dry and scaly, may become thickened and folded, and may crack and bleed, leading to secondary infections. The common association of hedgehog ringworm (*Trichophyton erinacei*) with heavy *C. tripilis* infestations suggests that invasion of the skin by this fungus may contribute to severity of the resultant mange. In some cases, body spines and hairs may fall out and lesions about the eyes may cause blindness (Brockie, 1974). Heavily infested animals become listless, lose weight, scratch the affected skin, and may abandon their normal nocturnal behavior to become active in the daytime. Male hedgehogs are usually more severely affected than females, with higher mortality occurring in captive than in wild hedgehogs.

Caparinia erinacei, the **African hedgehog mange mite**, infests the African hedgehog (*Atelerix albiventris*). Unlike *C. tripilis*, this mite does not form clusters on its hosts and exhibits low pathogenicity, occurring most abundantly on the dorsal parts of the body and rarely on the face. It has been reported to parasitize more than 70% of wild hedgehogs in Kenya (Gregory, 1981).

Harpirhynchidae

Harpirhynchid mites are typically parasites of birds, in which they invade feather follicles and the skin. Evidence of infestations ranges from small white cysts or lumps associated with individual feathers to large, irregularly lobed, papilloma-like cysts that can occur on any part of the body. The lesions are typically pale yellow with a dry, granular appearance. Histological preparations of the lesions reveal multiple spaces lined by epidermal cells and packed with large numbers of mites, sometimes thousands. The most common genus involved is *Harpyrhynchoides,* which has been reported to cause disfiguring ruffling of feathers and feather loss in a variety of avian hosts (e.g., lorikeets, warblers, eagles) in Australia, the southwestern Pacific region, and North America. Females of *Crassacarus* form large cysts, typically in and around the ear openings of passerine birds. Females of the genus *Ophioptes* excavate small, crater-like pits in the body scales of snakes, where they deposit their eggs. The larvae and nymphs lack legs, feed on host tissues, and develop to adults within the pits. Most reported cases have occurred in colubrid snakes in South America and Australia. Except for localized damage to individual scales, these mites do not cause significant harm to their hosts.

Hypoderatidae

Hypoderatid mites are parasites of birds, and rarely mammals, in which they develop in subcutaneous fat tissue. These are unusual mites in that they are parasitic as deutonymphs, invading host tissues, where they undergo growth and sometimes extreme engorgement. The other life stages are nest-inhabiting detritivores; they are rarely nonfeeding. The most common hypoderatid species is *Hypodectes propus*, which parasitizes pigeons and doves in North America, Europe, and Africa. The mites are visible at necropsy as tiny white nodules embedded in fat tissue just beneath the skin. For further information on hypoderatid mites and their host associations, see Pence et al. (1997).

Mite-Induced Allergies

In addition to sensitization of animals to mites that invade the skin (e.g., demodecid and sarcoptid mites), pets and other domestic animals occasionally develop allergic reactions to nonparasitic mites. Reported cases, for example, have involved mites in the family Acaridae that infest dry pet foods and livestock or poultry feed. Although the ingestion of even heavily mite-infested feed usually causes no apparent harm to animals, some individuals may become sensitized and experience anaphylaxis similar to oral mite allergy in humans.

Internal Acariasis

Approximately 15 families of mites include species that cause internal acariasis in animals of veterinary interest. The most common type is respiratory acariasis, which may involve the nasal passages, nasal sinuses, trachea, bronchi, or pulmonary tissues. Less commonly, mites cause oral, esophageal, gastric, and enteric acariasis, and occasionally they invade other internal organs, the body cavity, lymph, and blood.

Species in five families may be found in oral tissues or the alimentary tract of their hosts. Certain *Radfordiella* species (Macronyssidae) invade the oral mucosa of the gums and hard palate of bats, causing erosion of soft tissues and bone. Heavy infestations can result in significant oral lesions, with loss of teeth and sometimes exposure of the maxillary sinus. A few demodecid species invade the tongue, oral epithelium, and esophagus of bats, lemurs, and mice, but rarely cause noticeable problems. Occasionally *Demodex* mites have been recovered from the alimentary tract of dogs, without evidence of penetrating the epithelial lining. *Gastronyssus bakeri* (Gastronyssidae) attaches to the mucosa of the stomach and duodenum of pteropodid bats, whereas *Paraspinturnix globosus* lives in the anal canal of its bat hosts.

Cytodites nudus (Cytoditidae) has been found in the alimentary canal, peritoneum, and body cavity of chickens and is associated with peritonitis, enteritis, and occasionally death. At least some records of *Cytodites* outside the respiratory system could reflect gross dissections of infested hosts. This mite normally is found in the air sacs, but these collapse upon dissection, possibly leading to misleading reports of *Cytodites* in surrounding organs.

In unusual cases, mites have been found in other internal organs. For example, all stages of some *Demodex* species have been found in the liver and spleen (Kirk, 1949), whereas *Cytodites nudus* has been recovered from surface tissues of the heart, liver, and kidneys (Baker et al., 1956). *Demodex* species also have been found alive in lymph nodes, lymphatic vessels, and circulating blood. It is presumed that the mites invade the lymphatic and circulatory systems, where extensive destruction of the surrounding dermal tissue occurs in severely infested hosts.

The two most common internal sites of animals that parasitic mites have exploited are the ear canals and respiratory passages.

Ear Mites

Several species of mites infest the ears of domestic and wild animals, causing problems that may warrant veterinary attention. The families most commonly involved are Psoroptidae, Trombiculidae, and Raillietidae.

Psoroptic Ear Mite (Psoroptes ovis)

Psoroptic ear mites have generally been referred to as *P. cuniculi* but are now regarded as *P. ovis* (see discussion of psoroptic scab mites). Known as the **psoroptic ear mite** or **ear mange mite** of rabbits, *P. ovis* causes lesions in laboratory rabbits and commercial rabbit operations (Fig. 26.54). Such infestations are referred to as **psoroptic otoacariasis**. This cosmopolitan mite also infests the ears of sheep, goats, horses, and occasionally deer, antelope, and laboratory guinea pigs. Lesions occur primarily in the ears, causing crust formation, malodorous discharges in the external ear canal, and behavioral responses such as scratching the ears, head shaking, loss of equilibrium, and spasmodic contractions of neck muscles (torticollis). In severe cases, *P. ovis* infestations may spread to other parts of the body, notably the face, neck, and legs.

In the ears, *Psoroptes ovis* lives its entire life under the margins of scabs formed at infested sites. There, the eggs are deposited and hatch in 4 days. The complete life cycle takes about 3 weeks. All stages of this nonburrowing mite pierce the stratum corneum to feed on epidermal tissue. Transmission between animals occurs by direct contact.

Infestations of sheep with *P. ovis* can cause varying degrees of problems in the ears. Lesions in lambs are generally mild and characterized by small, discrete, crusty lesions on the inner surface of the pinnae and around the entrance to the outer auditory canal. Similar lesions may occur at the base of the ears. Severe infestations in older animals can lead to inflammation of the ears (otitis), hematomas, and suppurating abscesses. In other cases, sheep may show no clinical signs despite confirmation of mites in the ear canal on otoscopic examination. Survey of sheep flocks in England have shown that the prevalence of *P. ovis* is usually higher in lambs than in adults and that up to 60% of some infested flocks are positive for this mite (Morgan, 1992).

Both domestic and wild goats are subject to *P. ovis* infestations. Prevalence rates as high as 80%–90% have been reported in dairy goats, including both kids and adults, in the United States (Williams and Williams, 1978). Goats less than 1 year old generally exhibit much higher infestation rates than do older animals. Clinical signs of *P. ovis* mites in kids are often observed as early as

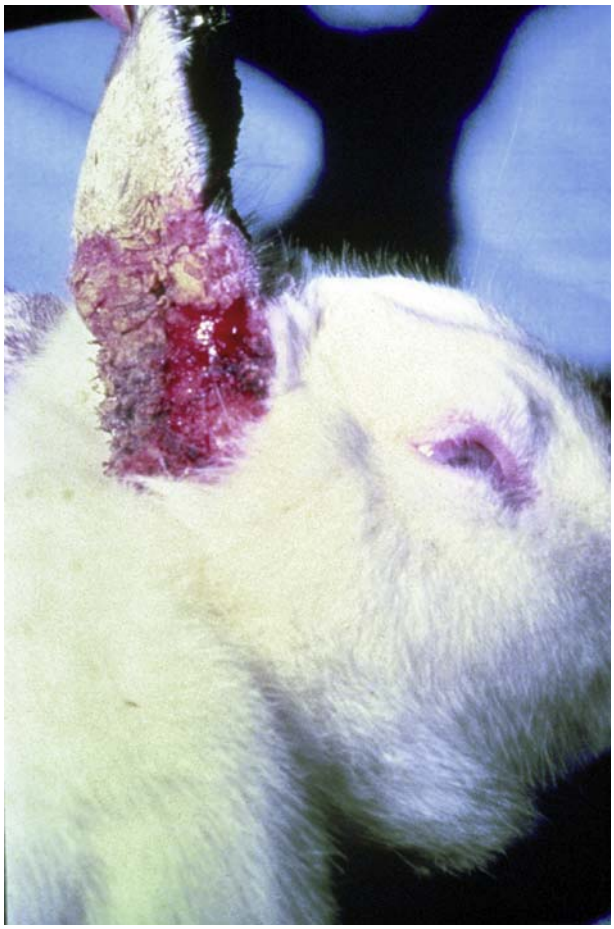


FIGURE 26.54 Rabbit with ears heavily infested by rabbit ear mite, *Psoroptes ovis* (Psoroptidae). Note skin injury, crusty scabs, and bleeding caused by rubbing and scratching. *Courtesy of Department of Pathobiology, Auburn University College of Veterinary Medicine.*

3 weeks after birth, reflecting transfer of mites between mother and young. By 6 weeks of age, most kids in infested goat herds are likely to harbor these mites. There is no evidence, however, for cross-infections between goats and sheep, even when held in common enclosures (Williams and Williams, 1978). Infestations can cause scaling, crusting, inflammation, and hair loss about the ears; accumulations of wax in the external ear canal; ear scratching, head shaking, and rubbing of the head and ears against objects in an effort to alleviate the discomfort. Chronic infestations also can lead to anemia and weight loss. Extreme cases are sometimes fatal; death is preceded by circling, violent fits, and other aberrant behavior. Nondomestic goats reportedly parasitized by *P. ovis* include the Nubian mountain goat (*Capra ibex nubiana*) and cross-breeds between domestic and mountain goats, such as Yaez.

Cervid hosts of *P. ovis* include both free-ranging and captive white-tailed deer (*Odocoileus virginianus*) and mule deer (*O. hemionus*) in North America. Most reports have come from the southeastern United States, where up to 80% of some white-tailed deer populations have been found to be infested. Cases are typically mild, with loss of hair about the ears and base of the antlers and yellow crusty lesions, accumulated cerumen (ear wax), and serous exudates in the external ear. In more severe infestations, the ear canal may become inflamed and infected, leading to pyogenic bacterial otitis and neurologic disorders (Rollor et al., 1978). In such cases, the infected ear canal and tympanic cavity become filled with mucopurulent exudates, with or without damaging the tympanic membrane. This affects the sensory organs in the inner ear, causing neurologic signs such as excessive salivation, circling behavior, difficulty standing, loss of muscular coordination, and torticollis.

Psoroptes ovis is the common ear mange mite of laboratory rabbits that causes **psoroptic ear canker**. Occasionally, wild rabbits and hares (e.g., *Lepus europaeus* in Europe) also are affected. Ear infestations are characterized by loose crusty lesions, excessive cerumen, inflammation, accumulated exudates, and necrotic debris in the external ear. In some heavy infestations, lesions may spread from the ears to other parts of the body, including the face, neck, and genitalia. Often the first signs are behavior such as tilting or shaking of the head, drooping ears, and scratching or self-inflicted trauma to the ears. Inflammation of the middle ear (otitis media) and brown, malodorous discharges in the external auditory canal are common. Cases can become complicated by bacterial infections, causing loss of equilibrium, torticollis and, in some cases, fatal meningitis.

Other animals occasionally infested with *P. ovis* in the ears are horses, donkeys, mules, antelopes, and guinea pigs. Crusty lesions with accumulated exudates in the ear canal are typical of other hosts. In heavy infestations, mites may spread to other parts of the body (e.g., face, belly, hind legs), causing erythematous and pruritic lesions complicated by secondary bacterial infections. Severe cases of this nature have been reported in the blackbuck antelope (*Antilope cervicapra*) (Wright and Glaze, 1988) and guinea pigs (Yeatts, 1994).

Otodectic Ear Mite (Otodectes cynotis)

This mite (Fig. 26.55) is known as the ear mite or ear canker mite of cats and dogs and as the cause of **otodectic mange**. It occurs worldwide and parasitizes other carnivores such as foxes, ferrets, wolverines, and raccoons. *Otodectes cynotis* is closely related to *Psoroptes* species, which it resembles in size and general appearance. It can be distinguished from *Psoroptes*, however, by its short, unsegmented tarsal stalks supporting the ambulacral suckers in both sexes and the greatly reduced hind pair of legs in the female, which terminates in two long, whiplike setae.

Otodectes cynotis typically occurs deep in the external ear canal, where all of the developmental stages are found. Occasionally it secondarily infests other parts of the body including the head, back, tip of the tail, and feet. It does not burrow into the skin but lives as a surface parasite that may pierce the skin to feed on blood, serum, and lymph. Some workers contend that it feeds more commonly on desquamated epithelial cells and possibly cerumen or other aural exudates. It is believed that development of clinical signs reflects allergic hypersensitivity on the part of the host to antigenic substances introduced while the mites are feeding. This can lead to highly variable responses ranging from asymptomatic or mild cases to severe otitis and convulsive seizures.

The ear canals of animals infested with *O. cynotis* become excessively moistened with accumulations of cerumen and purulent, brown-black exudates resembling coffee grounds. This is accompanied by inflammation and pruritus usually involving both ears. As a result of intense itching, infested cats and dogs scratch their ears, shake their head or hold it to one side, and may turn in circles. When the ear canal is massaged, the animal typically responds with pleasurable grunting sounds and by thumping its hind leg on the corresponding side. Severe, untreated cases can lead to emaciation, self-induced trauma, spasms, and convulsions, especially in cats. Diagnosis of *O. cynotis* is confirmed by otoscopic examination and by recovering the mite from aural scrapings.

Cattle and Goat Ear Mites (Raillietia spp.)

Mites of the genus *Raillietia* (family Halarachnidae) are the only known mesostigmatid species that live in the ear canals of domestic animals. The most widespread species

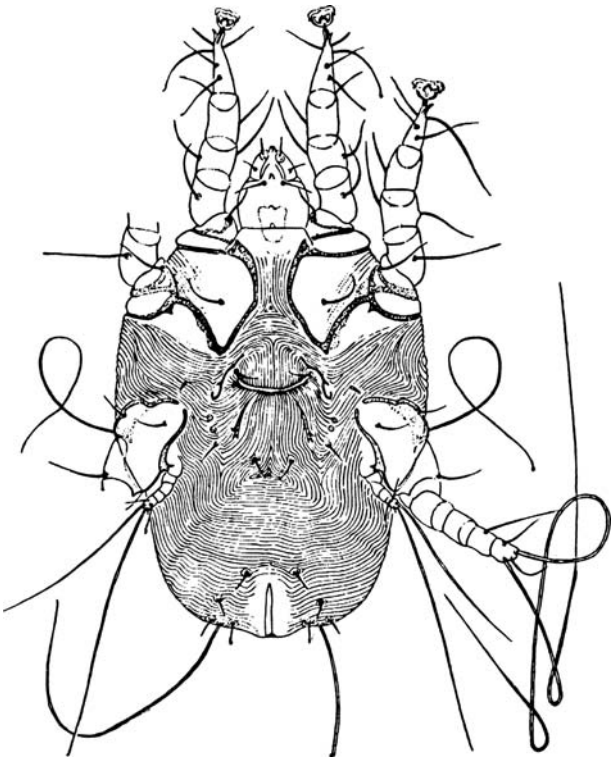


FIGURE 26.55 Ear mite of cats and dogs, *Otodectes cynotis* (Psoroptidae), female, ventral view. *From Baker et al., 1956.*

is the cattle ear mite (*R. auris* (Fig. 26.56), which infests dairy and beef cattle in North America, South America, Europe, western Asia, and Australia. Although it is generally considered to be a relatively harmless mite

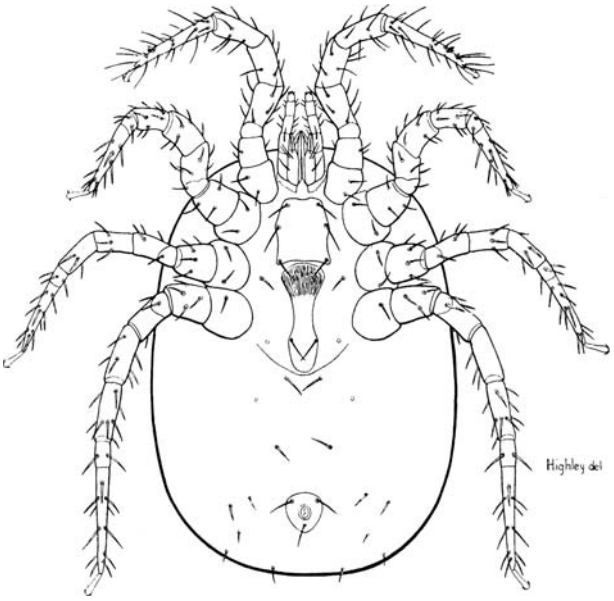


FIGURE 26.56 *Rallietia auris* (Halarachnidae), female, ventral view. *From Hirst, 1922.*

living in cerumen, *R. auris* can cause blockage of the auditory canal by plugs of paste-like wax. Severe cases can result in inflammation of the ear canal, pus formation, ulcerated lesions, and hemorrhaging, with accompanying hearing loss in some animals. Based on studies of *R. flechtmanni* that infests cattle and buffalo (*Bubalus bubalis*) in Brazil, adult mites on pasture vegetation enter the ear canals of grazing animals, where they feed, mate, and oviposit. Upon hatching from the eggs, larvae leave the host to complete their development as nymphs and adults on pasture (Costa et al., 1992). Other *Raillietia* species include *R. caprae* and *R. manfredi* of goats in Brazil and Australia, respectively; *R. acevedoi* of the Alpine ibex (*Capra ibex*) in Europe and Asia; *Raillietia* species infest the ear canals of the waterbuck (*Kobus ellipsiprymus*) and Uganda kob (*Kobus kob*) in Africa, banteng (*Bos javanius*) in Indonesia, and wombat (*Vombatus ursinus*) in Australia. *Raillietia caprae* has been implicated in mycoplasma infections of goats (DaMassa et al., 1992).

Histiostomatid Mites

Occasionally, mites of the family Histiostomatidae, called slime mites, have been found infesting the outer ear canal of large mammals, including elephants, African buffalo, horses, and donkeys. The genera involved are *Auricanoetus*, *Loxanoetus*, and *Otanoetus*. A related, undescribed species also has been found living and reproducing in the ear canal of a human (Al-Arfaj et al., 2007). Although these mites have been associated with ear infections, they probably have a secondary role, rather than causing the initial infection.

Respiratory Mites

Representatives of several families of mites are specialized, obligate parasites in the respiratory tracts of reptiles, birds, and mammals. They commonly live in the nasal passages and lungs, causing nasal acariasis and pulmonary acariasis**,** respectively. Among the taxa of veterinary interest are members of the families Entonyssidae, Rhinonyssidae, Halarachnidae, Ereynetidae, Trombiculidae, Lemurnyssidae, Turbinoptidae, Cytoditidae, Pneumocoptidae, and Gastronyssidae. For keys to species, host lists, and a bibliography for nasal mites of North American birds, see Pence (1975). The Gastronyssidae, Lemurnyssidae, and Pneumocoptidae were reviewed by Bochkov et al. (2008).

Entonyssidae

These mites are endoparasites that infest the tracheae and lungs of snakes (Fain, 1961). They rarely seem to cause

problems for their hosts, but occasionally induce congestion of the lungs when mite numbers are high.

Rhinonyssinae

Rhinonyssid mites are endoparasites in the nasal passages and occasionally the tracheae of birds throughout the world. They are common, typically infesting 30%–50% of birds examined in local surveys (Domrow, 1969; Pence, 1973; Spicer, 1987). Their chelicerae are reduced, membranous structures that are used to imbibe liquid food, including blood, from their hosts as they crawl about on the mucous membranes lining the nasal airways. Feeding is facilitated by the claws on leg I that are used in lieu of the chelicerae to tear or otherwise penetrate respiratory tissues. Such injury can cause rhinitis or sinusitis, especially in heavy mite infestations. In most cases, however, infested birds do not experience apparent respiratory problems.

An exception is *Sternostoma tracheacolum* (Fig. 26.57), which parasitizes the tracheae, bronchi, parenchymal lung tissue, and air sacs of both wild and captive birds. It does not occur in the nasal cavities. Typical hosts are canaries, parakeets, swallows, and finches. This mite is sometimes called the canary lung mite because of the respiratory problems it causes in captive canaries. Little is known about the behavior of *S. tracheacolum* except that it crawls freely about in mucous lining the trachea and bronchi. The mite also may invade the air sacs and lung tissue, where it dies and disintegrates. Its presence causes inflammation and the development of characteristic nodular lesions containing masses of dead mites and purulent, fibrous exudates. Early signs of respiratory distress include listlessness and difficulty breathing.

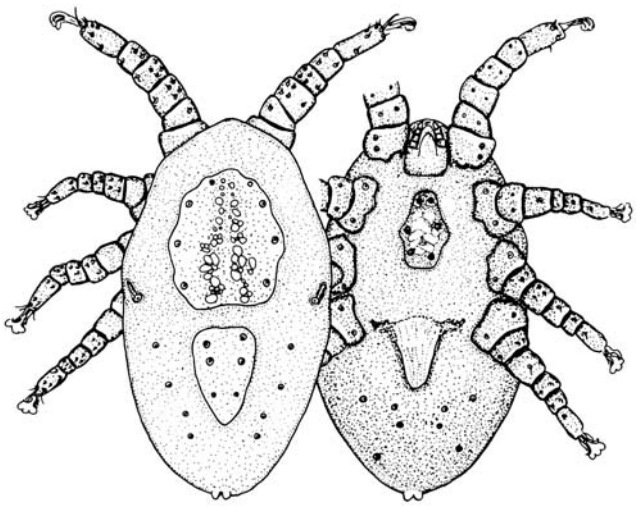


FIGURE 26.57 *Sternostoma tracheacolum* (Rhinonyssidae), female; dorsal (left) and ventral (right) views. *From Nutting, 1984.*

Heavy infestations can result in tracheitis, bronchitis, hemorrhaging in parenchymal tissue surrounding the terminal bronchi, small foci of bronchial pneumonia, lung congestion, and pneumonitis. As the damage progresses, infested birds may become emaciated and die. Although wild birds are not as severely affected as captive ones, they can develop bronchopneumonia and inflammation of the air sacs.

Halarachnidae

Mites in this family are obligate parasites in the respiratory tracts of a variety of mammals. Their hosts include marine mammals, porcupines, squirrels, canids, and nonhuman primates. Halarachnid mites generally are regarded as benign, except for a few species that infest domestic dogs and captive primates. Most species occur in the nasal passages, whereas others may live in sinuses, tracheae, bronchi, or lung tissue. The larvae and adults are the active stages, typically piercing the epithelium with their long chelicerae to feed on lymph and other fluids. Only a few taxa are known to feed on blood. Transmission is presumed to occur by direct transfer of larvae around the host nostrils or by sneezing and coughing of infested animals. Although most infestations are relatively asymptomatic, others can result in inflammation of the respiratory passages, pulmonary nodules, lung congestion, and host death. Diagnosis of halarachnid infestations is usually based on recovery of larvae in tracheobronchial washings or histological examination of pulmonary tissues. The genera of particular veterinary interest are *Pneumonyssus* of Old World monkeys and apes and *Halarachne* and *Orthohalarachne* of seals, sea lions, sea otters, and walruses.

Pneumonyssus simicola, the monkey lung mite (Fig. 26.58), is the most common halarachnid mite of primates, infesting the lungs of rhesus, cynomolgus, and macaque monkeys in Africa. It is especially a problem in rhesus colonies, in which up to 100% of the individuals may be infested. Although relatively benign in wild hosts, infestations of *P. simicola* in captive primates in laboratories and zoological parks can cause a wide range of respiratory problems, occasionally proving fatal. The damage results directly from mites attached to the bronchiolar walls, piercing the surrounding parenchyma to feed on blood, lymph, and pulmonary epithelial cells. The severity depends largely on the number of mites. Low levels of infestation can cause inflammation of the bronchioles and mild coughing or sneezing. As the number of mites increases, lesions are produced in the form of soft, yellowish nodules containing up to 20 mites each. Other lesions appear as pale spots containing golden-brown, needlelike crystals and dark pigments. The latter are believed to be breakdown products of host blood excreted by the mites. Deaths, in cases of massive infestations, are attributed to congestion of the lungs and

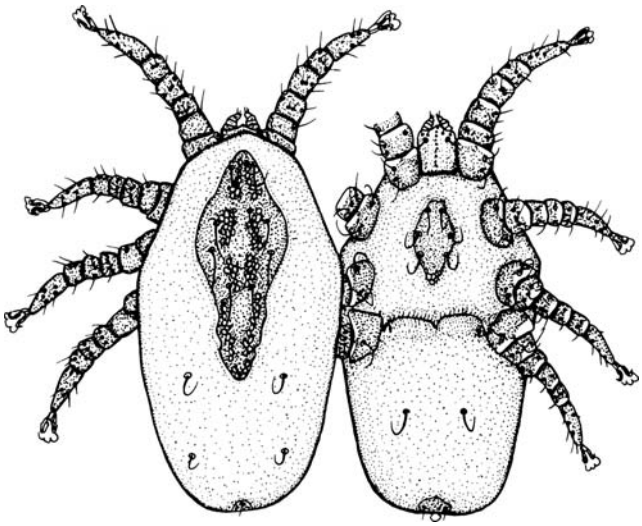


FIGURE 26.58 Monkey lung mite, *Pneumonyssus simicola* (Halarachnidae), female; dorsal (left) and ventral (right) views. *From Nutting, 1984.*

alveolar collapse. For additional information on *Pneumonyssus* species, see Hull (1970).

Halarachne species are nasal mites of earless seals (Phocidae) and the Pacific sea otter (*Enhydra lutris*). Captive sea otters may develop heavy infestations of *H. miroungae*, resulting in inflammation of the nasal mucosa, obstruction of nasal passages, destruction of associated bony tissues (turbinates), and pulmonary congestion. More than 3000 mites were reported infesting one sea otter that had died. For a key to *Halarachne* species and a review of the genus, see Furman and Murray (1980). Sea lions and fur seals (Otariidae) and walruses (Odobenidae) are hosts for *Orthohalarachne* species. *O. elongata* infests the nasopharynx, whereas *Orthohalarachne diminuta* infests the lungs. Fur seals often are simultaneously parasitized by both of these mites; entire populations of seals over 3 months old may be infested. Clinical signs are mucus-filled turbinates, nasal discharges, sneezing, coughing, and impaired respiration. Heavy infestations can lead to alveolar emphysema and a predisposition to more serious ailments that can kill host animals (Kim et al., 1980).

Pneumonyssoides caninum, the **dog nasal mite** (Fig. 26.59), lives in the nasal sinuses of dogs (Fig. 26.60) in many parts of the world, including the continental United States and Hawaii, Europe, South Africa, and Australia. Details about its life cycle are largely unknown. This mite is regarded as relatively nonpathogenic; clinical signs of most infestations are limited to excessive nasal secretions and hyperemia of the nasal mucosa. More severe cases may involve listlessness, loss of appetite, tearing of the eyes, chronic sneezing, bronchial cough, and rhinitis or sinusitis (Koutz et al., 1954). There also is evidence that *P. caninum* can penetrate host tissues and move beyond the respiratory system to cause lesions in the liver and kidneys (Garlick, 1977).

Ereynetidae

Ereynetid mites are primarily free-living detritivores. However, members of two subfamilies are obligate parasites in the respiratory tracts of terrestrial vertebrates. Mites of the subfamily Lawrencarinae infest the nares and nasal passages of amphibians, notably African frogs and toads. Examples are *Lawrencarus eweri* and *Xenopacarus africanus*. Although both feed on tissue fluids including blood, it is not clear how much harm they cause to their hosts.

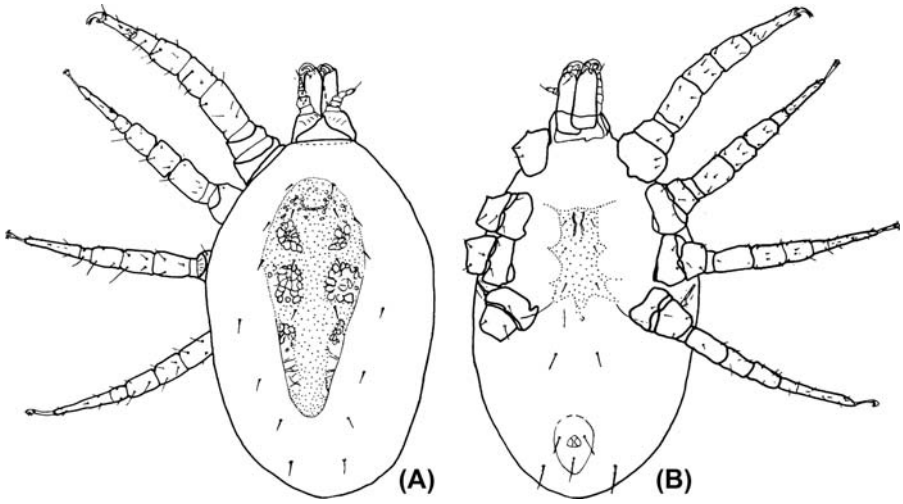


FIGURE 26.59 Dog nasal mite, *Pneumonyssoides caninum* (Halarachnidae), male. (A) Dorsal view; (B) ventral view. *From Strandtmann and Wharton, 1958.*

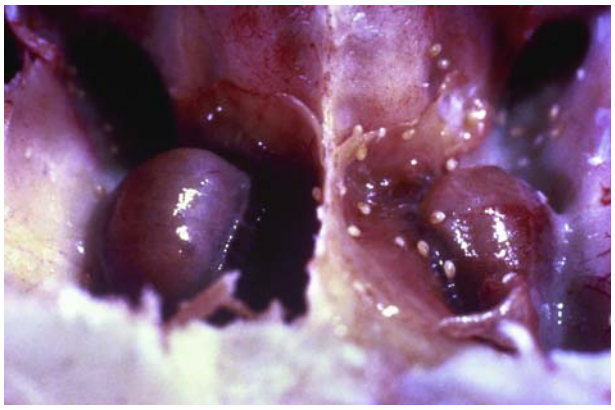


FIGURE 26.60 Nasal sinuses of dog infested with *Pneumonyssoides caninum* (Halarachnidae); whitish mites are seen crawling over the tissue surface. *Courtesy of Department of Pathobiology, Auburn University College of Veterinary Medicine.*

Mites of the subfamily Speleognathinae live in the mucus-lined nasal passages of a wide range of birds and mammals throughout the world. Occasionally, they also invade the lungs. They rarely seem to harm their hosts although some species feed on blood (e.g., *Boydaia sturnellus* of meadowlarks, the United States). *Speleognathus australis* parasitizes cattle and bison. Unlike other respiratory mites, which are typically found embedded in mucous, the cuticle of ereynetids is hydrophobic and the mites skate about on the surface of the mucosa. For additional information on speleognathine mites, see Lawrence (1952), Clark (1960), Baker (1973), Pence (1973, 1975), Fain and Hyland (1975), and Spicer (1987).

Trombiculidae, Leeuwenhoekiide, and Walchiidae

Approximately 20 genera of trombiculoid mites are parasitic as larvae (**chiggers**) in the nasal passages of reptiles, birds, and mammals in both the Old and New Worlds. Rodents and bats are the most common hosts, parasitized by *Ascoschoengastia*, *Doloisia*, *Gahrliepia*, *Microtrombicula*, *Schoutedenichia*, and other genera. Other hosts of intranasal chiggers include marsupials (e.g., common ringtail possum, water opossum, bandicoots), edentates (e.g., armadillos and anteaters), hyraxes (e.g., tree hyrax), lagomorphs (e.g., hares), birds (e.g., sooty tern), felids (e.g., African wild cat), marine iguanas, and sea snakes (Nadchatram, 1970). *Vatacarus* species, which parasitize the latter two groups of reptiles, infest not only the nasal fossae but also the tracheae and lungs of their hosts. The nymphs and adults of intranasal mites are free-living and presumably inhabit the nests, dens, and other sheltered locations of their respective hosts. Virtually nothing is known about possible adverse effects that these chiggers have on their hosts.

Lemurnyssidae

Mites in this psoroptoid family occur as intranasal parasites of lorisid primates (lorises and bush babies) in Africa and monkeys (Cebidae) in South America (Fain, 1964b). They live in the nasal fossae, where they apparently cause little or no harm to their hosts.

Turbinoptidae

Members of this family are exclusively intranasal mites of birds and occur in both the Old and New Worlds. They infest the nasal fossae without causing apparent harm to their avian hosts. For a review of North American turbinoptid mites, keys to genera and species, and a list of hosts, see Pence (1973, 1975). For African and European species, see Fain (1957, 1970b); for eastern Australia species, see Domrow (1969).

Cytoditidae

Mites in this family are internal parasites of birds that typically infest the respiratory system but also may invade the peritoneum and visceral organs. Members of the genus *Cytonyssus* are usually found only in the nasal passages, whereas *Cytodites* species typically inhabit the lungs and air sacs. The species of greatest veterinary importance is *C. nudus*, the air-sac mite of chickens (Fig. 26.61), which

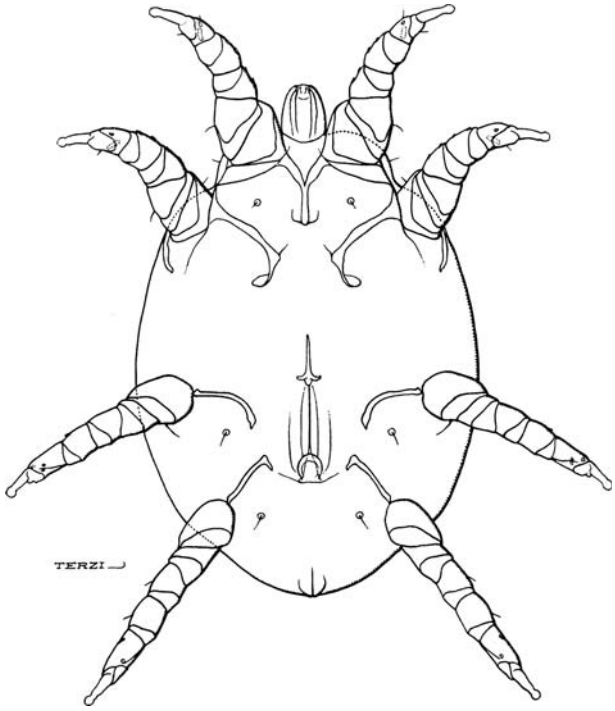


FIGURE 26.61 Air-sac mite of chickens, *Cytodites nudus* (Cytoditidae), female, ventral view. *From Hirst, 1922.*

occurs worldwide. Although low-level infestations do not cause apparent harm, heavy infestations can lead to severe clinical signs and occasionally host death. *Cytodites nudus* is found most commonly in the lining of the air sacs, but it also can invade the air passages and lungs, causing the accumulation of mucus in the tracheae and bronchi. Clinical signs include coughing, obstruction of air flow, pulmonary edema, and pneumonia. Infested birds may exhibit weight loss, general weakness, and loss of balance or coordination. In some cases, *C. nudus* invades the body cavity and visceral organs, including the heart, alimentary tract, liver, and kidneys. It shows a particular predilection for the peritoneum. Deaths are usually associated with peritonitis, enteritis, emaciation, and respiratory complications.

Cytodites nudus has also been reported to infest canaries and ruffed grouse, and in one case it was reportedly recovered from the peritoneum of a human in Uganda (Castellani, 1907). Symptoms in canaries are similar to those in chickens but may include bulging eyes and sores or swellings at the corners of the beak (Higby, 1946). Little is known about the life history of this mite. Transmission is presumed to be by coughing.

Pneumocoptidae

Members of this family infest the lungs of rodents. *Pneumocoptes jellisoni* and *P. penrosei* are common pulmonary parasites of prairie dogs (*Cynomys* spp.) and mice (*Peromyscus* and *Onychomys* spp.) in the midwestern United States; *P. banksi* parasitizes the California ground squirrel (*Spermophilus beecheyi*), whereas *P. tiollaisi* parasitizes voles (*Myodes* spp.) in Europe. These mites rarely cause apparent harm to their hosts. A possible exception is a case involving a captive black-tailed prairie dog (*Cynomys ludovicianus*) at the Philadelphia Zoological Gardens (the United States), which died of acute bronchopneumonia. Large numbers of *P. penrosei* were found to infest the lungs at necropsy, with postmortem evidence of emphysema and dilatation of the bronchi and bronchioles (Wiedman, 1916).

Gastronyssidae

Gastronyssid mites are primarily intranasal parasites of Old World rodents and bats. Examples include *Opsonyssus* and *Rodhainyssus* species in the nasal fossae of African and Asian bats, and *Sciuracarus paraxeri* in the nasal fossae of South African sun squirrels (Fain 1956, 1964a, 1967). *Opsonyssus* species also have been reported to infest eye orbits, whereas *Gastronyssus bakeri* lives in the alimentary tract of fruit bats (Pteropodidae). *Yunkeracarus* is common in a diversity of rodents worldwide. Any deleterious effects they may have on their respective hosts remain unknown.

Mite-Borne Diseases

With the exception of ticks and chiggers, only a few species or groups of mites are known to have a significant role in transmitting disease agents to domestic and wild animals. In many cases, evidence of mite involvement is circumstantial, based primarily on experimental studies. *Dermanyssus gallinae*, for example, has been shown to transmit the viral agents of fowl pox, St. Louis encephalitis, and eastern equine encephalitis among birds, for which mosquitoes are the usual natural vectors. Similarly, *Cheyletiella parasitivorax* is capable of experimentally infecting rabbits with the virus causing myxomatosis. Other examples include viruses that cause Newcastle disease of birds involving *Ornithonyssus sylviarum* and western equine encephalitis involving *Dermanyssus americanus*. Perhaps more significant is experimental evidence that *Ornithonyssus bacoti* possibly serves as both a vector and reservoir of Hantaan virus, the causative agent of Korean hemorrhagic fever that infects mice and humans (Meng et al., 1991).

The importance of mites in the ecology of these diseases should be viewed with caution. The isolation of viruses and other pathogens from naturally infected mites, in the absence of documented evidence of transmission capabilities and natural host associations, should not be construed as evidence that such mites necessarily have a role in transmitting the respective disease agents.

With the exception of rickettsial organisms transmitted by ticks and chiggers, bacterial disease agents of animals seldom involve mites as vectors. However, some reports suggest the possible role of hay-infesting mites as reservoirs for the causative agents of scrapie in sheep and bovine spongiform encephalopathy, also known as mad cow disease, in cattle (Wisniewksi et al., 1996). Both diseases are believed to be caused by brain-destroying proteins called prions.

The only significant protozoan disease agents of vertebrates transmitted by mites are blood parasites of the hemogregarine genus *Hepatozoon* that cause **hepatozoonosis**. Their life cycles are similar in many ways to *Plasmodium* species that cause malaria. *Hepatozoon* species parasitize a wide range of hosts, including amphibians (anurans), reptiles, birds, and mammals, in which they undergo development and multiplication in the liver and circulating blood cells. Hematophagous mites ingest infected blood cells while feeding. The *Hepatozoon* parasites are released in the lumen of the mite midgut, where they penetrate the gut wall to enter the hemocoel. There, they form oocysts containing sporozoites. Vertebrates become infected by ingesting mites with infective oocysts. Among the more common hosts of *Hepatozoon* species that use mites as vectors are rodents (e.g., field mice, voles, rats, squirrels), skinks, and other lizards. Mites involved are typically mesostigmatid species, including the genera

Haemogamasus, *Laelaps*, and *Ophionyssus*. Three *Hepatozoon* species are known to infest domestic animals: *H. canis* of dogs, *H. felis* of cats, and *H. muris* of laboratory cotton rats. In addition to mites, many hematophagous insects serve as vectors of other *Hepatozoon* species, including fleas, triatomine bugs, sucking lice, mosquitoes, sand flies, and tsetse flies. For further information on *Hepatozoon* species and their hosts, see Smith (1996).

The only notable nematode parasite of domestic animals transmitted by mites is the filarial worm *Litomosoides sigmodontis* (formerly *L. carinii*), the causative agent of cotton rat **filariasis**. This nematode occurs in the southeastern United States, where it is transmitted among cotton rats (*Sigmodon hispidus*) and squirrels by the tropical rat mite *Ornithonyssus bacoti*. Related *Litomosoides* species occur in Central and South America, where their hosts include wood rats (*Neotoma* spp.), marsh rats (*Holochilus* spp.), and the house mouse (*Mus musculus*). Microfilariae of *L. sigmodontis* in host blood are ingested by *O. bacoti*, where they develop into infective third-stage larvae. Rodents become infested when the mite takes a subsequent blood meal. The nematodes become established in the pleural cavity (also the peritoneal cavity in heavy infestations), where they develop into adults. Laboratory-infected cotton rats subjected to chronic reinfection with *L. sigmodontis* tend to lose weight, become feverish, exhibit shallow respiration, and undergo behavioral changes, e.g., sitting in a hunched position with raised fur.

The tropical rat mite (*Ornithonyssus bacoti*) is a vector of *Litomosoides carinii*, a filarial nematode in rats and other wild rodents. The cotton rat (*Sigmodon hispidus*) is a common host in the southern United States. The mites ingest microfilariae with blood from infested rats. The microfilariae penetrate the gut wall and move through the hemocoel to invade the salivary glands, fat-body cells, coxal glands, and glands associated with the female reproductive organs. There, they develop into infective third-stage larvae in about 2 weeks. Infective larvae are introduced to rodent hosts when the mite subsequently feeds, developing into adult worms.

Mites as Intermediate Hosts of Tapeworms

Free-living oribatid mites serve as intermediate hosts for about 27 species (14 genera) of tapeworms in the family Anoplocephalidae. Of this number, approximately 20 species are parasites of domestic animals (Table 26.4). The most important anoplocephalan genus that infests domestic animals is *Moniezia*, represented by two species that parasitize ruminants worldwide. *Moniezia benedeni* is primarily a parasite of cattle, whereas *M. expansa* is primarily a parasite of sheep and goats. These tapeworms are especially prevalent in young host animals less than 6–8 months old. Older animals tend to be less susceptible and after 2 years of age seldom have more than one or a few worms.

Most anoplocephalid tapeworms cause little apparent harm to their hosts even when the parasite burden is high. In some cases, however, they can cause loss of weight gain, unthriftiness, colic, and intestinal blockage that may acquire veterinary attention. Sheep are the most adversely affected, whereas cattle and horses seldom experience significant health problems owing to cestodes associated with oribatid mites. Heavy infestations can lead to severe health problems, especially in Asia, that involve weight loss, reduced wool yield, anemia, enteritis, diarrhea, intestinal obstruction, toxemia, convulsions, and death. In the case of *Stilesia hepatica*, which infests the bile ducts of sheep, economic losses can result from condemnation of sheep livers at meat inspection. For further details on the biology and pathogenic consequences of anoplocephalid tapeworms infesting domestic animals, see Graber (1959), Soulsby (1965, 1982), Narsapur (1988), and Kauffmann (1996).

Although tapeworms of the genus *Avitellina* are common parasites of domestic ruminants in Europe, Asia, and Africa, they do not appear to harm their hosts significantly. The taxonomy of *Avitellina* remains unstable, with several long-recognized species (e.g., *A. goughi*, *A. lahorea*, *A. sudanea*) being regarded as synonyms of the widespread species *A. centripunctata* by some workers (Raina, 1975) but as valid species by others (Malhotra and Capoor, 1982). At the same time, there is evidence that *A. centripunctata* is represented in Africa as a complex of at least five cryptic species (Ba et al., 1994). This has complicated the interpretation of any meaningful associations of individual oribatid species as intermediate hosts of several *Avitellina* species.

The development and life history of *Moniezia* species is representative of anoplocephalid tapeworms in general. The proglottids of adult tapeworms containing eggs are passed in the host feces, contaminating pasture grasses associated with oribatid mites. The mites feed on the eggs by breaking the shell with their chelicerae and ingesting the developing embryo, or oncosphere. In the mite, the oncosphere penetrates the midgut wall to enter the hemocoel, where it slowly develops in about 4 weeks to the cysticercoid stage (Fig. 26.62). The developmental time, however, varies significantly with different species and environmental temperatures, ranging from 2 to 7 months. Infested oribatid mites are consumed with grasses and other forage by ruminants as they graze. The cysticercoids are released as the mites are digested and attach to the wall of the alimentary tract, or bile ducts in some species, where they grow and mature to adult tapeworms in about 5–6 weeks. Mature tapeworms typically live 2–6 months before being spontaneously eliminated. During this time, they release egg-filled proglottids that are passed in the host feces.

TABLE 26.4 Anoplocephalid Tapeworms of Domestic Animals for Which Oribatid Mites Serve as Intermediate Hosts

Tapeworm Species	Domestic Hosts	Intermediate Hosts (*Oribatid* Genera)	Geographic Occurrence
Anoplocephala perfoliata	Horse, donkey	*Achipteria, Carabodes, Ceratozetes, Eremaeus, Galumna, Hermanniella, Liacarus, Liebstadia, Parachipteria, Platynothrus, Scheloribates, Trichoribates, Urubambates, Zygoribatula*	Cosmopolitan
Anoplocephala magna	Horse, donkey	*Scheloribates*	Cosmopolitan
Avitellina bangaonensis	Cattle, goats	Oribatids and/or psocids?	India
Avitellina centripunctata sensu latu (including *Avitellina goughi*, *Avitellina lahorea*, *Avitellina sudanea*)	Sheep (primarily), goat, cattle, buffalo, zebu, camel, other ruminants	*Punctoribates, Scheloribates, Trichoribates* (also psocids, collembolans)	Europe, Asia, India, Africa
Avitellina chalmersi	Sheep, goat	Oribatids and/or psocids?	India, Africa
Avitellina tatia	Goat	Oribatids and/or psocids?	India
Avitellina woodlandi	Sheep, goat	Oribatids	India
Moniezia autumnalia	Sheep, cattle	Oribatids?	Bulgaria, Tadzhikistan, Russia
Moniezia benedeni	Cattle (primarily), water buffalo, bison, sheep, goat, other ruminants	*Achipteria, Ceratoppia, Ceratozetes, Galumna, Liebstadia, Oribatula, Pergalumna, Platynothrus, Punctoribates, Scheloribates, Spatiodamaemus, Trichoribates, Zygoribatula*	Cosmopolitan
Moniezia expansa	Sheep (primarily), goat, cattle, ibex, gazelle, camel; other ruminants	*Achipteria, Allogalumna* (*Galumna?*), *Cepheus, Ceratoppia, Ceratozetes, Eremaeus, Eupelops, Euzetes, Furcoribula, Galumna, Hermanniella, Liacarus, Oribatella, Oribatula, Parachipteria, Peloptulus, Peloribates, Pergalumna, Platynothrus, Protoribates, Punctoribates, Scheloribates, Scutovertex, Spatiodamaeus, Trichoribates, Unguizetes, Xenillus, Zygoribatula*	Cosmopolitan
Moniezia denticulata	Cattle, sheep, goat, others	Oribatids	Cosmopolitan
Moniezia neumani	Sheep	*Punctoribates, Scheloribates, Trichoribates*	?
Paranoplocephala mamillana	Horse	*Achipteria, Allogalumna, Ceratozetes, Galumna, Scheloribates*	?
Stilesia globipunctata	Sheep, goat, cattle, zebu, gazelle, camel; other ruminants	*Africacarus, Allogalumna* (*Galumna?*), *Scheloribates, Zygoribatula*; (psocids?)	Europe (Spain), Asia Minor (Turkey), Asia, Africa
Stilesia hepatica	Sheep, goat, cattle (rarely); wild ruminants	Oribatids?	Asia, Africa
Stilesia vittata	camel and dromedary (primarily), sheep, goat	Oribatids?	Uzbekistan, India, Africa (eastern and southern)
Thysaniezia giardi (synonym *Thysaniezia ovilla*)	Sheep, goat (primarily); cattle, buffalo	*Achipteria, Liebstadia, Punctoribates, Scheloribates, Trichoribates, Zygoribatula*	Cosmopolitan
Thysanosoma actinioides	Sheep, goat; cattle, deer, antelope	Oribatids and/or psocids?	North America, South America (western regions)

The *Oribatid* genera listed include both those that have been found to be naturally infected and those that have been shown experimentally to support the development of Cysticercoids.

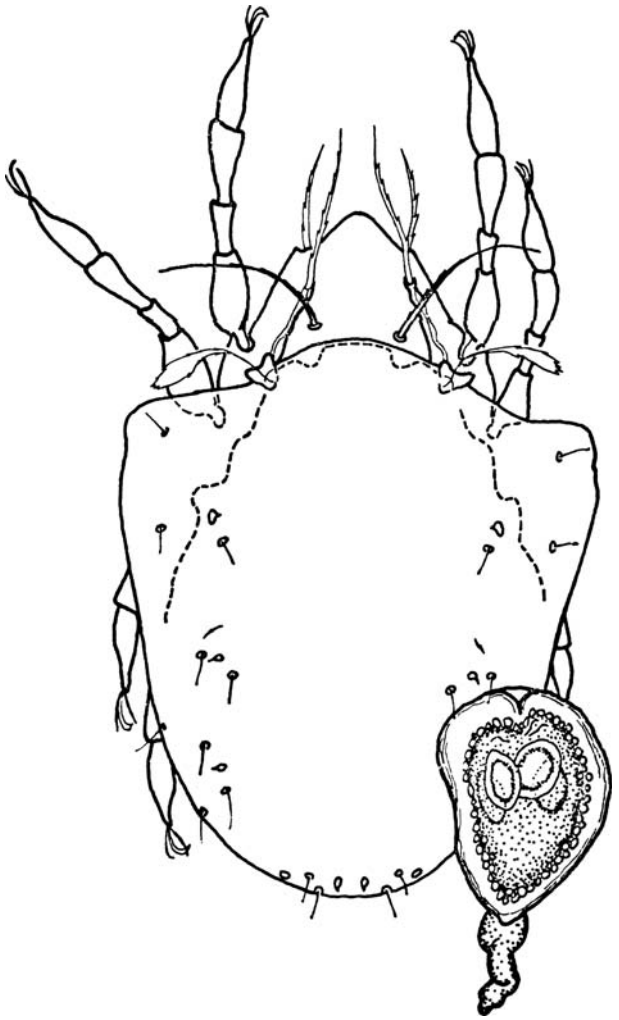


FIGURE 26.62 Oribatid mite as intermediate host for cysticercoid stage of tapeworms; cysticercoid of tapeworm is depicted at *lower right*. *From Sengbusch, 1977.*

TABLE 26.5 Families and Genera of Oribatid Mites Known to Support the Development of Anoplocephalid Tapeworms That Parasitize Domestic Animals

Achipteriidae	Galumnidae	Oribatellidae
Achipteria (2)	*Allogalumna*	*Oribatella* (1)
Parachipteria (2)	*Galumna* (15)	Oribatulidae
Astegistidae	*Pergalumna* (2)	*Liebstadia* (1)
Furcoribula (1)	*Pilogalumna* (1)	*Oribatula* (2)
Camisiidae	Haplozetidae	*Zygoribatula* (11)
Platynothrus (1)	*Peloribates* (2)	Pelopidae
Carabodidae	Hermanniellidae	*Peloptulus*
Carabodes (1)	*Hermanniella* (2)	Phenopelopidae
Cepheus (1)	Liacaridae	*Eupelops* (1)
Ceratozetidae	*Adoristes* (2)	Protoribatidae
Ceratozetes (4)	*Liacarus* (1)	*Protoribates* (1)
Hypozetes (1)	*Xenillus* (1)	Scheloribatidae
Trichoribates (3)	Metrioppidae	*Scheloribates* (14)
Damaeidae	*Ceratoppia* (1)	*Urubambates* (1)
Spatiodamaeus (1)	Mochlozetidae	Scutoverticidae
Epilohmanniidae	*Unguizetes* (1)	*Scutovertex* (1)
Epilohamminia (1)	Mycobatidae	Xylobatidae
Eremaeidae	*Punctoribates* (3)	*Xylobates* (1)
Eremaeus (2)	Oppiidae	Family?
Euzetidae	*Oppiella* (1)	*Africacarus* (1)
Euzetes (1)		

The approximate number of species is indicated in parentheses after each genus.
Based on Allred (1954), Sengbusch (1977), Narsapur (1988), and Denegri (1993)

More than 125 species of oribatid mites, representing 37 genera and 25 families, have been shown to support development of anoplocephalid tapeworms (Denegri, 1993) (Table 26.5). Of this number, only about 45 oribatid species have been found to be naturally infected with cysticercoids. This indicates that many oribatid species are capable of supporting development of cysticercoids when experimentally infected, but do not necessarily ingest tapeworm eggs under natural conditions. The most important mite families that serve as intermediate hosts are Ceratozetidae, Galumnidae, Oribatulidae, and Scheloribatidae, which apparently reflects their relatively large size and ability to ingest tapeworm eggs. Other contributing factors are the size and structure of the chelicerae, the natural diet of the mites, and their tendency to move upward from the soil onto grasses where ruminants are grazing. This combination of traits explains why *Galumna*, *Scheloribates*, and *Zygoribatula* species are the oribatids most commonly infested with cysticercoids of anoplocephalid tapeworms. For further information on oribatid taxa as intermediate hosts for tapeworms, see Sengbusch (1977), Narsapur (1988), and Denegri (1993).

Heavy tapeworm infestations in ruminants are directly correlated to large oribatid mite populations. Newly seeded and first-year pastures have low mite numbers and result in low infestation rates even in young animals. Older pastures that have been left undisturbed for 2 or more years support the buildup of oribatid numbers, significantly increasing the risk of parasitism. To reduce mite populations and avoid this problem, lambs and calves should be started on new pastures, permanent pastures should be cultivated annually, and pastures should be fenced off to prevent livestock from grazing in adjacent rough pasture and woodland.

A few anoplocephalid tapeworms in the genus *Cittotaenia* that use oribatid mites as intermediate hosts are parasites of rabbits and hares in North America, Europe, and Asia. Heavy infestations by those tapeworms can cause digestive disturbances, emaciation, and death of hosts. In

addition, *C. pusilla* occasionally parasitizes laboratory mice and rats in North America, Europe, and Japan. Its natural hosts include the Norway rat, black rat, house mouse, voles (*Microtus* spp.), and other wild rodents. The grain-infesting mite *Glycyphagus domesticus* (Family Glycyphagidae) reportedly serves as the intermediate host (Allred, 1954).

REFERENCES AND FURTHER READING

Al-Arfaj, A., Mullen, G. R., Rashad, R., Abdel-Hameed, A., OConnor, B. M., Alkhalife, I., et al. (2007). A human case of otoacariasis involving a histiostomatid mite (Acari: Histiostomatidae). *The American Journal of Tropical Medicine and Hygiene, 76*, 967−971.

Allred, D. M. (1954). Mites as intermediate hosts of tapeworms. *Proceedings of the Utah Academy of Sciences, 31*, 44−51.

Andrews, J. R., & Ramsay, G. W. (1982). A case of papular dermatosis in man attributed to an ascid mite (Acari). *Journal of Medical Entomology, 19*, 111−112.

Arlian, L. G. (2002). Arthropod allergens and human health. *Annual Review of Entomology, 47*, 395−433.

Arlian, L. G., & Hart, B. J. (1992). Water balance and humidity requirements of house dust mites. *Experimental and Applied Acarology, 16*, 15−35.

Arlian, L. G., Morgan, M. S., & Neal, J. S. (2002). Dust mite allergens: Ecology and distribution. *Current Allergy and Asthma Reports, 2*, 401−411. BioMed Central Ltd., London.

Arlian, L. G., Morgan, M. S., Vyszenski-Moher, D. L., & Stemmer, B. L. (1994a). *Sarcoptes scabiei:* The circulating antibody response and induced immunity to scabies. *Experimental Parasitology, 78*, 37−50.

Arlian, L. G., Rapp, C. M., Vyszenski-Moher, D. L., & Morgan, M. S. (1994b). *Sarcoptes scabiei:* Histopathological changes associated with acquisition and expression of host immunity to scabies. *Experimental Parasitology, 78*, 51−63.

Aylesworth, R., & Baldridge, D. (1983). *Dermatophagoides schereemetewski* and feather pillow dermatitis. *Minnesota Medicine, 66*, 43.

Ba, C. T., Wang, X. Q., Renaud, R., Euzet, L., Marchand, B., & de Meeus, T. (1994). Diversity in the genera *Avitellina* and *Thysaniezia* (Cestoda: Cyclophyllidea): Genetic evidence. *Journal of the Helminthological Society of Washington, 61*, 57−60.

Baker, R. A. (1973). Notes on the internal anatomy, the food requirements and development in the family Ereynetidae (Trombidiformes). *Acarologia, 15*, 43−52.

Baker, E. W., Evans, T. M., Gould, D. J., Hull, W. B., & Keegan, H. L. (1956). *A manual of parasitic mites*. New York: National Pest Contol Assoc., 170 pp.

Baker, A. S. (1999). *Mites and ticks of domestic animals. An identification guide and information source*. London: H.M. Stationery Office, 240 pp.

Bates, P. G. (1999). Inter- and intra-specific variation within the genus *Psoroptes* (Acari: Psoroptidae). *Veterinary Parasitology, 83*, 201−217.

Beaulieu, F., Dowling, A. P. G., Klompen, H., Moraes, G. J., & de, Walter, D. E. (2011). Superorder Parasitiformes Reuter, 1909. In Z.-Q. Zhang (Ed.), *Zootaxa: Vol. 3148. Animal biodiversity: An outline of higher-level classification and survey of taxonomic richness* (pp. 123−128).

Betz, T. G., Davis, B. L., Fournier, P. V., Rawlings, J. A., Elliot, L. B., & Baggett, D. A. (1982). Occupational dermatitis associated with straw itch mites (*Pyemotes ventricosus*). *The Journal of the American Medical Association, 247*, 2821−2823.

Bochkov, A. V. (2010). A review of mammal-associated Psoroptidia (Acariformes: Astigmata). *Acarina, the Russian Journal of Acarology, 18*, 99−260.

Bochkov, A. V., Klimov, P. B., Hestvik, G., & Saveljev, A. P. (2014). Integrated Bayesian species delimitation and morphological diagnostics of chorioptic mange mites (Acariformes: Psoroptidae: *Chorioptes*). *Parasitology Research, 113*, 2603−2627.

Bochkov, A. V., & Saveljev, A. P. (2014). Mites of the genus *Schizocarpus* Trouessart, 1896 (Acariformes: Chirodiscidae) from the North American beavers (*Castor canadensis*) in Russia. *Parazitologiya (St. Petersburg), 48*, 430−436.

Bochkov, A. V., Zabludovskaya, S., & OConnor, B. M. (2008). In , *Zootaxa: Vol. 1951. Phylogeny and systematics of the endoparasitic astigmatid mites (Acari: Sarcoptiformes) of mammals: Families Gastronyssidae, Lemurnyssidae, and Pneumocoptidae* (pp. 1−152).

Borstein, S., & Zakrisson, G. (1993). Clinical picture and antibody response in pigs infected by *Sarcoptes scabiei* var. *suis*. *Veterinary Dermatology, 4*, 123−131.

Boyce, W. M., & Brown, R. N. (1991). Antigenic characterization of *Psoroptes* spp. (Acari: Psoroptidae) mites from different hosts. *The Journal of Parasitology, 77*, 675−679.

Brettman, L. R., Lewin, S., Holzman, R. S., Goldman, W. D., Marr, J. S., Kechijian, P., et al. (1981). Rickettsialpox: Report of an outbreak and a contemporary review. *Medicine (Baltimore), 60*, 363−372.

Broce, A. B., Zurek, L., Kalisch, J. A., Brown, R., Keith, D. L., Gordon, D., et al. (2006). *Pyemotes herfsi* (Acari: Pyemotidae), a mite new to North America as the cause of bite outbreaks. *Journal of Medical Entomology, 43*, 610−613.

Brockie, R. E. (1974). The hedgehog mange mite, *Caparinia tripilis*, in New Zealand. *New Zealand Veterinary Journal, 2*, 243−247.

Bukva, V. (1986). *Demodex tauri* sp.n. (Acari: Demodicidae), a new parasite of cattle. *Folia Parasitology, 33*, 363−369.

Bukva, V. (1990). Three species of the hair follicle mites (Acari: Demodicidae) parasitizing sheep, *Ovis aries* L. *Folia Parasitology, 37*, 81−91.

Burns, D. A. (1992). Follicle mites and their role in disease. *Clinical Experimental Dermatology, 17*, 152−155.

Camin, J. H. (1953). Observations on the life history and sensory behavior of the snake mite, *Ophionyssus natricis* (Gervais) (Acarina: Macronyssidae). *Chicago Academy of Sciences Special Publications, 10*, 75.

Carlson, B. L., Roher, D. P., & Nielsen, S. W. (1982). Notoedric mange in gray squirrels (*Sciurus carolinensis*). *Journal of Wildlife Diseases, 18*, 347−348.

Castellani, A. (1907). Note on an acarid-like parasite found in the omentum of a Negro. *Centralblatt für Bakteriologie, Parasitenkunde und Infektionskrankheiten I, 43*, 372 (Cytoditidae).

Chakrabarti, A. (1986). Human notoedric scabies from contact with cats infested with *Notoedres cati*. *International Journal of Dermatology, 25*, 646−648.

Chen, X. B., Fu, C. B., Chen, X. B., & Fu, C. B. (1992). Mites causing pulmonary, intestinal and urinary acariasis. In X. B. Chen, & E. P. Ma (Eds.), *Researches of acarology in China* (pp. 109−113). Chongqing, China: Chongqing Publishing House.

Chen, X. B., Sun, X., & Hu, S. F. (1990). Clinical manifestation and treatment of pulmonary acariasis. *Chinese Journal of Parasitology and Parasitic Diseases, 8*, 41–44 (in Chinese).

Clark, G. M. (1960). Three new nasal mites (Acarina: Speleognathidae) from the grey squirrel, the common grackle, and the meadowlark in the United States. *Proceedings of the Helminthological Society of Washington, 27*, 103–110.

Cochrane, G. (1994). Effects of *Psoroptes ovis* on lamb carcasses. *Veterinary Record, 134*, 72.

Cole, N. A., & Guillot, F. S. (1987). Influence of *Psoroptes ovis* on the energy metabolism of heifer calves. *Veterinary Parasitology, 23*, 285–295.

Colloff, M. J. (2009). *Dust mites*. Clayton, Australia: CSIRO Publishing, 583 pp.

Colloff, M. J., ChannaBasavanna, G. P., & Viraktamath, C. A. (1989). Human semen as a dietary supplement for house dust mites (Astigmata: Pyroglyphidae). *Progress in Acarology, 1*, 141–146.

Colloff, M. J., & Hart, B. J. (1992). Age structure and dynamics of house dust mite populations. *Experimental and Applied Acarology, 16*, 49–74.

Cosoroabă, I. (1994). *Acarologie Veterinaria*. Bucharest: Ceres Publishing House, 252 pp. (in Romanian).

Costa, A. L., Leite, R. C., Faccini, J. L. H., & DaCosta, A. L. (1992). Preliminary investigations on transmission and life cycle of the ear mites of the genus *Raillietia* Trouessart (Acari: Gamasida) parasites of cattle. *Memorias Do Instituto Oswaldo Cruz, 87*(Suppl. I), 97–100.

Cremers, H. J. W. M. (1985). The incidence of *Chorioptes bovis* (Acarina: Psoroptidae) on the feet of horses, sheep, and goats in The Netherlands. *Veterinry Quarterly, 7*, 283–289.

Cuthbert, O. D. (1990). Storage mite allergy. *Clinical Reviews in Allergy & Immunology, 8*, 69–86.

Czarnecki, N., & Kraus, H. (1978). Milbendermatitis durch *Tyrophagus dimidiatus*. *Zeitschrift Fur Hautkrankheiten, 53*, 414–416.

Dahl, F. (1910). Milben als Erzeuger von Zellwucherunge. *Centralblatt für Bakteriologie, Parasitenkunde und Infektionskrankheiten I, 53*, 524–533.

DaMassa, A. J., Wakenall, P. S., & Brooks, D. L. (1992). Mycoplasmas of goats and sheep. *Journal of Veterinary Diagnostic Investigation, 4*, 101–113.

Davis, J. W., & Anderson, R. C. (1971). *Parasitic diseases of wild mammals*. Ames: Iowa State Univ. Press, 364 pp.

Denegri, G. M. (1985). Desarrollo experimental de *Bertiella mucronata* Meyner, 1895. (Cestoda: Anoplocephalidae) de origen humano en su huésped intermediario. *Zentralblatt für Veterinärmedizin Reihe B, 32*, 498–504.

Denegri, G. M. (1993). Review of oribatid mites as intermediate hosts of tapeworms of the Anoplocephalidae. *Experimental and Applied Acarology, 17*, 567–580.

Desch, C., & Nutting, W. B. (1972). *Demodex folliculorum* (Simon) and *D. brevis* Akbulatova of man: Redescription and reevaluation. *The Journal of Parasitology, 58*, 169–177.

Dietrich, M., & Horstmann, R. D. (1983). Urtikarielle Dermatitis bei Acariasis durch *Dermanyssus hirundinis*. Schwalben-Trugkratze, Pseudoscabies. *Medizinische Welt, 34*, 595–597.

Domrow, R. (1969). The nasal mites of Queensland birds (Acari: Dermanyssidae, Ereynetidae and Epidermoptidae). *Proceedings of the Linnean Society of New South Wales, 93*, 297–426.

Domrow, R. (1988). Acari Mesostigmata parasitic on Australian vertebrates: An annotated checklist, keys and bibliography. *Invertebrate Taxonomy, 1*, 817–948.

Domrow, R. (1991). Acari Prostigmata (excluding Trombiculidae) parasitic on Australian vertebrates: An annotated checklist, keys and bibliography. *Invertebrate Taxonomy, 4*, 1283–1376.

Domrow, R. (1992). Acari Astigmata (excluding feather mites) parasitic on Australian vertebrates: An annotated checklist, keys and bibliography. *Invertebrate Taxonomy, 6*, 1459–1606.

Elixmann, J. H., Jorde, W., & Schata, M. (1991). Incidence of mites in domestic textiles in Germany. *Allergologie, 14*, 451–460.

English, F. P., & Nutting, W. B. (1981). Demodicosis of ophthalmic concern. *American Journal of Ophthalmology, 91*, 362–372.

Evans, G. O. (1992). *Principles of acarology*. C.A.B. International, 563 pp.

Fain, A. (1956). Une nouvelle famille d'acariens endoparasites des chauves-souris: Gastronyssidae fam. nov. *Annales de la Société Belge de Médecine Tropicale, 36*, 87–98.

Fain, A. (1957). Les Acariens des familles Epidermoptidae et Rhinonyssidae parasites des fosses nasales doiseaux au Ruanda-Urundi et au Congo Belge. *Annales du Musee Royal du Congo Belge, 8*(60), 1–176.

Fain, A. (1961). Les acariens parasites endopulmonaires des serpents (Entonyssidae: Mesostigmata). *Bulletin de l'Institut royal des sciences naturelles de Belgique, 37*, 1–135.

Fain, A. (1964a). Chaetotaxie et classification des Gastronyssidae avec description dun nouveau genre parasite nasicole d'un Ecureuil sudafricain (Acarina: Sarcoptiformes). *Revue de Zoologie et de Botanique Africaines, 70*, 40–52.

Fain, A. (1964b). Les Lemurnyssidae parasites nasicoles des Lorisidae africains et des Cebidae sud-américaines. Description dune espèce nouvelle (Acarina: Sarcoptiformes). *Annales de la Societe belge de medecine tropicale, 44*, 453–458.

Fain, A. (1965). A review of the family Epidermoptidae Trouessart parasitic on the skin of birds. Parts I-II. In Kon Acad. Wetensch., Lett. Schone Kunsten Belg. *(Wetensch.), 27(84)* pp. 1–176), 5–144.

Fain, A. (1967). Observations sur les Rodhainyssinae acariens parasites des voies respiratoires des chauves-souris (Gastronyssidae: Sarcoptiformes). *Acta zoologica et pathologica Antverpiensia, 44*, 3–35.

Fain, A. (1968). Étude de la variabilite de *Sarcoptes scabiei* avec une revision des Sarcoptidae. *Acta zoologica et pathologica Antverpiensia, 47*, 3.

Fain, A. (1970a). Sur une nouvelle espèce du genre *Psoroptes* produisant la gale chez le buffle africain (Acarina: Sarcoptiformes). *Revue de Zoologie et de Botanique Africaines, 81*, 95–100.

Fain, A. (1970b). Novoeaux acarines nasicoles de le famillie Turbinoptidae (Sarcoptiformes). *Bulletin et Annales de la Société Royale d'Entomologie de Belgique, 106*, 28–36.

Fain, A., & Hyland, K. E. (1975). Speleognathinae collected from birds in North America (Acarina: Ereynetidae). *Journal of the New York Entomological Society, 83*, 203–208.

Farley, M. L., Mabry, L. C., & Hieger, L. R. (1989). Mites in pulmonary cytology specimens. *Diagnostic Cytopathology, 5*, 416–426.

Fox, J. G., & Hewes, K. (1976). *Cheyletiella* infestation in cats. *Journal of the American Veterinary Medical Association, 169*, 332–333.

Franklin, C. D., & Underwood, J. C. (1986). *Demodex* infestation of oral mucosal sebaceous glands. *Oral Surgery, Oral Medicine, Oral Pathology, 61*, 80–82.

Flynn, R. J. (1973). *Parasites of laboratory animals*. Ames: Iowa State Univ. Press, 884 pp.

Furman, D. P. (1959). Feeding habits of symbiotic mesostigmatid mites of mammals in relation to pathogen-vector potentials. *The American Journal of Tropical Medicine and Hygiene, 8*, 5.

Furman, D. P., & Dailey, M. D. (1980). The genus *Halarachne* (Acari: Halarachnidae), with the description of a new species from the Hawaiian monk seal. *Journal of Medical Entomology, 17*, 352–359.

Garlick, N. L. (1977). Canine pulmonary acariasis. *Canine Practice, 4*, 42–47.

Gaud, J., & Atyeo, W. T. (1996). Feather mites of the world (Acarina, Astigmata): The supraspecific taxa. Part I. Text. Part II. Illustrations of feather mite taxa. *Annalen Zoologische Wetenschappen-Koninklijk Museum voor Midden-Africa/Musee Royal de l=Afrique Centrale, Tervuren, Belgium*, 277 (1) 1193 pp. 277 (2) 2436 pp.

Georgi, J. R., & Whitlock, J. H. (1980). Arachnids. Chapter 3. In J. R. Georgi (Ed.), *Parasitology for veterinarians* (3rd ed., pp. 41–67). W.B. Saunders Co., 460 pp.

Gerson, U., Fain, A., & Smiley, R. L. (1999). Further observations on the Cheyletidae (Acari), with a key to the genera of the Cheyletinae and a list of all known species in the family. *Bulletin de L'Institut Royal des Sciences Naturelles de Belgique Entomologie, 69*, 35–86.

Graber, M. (1959). *I.A.C.E.D. Symposium on helminthiasis in domestic animals* (pp. 81–130). Nairobi, Kenya: C.C.T.A.

Gregory, M. W. (1981). Mites of the hedgehog *Erinaceus albiventris* Wagner in Kenya: Observations on the prevalence and pathogenicity of *Notoedres oudemansi* Fain, *Caparinia erinacei* Fain and *Rodentopus sciuri* Fain. *Parasitology, 82*, 149–157.

Guillot, F. S., & Stromberg, P. C. (1987). Reproductive success of *Psoroptes ovis* (Acari: Psoroptidae) on Hereford calves with a previous infestation of psoroptic mites. *Journal of Medical Entomology, 24*, 416–419.

Harada, S. H., & Sadaji, T. (1925). On a case of mites found in human urine. *Chugai Iji Shimpo, 44*, 859–866 (in *Japanese*).

Hardison, J. L. (1977). A case of *Eutrombicula alfreddugesi* (chiggers) in a cat. *Veterinary Medicine, Small Animal Clinician, 72*, 47.

Hartley, M., & English, A. (2005). *Sarcoptes scabiei* var. wombati infection in the common wombat (*Vombatus ursinus*). *European Journal of Wildlife Research, 51*, 117–121.

Harwood, R. F., & James, M. T. (1979). *Entomology in human and animal health* (7th ed., p. 358). New York: Macmillan Co..

Hay, D. B., Hart, B. J., & Douglas, A. E. (1993). Effects of the fungus *Aspergillus penicillioides* on the house dust mite *Dermatophagoides pteronyssinus*: An experimental re-evaluation. *Medical and Veterinary Entomology, 7*, 271–274.

Hay, D. B., Hart, B. J., Pearce, R. B., Kozakiewicz, Z., & Douglas, A. E. (1992). How relevant are house dust mite-fungal interactions in laboratory culture to the natural dust system? *Experimental and Applied Acarology, 16*, 37–47.

Heilesen, B. (1946). *Studies on Acarus scabiei and scabies*. Copenhagen: Rosenkilde & Bagger, 370 pp.

Henschel, J. (1929). Reizphysiologische Untersuchung der Käsemilbe *Tyrolichus casei* (Oudemans). *Zeitschrift für vergleichende Physiologie, 9*, 802–837.

Herrin, C. S. (1974). The taxonomic status of *Hirstionyssus butantanensis* (Fonseca, 1932) (Acari: Mesostigmata). *Journal of Medical Entomology, 11*, 341–346.

Hewitt, M., Barrow, G. I., Miller, D. C., Turk, F., & Turk, S. (1973). Mites in the personal environment and their role in skin disorders. *British Journal of Dermatology, 89*, 401–409.

Higby, W. E. (1946). A new canary plague. *All Pets Magazine December*, 8–9 (Cytoditidae).

Hirst, S. (1922). Mites injurious to domestic animals. In *British museum of natural history, econ. ser. no. 13*, 107 pp.

Hoban, P. A. (1990). A review of desert bighorn sheep in the San Andres Mountains, New Mexico. *Desert Bighorn Council Transact., 34*, 14–22.

Hogsette, J. A., Butler, J. F., Miller, W. V., & Hall, R. D. (1988). Annotated bibliography of the northern fowl mite, *Ornithonyssus sylviarum* (Canestrini & Fanzago), (Acari: Macronyssidae). *Misc. Publ. Entomol. Soc. Am., 76*, 104.

Hong, C. S., Park, H. S., & Oh, S. H. (1987). *Dermatophagoides farinae,* an important allergenic substance in buckwheat-husk pillows. *Yonsei Medical Journal, 28*, 274–281.

Houck, M. A. (1994). *Mites: Ecological and evolutionary analyses of life-history patterns*. London: Chapman & Hall, 357 pp.

Huebner, R. J., Jellison, W. L., & Pomerantz, C. (1946). Rickettsialpox–a newly recognized rickettsial disease. IV. Isolation of a rickettsia apparently identical with the causative agent of rickettsialpox from *Allodermanyssus sanguineus*, a rodent mite. *Public Health Reports, 61*, 1677–1682.

Hughes, A. M. (1976). The mites of stored food and houses. In *Ministry of agriculture, fisheries and food, tech. bull. 9*. London: Her Majesty's Stationery Office, 400 pp.

Hull, W. B. (1970). Respiratory mite parasites in nonhuman primates. *Laboratory Animal Care, 20*, 402–406.

Izdebska, J. N. (2010). *Demodex* spp. (Acari, Demodicidae) and demodecosis in dogs: Characteristics, symptoms, occurrence. *Bulletin of the Veterinary Institute in Pulawy, 54*, 335–338.

Johnson, L. S., & Albrecht, D. J. (1993). Effects of haematophagous ectoparasites on nestling house wrens, *Troglodytes aedon*: Who pays the cost of parasitism? *Oikos, 66*, 255–262.

Kambarage, D. M. (1992). Sarcoptic mange infestation in goats. *Bulletin of Animal Health and Production in Africa, 40*, 239–244.

Kass, E. M., Szaniawski, W. K., Levy, H., Leach, J., Srinivasan, K., & Rive, C. (1994). Rickettsialpox in a New York city hospital, 1980–1989. *New England Journal of Medicine, 331*, 1612–1617.

Kaufmann, J. (1996). *Parasitic infections of domestic animals: A diagnostic manual*. Basel: Birkhaser Verlag, 423 pp.

Kawamura, A., Jr., Tanaka, H., & Tamura, A. (1995). *Tsutsugamushi disease*. Tokyo: University of Tokyo Press, 362 pp.

Kazacos, E. A., Kazacos, K. R., & Demaree, H. A., Jr. (1983). Notoedric mange in two fox squirrels. *Journal of the American Veterinary Medical Association, 183*, 1281–1282.

Keh, B. (1974). Dermatitis caused by the bat mite *Chiroptonyssus robustipes* (Ewing) in California. *Journal of Medical Entomology, 11*, 498.

Kethley, J. (1982). Acariformes. In S. P. Parker (Ed.), *Synopsis and classification of living organisms* (Vol. 2, p. 117). New York: McGraw-Hill.

Kilpio, O., & Pirila, V. (1952). A new tyroglyphid mite causing dermatitis. *Acta Dermato-Venereologica, 32*, 197–200.

Kim, K. C., Haas, V. L., & Keyes, M. C. (1980). Populations, microhabitat preference and effects of infestations of two species of *Orthohalarachne* (Halarachnidae: Acarina) in the northern fur seal. *Journal of Wildlife Diseases, 16*, 45–51.

Kirk, H. (1949). Demodectic mange. *Veterinary Record, 61*, 394.

Kirkwood, A. C. (1980). Effect of *Psoroptes ovis* on the weight of sheep. *Veterinary Record, 107*, 469–470.

Klompen, J. S. H. (1989). Ontogeny of *Rhyncoptes grabberi,* n. sp. (Acari: Astigmata: Rhyncoptidae) associated with *Macaca mulatta*. *Journal of Medical Entomology, 26*, 81–87.

Klompen, J. S. H. (1992). Phylogenetic relationships in the mite family Sarcoptidae (Acari: Astigmata). *Miscellaneous Publications, Museum of Zoology, University of Michigan, 180*, 1–154.

Koutz, F. R., Chamberlain, D. M., & Cole, C. R. (1954). *Pneumonyssus caninum* in the nasal cavity and paranasal sinuses. *Journal of the American Veterinary Medical Association, 122*, 106.

Krantz, G. W., & Walter, D. E. (Eds.). (2009). *A manual of acarology*. Lubbock: Texas Tech University Press.

Krinsky, W. L. (1983). Does epizootic lymphocytic choriomeningitis prime the pump for epidemic rickettsialpox? *Reviews of Infectious Diseases, 5*, 1118–1119.

Kumar, D., Raisinghani, P. M., & Manohar, G. S. (1992). Sarcoptic mange in camels: A review. In W. R. Allen, A. J. Higgins, I. G. Mayhew, D. H. Snow, & J. F. Wade (Eds.), *Proceedings of the first international camel conference, Dubai* (pp. 79–82). Newmarket, UK: R.W. Publications Ltd.

Kummel, B. A., Estes, S. A., & Arlian, L. G. (1980). *Trixacarus caviae* infestation of Guinea pigs. *Journal of the American Veterinary Medical Association, 177*, 903–908.

Lackman, D. B. (1963). A review of information on rickettsialpox in the United States. *Clinical Pediatrics, 2*, 296–301.

Lange, R. E., Sandoval, A. V., & Meleney, W. P. (1980). Psoroptic scabies in bighorn sheep (*Ovis canadensis mexicana*) in New Mexico. *Journal of Wildlife Diseases, 16*, 77–82.

Lawrence, R. F. (1952). A new parasitic mite from the nasal cavities of the South American toad *Bufo regularis* Reuss. *Proceedings of the Zoological Society of London, 121*, 747–752.

Lee, K. J., Lang, C. M., Hughes, H. C., & Hartshorn, R. D. (1981). Psorergatic mange (Acari: Psorergatidae) of the stumptail macaque (*Macaca arctoides*). *Laboratory Animal Science, 31*, 77–79.

Lefer, L. G., & Rosier, R. P. (1988). Presence of a mite in the female genital tract: Some comments. *International Journal of Acarology, 14*, 91–92 (Pyroglyphidae, Dermatophagoides).

Fichoux, Le, Rack, Y. G., Motte, P., Dellamonica, P., & Marty, P. (1980). Dermatite prurigineuse due a *Pyemotes zwoelferi* Krczal, 1963, a propos de plusieurs cas dans les alpes-maritimes. *Acta Tropica, 37*, 83–89.

Li, C., & Li, L. (1990). Human pulmonary acariasis in Anhui Province: An epidemiological survey. *Chinese the Journal of Parasitology & Parasitic Diseases, 8*, 41–44 (in Chinese).

Lowenstine, L. J., Carpenter, J. L., & OConnor, B. M. (1979). Trombiculosis in a cat. *Journal of the American Veterinary Medical Association, 175*, 289–292.

Ma, E. P., & Wang, R. S. (1992). Tarsonemid mites. In X. B. Chen, & E. P. Ma (Eds.), *Researches of acarology in China* (pp. 34–37). Chongqing: Chongqing Publishing House.

Malhotra, S. K., & Capoor, V. N. (1982). A new species of *Avitellina* Gough (1911) from Garhwal Hills with a revised key to species of subgenus *Avitellina* Raina (1975). *Proceedings of the Indian Academy of Parasitology, 3*, 12–16.

Martinez Maranon, R., & Hoffman, A. (1976). Tres casos de infestacion del intestino humano por acaros en el sur de Veracruz. *Revista de Investigacion en Salud Publica, 36*, 187–201.

Matthes, H. F. (1994). Investigations of pathogenesis of cattle demodicosis: Sites of predilection, habitat, and dynamics of demodectic nodules. *Veterinary Parasitology, 53*, 283–291.

Mazet, J. A. K., Boyce, W. M., Mellies, J., Gardner, I. A., Clark, R. K., & Jessup, D. A. (1992). Exposure to *Psoroptes* sp. mites is common among bighorn sheep (*Ovis canadensis*) populations in California. *Journal of Wildlife Diseases, 28*, 542–547.

McGarry, J. W. (1993). Identification of *Cheyletiella* eggs in dog feces. *Veterinary Record, 132*, 359–360.

Mellanby, K. (1972). *Scabies* (2nd ed.). Hampton, UK: E.W. Classey.

Meng, Y. C., Zhuge, H. X., Lan, M. Y., & Zhon, H. F. (1991). Experimental study on transmission of hemorraghic fever with renal syndrome virus by mites, *Ornithonyssus bacoti* (Hirst). In *Proc. VIII International congress of acarology, Ceske Budejovice, Czechoslovakia, 1990* (Vol. II, pp. 35–39).

Miller, W. H. (1984). Diseases of domestic animals. In W. B. Nutting (Ed.), *Vol. 2. Mammalian diseases and arachnids* (pp. 115–126). Boca Raton, FL: CRC Press.

Mills, O. H., Jr., & Kligman, A. M. (1983). The follicular biopsy. *Dermatologica, 167*, 57–63.

Mironov, S. V., Bochkov, A. V., & Fain, A. (2005). Phylogeny and evolution of parasitism in feather mites of the families Epidermoptidae and Dermationidae (Acari: Analgoidea). *Zoologischer Anezeiger, 243*, 155–179.

Möller, A. P. (1990). Effects of parasitism by a haematophagous mite on reproduction in the barn swallow. *Ecology, 71*, 2345–2357 *(Ornithonyssus bursa)*.

Morgan, K. L. (1992). Parasitic otitis in sheep associated with *Psoroptes* infestation: A clinical and epidemiological study. *Veterinary Record, 130*, 530–532.

Mulla, M., & Medina, M. S. (1980). *Domestic Acari of Colombia: Bionomics, ecology, and distribution of allergenic mites, their role in allergenic diseases* (bilingual, English/Spanish). Bogota: Colciencias, 270 pp..

Nadchatram, M. (1970). A review of intranasal chiggers with descriptions of twelve species from east New Guinea (Acarina: Trombiculidae). *Journal of Medical Entomology, 7*, 1–29.

Naltsas, S., Hodge, S. J., Gataky, G. J., Jr., & Owen, L. G. (1980). Eczematous dermatitis caused by *Dermanyssus americanus*. *Cutis, 25*, 429–431.

Narsapur, V. S. (1988). Pathogenesis and biology of anoplocephaline cestodes of domestic animals. *Annales de Recherches Vétérinaires, 19*, 1–17.

Nutting, W. B. (1976a). Hair follicle mites (Acari: Demodicidae) of man. *International Journal of Dermatology, 15*, 79–98.

Nutting, W. B. (1976b). Hair follicle mites (*Demodex* spp.) of medical and veterinary concern. *Cornell Veterinarian, 66*, 214–231.

Nutting, W. B. (1976c). Pathogenesis associated with hair follicle mites (Acari: Demodicidae). *Acarologia, 17*, 493–506.

Nutting, W. B. (1984). *Mammalian diseases and Arachnids*. Vol. I. Pathogen Biology and Clinical Management, 277 pp. Vol. II. Medico-Veterinary Laboratory, and Wildlife Diseases, and Control, 280 pp. Boca Raton, Florida: CRC Press, Inc.

Nutting, W. B., Firda, K. E., & Desch, C. E., Jr. (1989). Topology and histopathology of hair follicle mites (Demodicidae) of man. In G. P. ChannaBassavana, & C. A. Viraktamath (Eds.), *Progress in acarology* (Vol. 1, pp. 113–121). New Delhi: Oxford & IBH Publish. Co.

Oberem, P. T., & Malan, F. S. (1984). A new cause of cattle mange in South Africa: *Psorergates bos* Johnston. *Journal of the South African Veterinary Association, 55*, 121−122.

O'Brien, D. J., Gray, J. S., & OReilly, P. F. (1994). Survival and retention of infectivity of the mite *Psoroptes ovis* off the host. *Veterinary Research Communications, 18*, 27−36.

OConnor, B. M. (1984). Phylogenetic relationships among higher taxa in the Acariformes, with particular reference to the Astigmata. In D. A. Griffiths, & C. E. Brown (Eds.), *Vol. 1. Acarology VI* (pp. 19−27). Chichester: Ellis Horwood.

OConnor, B. M., & Klimov, P. B. (2015). Review and resolution of some nomenclatural issues regarding the genus *Psoroptes* (Acari: Psoroptidae), scab-mites of domestic and wild mammals. *Experimental and Applied Acarology, 66*, 337−345.

Oehlschlaegel, G., Bayer, F., Disko, R., Fechter, H., & Mahunka, S. (1983). *Tarsonemus hominis* in Hautbindegewebe. *Hautarzt, 34*, 632−634.

Onderscheka, K., Kutzer, E., & Richter, H. E. (1968). Die Raeude der Gemse und ihre Bekampfung. II. Zusammenhaenge zischen Ernaehrung und Raeude. *Zeitschrift für Jagdwissenschaft, 14*, 12.

Orkin, M., Maibach, H. I., Parish, L. C., & Schwartzman, R. M. (Eds.). (1977). *Scabies and pediculosis*. Baltimore: J.B. Lippincott, 203 pp.

Otto, Q. T., & Jordaan, L. C. (1992). An orf-like condition caused by trombiculid mites on sheep in South Africa. *The Onderstepoort Journal of Veterinary Research, 59*, 335−336.

Ottoboni, F., di Loreto, V., Cantoni, A., Lozzia, G. C., Rota, P., Melej, R., et al. (1989). [Investigatioins into allergic diseases among raw ham workers in Langhirano and San Daniele.] La difesa antiparassitaria nelle industrie alimentari e la protezion degli alimenti. *Atti del 4o simposio*, 235−241.

Parodi, A. S., Rugiero, H. R., Greenway, D. J., Mettler, Martinez, N. A., Boxaca, M., & De la Barerra, J. M. (1959). Aislamiento del virus Junin (F.H.E.) De los acaros de la zona epidemica (*Echinolaelaps echidnius*, Berlese). *La Prensa Médica Argentina, 46*, 2242−2244.

Pass, D. A., & Sue, L. J. (1983). A trombiculid mite infestation of canaries. *Austral Journal of Veterinary Sciences, 60*, 218−219.

Pegler, K. R., Evans, L., Stevens, J. R., & Wall, R. (2005). Morphological and molecular comparison of host derived populations of parasitic *Psoroptes* mites. *Medical and Veterinary Entomology, 19*, 392−403.

Pence, D. B. (1973). .The nasal mites of birds from Louisiana. IX. Synopsis. *The Journal of Parasitology, 59*, 881−892.

Pence, D. B. (1975). Keys, species and host list, and bibliography for nasal mites of North American birds (Acarina: Rhinohyssinae, Turbinoptinae, Speleognathinae, and Cytoditidae). *Museum of Texas Tech University, Special Publication No. 8*, 148 pp. + 728 figs.

Pence, D. B. (1984). Diseases of laboratory animals. In W. B. Nutting (Ed.), *Vol. 2. Mammalian diseases and arachnids* (pp. 129−187). Boca Raton, FL: CRC Press.

Pence, D. B., Matthews, F. D., III, & Windberg, L. A. (1982). Notoedric mange in the bobcat, *Felis rufus*, from south Texas. *Journal of Wildlife Diseases, 18*, 47−50.

Pence, D. B., Spalding, M. G., Bergan, J. F., Cole, R. A., Newman, S., & Gray, P. N. (1997). New records of subcutaneous mites (Acari: Hypoderatidae) in birds, with examples of potential host colonization events. *Journal of Medical Entomology, 34*, 411−416.

Philip, C. B. (1948). Tsutsugamushi disease (scrub typhus) in the World War II. *The Journal of Parasitology, 34*, 169−191.

Philip, C. B., & Kohls, G. M. (1945). Studies on Tsutsugamushi disease (scrub typhus, mite-borne typhus) in New Guinea and adjacent islands: Tsutsugamushi disease with high endemicity on a small South Sea island. *American Journal of Hygiene, 42*, 195−202.

Philip, C. B., & Hughes, L. E. (1948). The tropical rat mite, *Liponyssus bacoti*, as an experimental vector of rickettsialpox. *The American Journal of Tropical Medicine and Hygiene, 28*, 697−705.

Phillips, C. J., Jones, J. K., & Radovsky, F. J. (1969). Macronyssid mites in oral mucosa of long-nosed bats: Occurrence and associated pathology. *Science, 165*, 1368−1369 (*Radfordiella*).

Pitariu, T., Dinulescu, N., Panaitescu, D., & Silard, R. (1978). Cholangiocholecystitis, an acute attack with acarids in B bile. *Revista de Igiena, Bacteriologie, Virusologie, Parazitologie, Epidemiologie, Pneumoftiziologie, 23*, 189−192 (in Romanian).

Pitariu, T. N., Popescu, I. G., & Banescu, O. (1979). Acarids of pathological significance in urine. *Revista de Igiena, Bacteriologie, Virusologie, Parazitologie, Epidemiologie, Pneumoftiziologie, 24*, 55−59 (in Romanian).

Pratt, H. D., & Stojanovich, C. J. (1969). Acarina: Illustrated key to some common adult female mites and adult ticks. In *CDC pictorial keys: Arthropods, reptiles, birds and mammals of public health significance* (pp. 26−44). U.S. Dept. Health and Human Services, Public Health Service, Centers for Disease Control and Prevention, 192 pp.

Prisich, I. I., Dobarskaia, L. I., & Zosimova, A. G. (1986). Acariasis in children with chronic digestive system diseases. *Meditsinskaia Parazitologiia I Parazitarnye Bolezni*, 50−51 (in Russian).

Proctor, H. C. (2003). Feather mites (Acari: Astigmata): Ecology, behavior, and evolution. *Annual Review of Entomology, 48*, 185−209.

Prosl, H., Rabitsch, A., & Brabenetz, J. (1985). Zur Bedeutung der Herbstgrasmilbe-*Neotrombicula autumnalis* (Shaw 1790)-in der Veterinarmedizin: Nervale Symptome bei Hunden nach massiver Infestation. *Tierarztliche Praxis, 13*, 57−64.

Quoix, E., Mao, J., Hoyet, C., & Pauli, G. (1993). Prediction of mite allergen levels by guanine measurements in house-dust samples. *Allergy (Copenhagen), 48*, 306−309.

Rafferty, D. E., & Gray, J. S. (1987). The feeding behaviour of *Psoroptes* spp. mites on rabbits and sheep. *The Journal of Parasitology, 73*, 901−906.

Raina, M. K. (1975). A monograph on the genus *Avitellina* Gough, 1911 (Avitellinidae: Cestoda). *Zoologische Jahrbücher. Abteilung für Systematik, Geographie und Biologie der Tiere, 102*, 508−552.

Raulston, G. L. (1972). Psorergatic mites in patas monkeys. *Laboratory Animal Science, 22*, 107.

Rollor, E. A., III, Nettles, V. F., Davidson, W. R., & Gerrish, R. R. (1978). Otitis media caused by *Psoroptes cuniculi* in white-tailed deer. *Journal of the American Veterinary Medical Association, 173*, 1242−1243.

Rufli, T., & Mumculoglu, Y. (1981). The hair follicle mites *Demodex folliculorum* and *Demodex brevis*: Biology and medical importance. A review. *Dermatologica, 162*, 1−11.

Samšinák, K., Palika, P., Zítek, K., Mališ, L., & Vobrázková, E. (1976). Are the mites of the genus *Tarsonemus* really parasites of man? *Folia Parasitologia, 23*, 91−93.

Samuel, W. M., Welch, D. A., & Smith, B. L. (1991). Ectoparasites from elk (*Cervus elaphus nelsoni*) from Wyoming. *Journal of Wildlife Diseases, 27*, 446−451.

Sánchez-Borges, M., Fernández-Caldas, E., Capriles-Hulett, A., & Caballero-Fonseca, V. (2012). Mite-induced inflammation: More than allergy. *Allergy & Rhinology (Providence), 3*, e25–e29.

Sánchez-Borges, M., Suárez-Chacon, R., Capriles-Hulett, A., Caballero-Fonseca, V., & Fernández-Caldas, E. (2013). Anaphylaxis from ingestion of mites: Pancake anaphylaxis. *The Journal of Allergy and Clinical Immunology, 131*, 31–35.

Sánchez-Borges, M., Suárez-Chacon, R., Capriles-Hulett, A., Caballero-Fonseca, F., Iraola, V., & Fernández-Caldas, E. (2005). Pancake syndrome (oral mite anaphylaxis). *World Allergy Organization Journal, 2*, 91–96.

Schultz, H. (1975). Human infestation by *Ophionyssus natricis* snake mite. *British Journal of Dermatology, 93*, 695–697.

Schuster, R., & Murphy, P. W. (1991). *The Acari: Reproduction, development and life-history strategies*. London: Chapman & Hall, 554 pp.

Scott, D. W. (1979). Canine demodicosis. *Veterinary Clinics of North America, 9*, 79–92.

Seier, J. V. (1985). Psorergatic acariasis in vervet monkeys. *Laboratory Animal, 19*, 236–239.

Sengbusch, H. G. (1977). Review of oribatid mite-anoplocephalan tapeworm relationships (Acari; Oribatei: Cestoda; Anoplocephalidae). In D. L. Dindal (Ed.), *Biology of oribatid mites* (pp. 87–102). Syracuse, NY: State University of New York, College of Environmental Science and Forestry.

Shaddock, J. W., & Pakes, S. P. (1978). Protozoal and metazoal diseases. In K. Benirschke, F. M. Garner, & T. C. Jones (Eds.), *Vol. 2. Pathology of laboratory animals* (p. 1587). Berlin: Springer-Verlag.

Sheldon, W. (1966). Psorergatic mange in the sooty mangabey (*Cercocebus torquates atys*) monkey. *Laboratory Animal Care, 16*, 276.

Shelley, W. B., Shelley, E. D., & Welbourn, W. C. (1985). Polypodium fern wreaths (Hagnaya). A new source of occupational mite dermatitis. *The Journal of the American Medical Association, 253*, 3137–3138.

Skoda-Smith, S., Mullen, G. R., Oi, F., & Atkinson, T. P. (1996). Angioedema following dust mite exposure presenting as suspected food allergy. *The American Academy of Allergy, Asthma & Immunology, 97*, 228 *(Dermatophagoides farinae)*.

Smiley, R. L. (1970). A review of the family Cheyletiellidae. *Annals of the Entomological Society of America, 63*, 1056.

Smiley, R. L., & OConnor, B. M. (1980). Mange in *Macaca arctoides* (Primates: Cercopithecidae) caused by *Cosarcoptes scanloni* (Acari: Sarcoptidae) with possible human involvement and descriptions of the adult male and immature stages. *International Journal of Acarology, 6*, 283–290.

Smith, K. E., Quist, C. F., & Crum, J. M. (1997). Clinical illness in a wild Turkey with *Laminosioptes cysticola* infestation of the viscera and peripheral nerves. *Avian Diseases, 41*, 484–489.

Smith, T. G. (1996). The genus *Hepatozoon* (Apicomplexa: Adeleina). *The Journal of Parasitology, 82*, 565–585.

Snyder, D. E., Hamir, A. N., Hanlon, C. A., & Rupprecht, C. E. (1991). Notoedric acariasis in the porcupine (*Erethizon dorsatum*). *Journal of Wildlife Diseases, 27*, 723–726.

Soulsby, E. J. L. (1965). *Textbook of veterinary clinical parasitology*. Philadelphia: F.A. Davis.

Soulsby, E. J. L. (1982). *Helminths, arthropods and protozoa of domesticated animals* (7th ed.). Philadelphia: Lea & Febiger, 809 pp.

Southcott, R. V. (1984). Diseases and arachnids in the tropics. In W. B. Nutting (Ed.), *Mammalian diseases and arachnids* (Vol. 2, pp. 15–56). Boca Raton, FL: CRC Press.

Spicer, G. S. (1987). Prevalence and host-parasite list of some nasal mites from birds (Acarina: Rhinonyssidae, Speleognathidae). *The Journal of Parasitology, 73*, 259–264.

Staley, E. C., Staley, E. E., & Behr, M. J. (1994). Use of permethrin as a miticide in the African hedgehog (*Atelerix albiventris*). *Veterinary and Human Toxicology, 36*(2), 138.

Stone, W. B., Parks, E., Weber, B. L., & Parks, F. J. (1972). Experimental transfer of sarcoptic mange from red foxes and wild canids to captive wildlife and domestic animals. *New York Archives – Game & Fish Journal, 19*, 1–11.

Strandtmann, R. W., & Wharton, G. W. (1958). *A manual of mesostigmatid mites parasitic on vertebrates*. Contribution No. 4 (pp. 1–330). Institute of Acarology, University of Maryland, College Park.

Stromberg, P. C., Fisher, W. F., Guillot, F. S., Pruett, J. H., Price, R. E., & Green, R. A. (1986). Systemic pathologic responses in experimental *Psoroptes ovis* infestation of Hereford calves. *American Journal of Veterinary Research, 47*, 1326–1331.

Stromberg, P. C., & Guillot, F. S. (1987a). Hematology in the regressive phase of bovine psoroptic scabies. *Veterinary Pathology, 24*, 371–377.

Stromberg, P. C., & Guillot, F. S. (1987b). Bone marrow response in cattle with chronic dermatitis caused by *Psoroptes ovis*. *Veterinary Pathology, 24*, 365–370.

Stunkard, H. W. (1940). The morphology and life history of the cestode *Bertiella studeri*. *The American Journal of Tropical Medicine and Hygiene, 20*, 305–333.

Sweatman, G. K. (1957). Life history, non-specificity, and review of the genus *Chorioptes*, a parasitic mite of herbivores. *Canadian Journal of Zoology, 35*, 641.

Sweatman, G. K. (1958a). Biology of *Otodectes cynotis*, the ear canker mite of carnivores. *Canadian Journal of Zoology, 36*, 849.

Sweatman, G. K. (1958b). On the life history and validity of the species in *Psoroptes*, a genus of mange mites. *Canadian Journal of Zoology, 36*, 905–929.

Sweatman, G. K. (1958c). Redescription of *Chorioptes texanus* a parasitic mite from the ears of reindeer in the Canadian Arctic. *Canadian Journal of Zoology, 36*, 525.

Sweatman, G. K. (1971). Mites and pentastomes. Chapter 1. In J. W. Davis, & R. C. Anderson (Eds.), *Parasitic diseases of wild mammals* (pp. 3–64). Ames: Iowa State University Press, 364 pp.

Sweatman, G. K. (1984). Diseases of wildlife, Chapter 8. In W. B. Nutting (Ed.), *Mammalian diseases and arachnids* (Vol. 2, pp. 189–232). Boca Raton, FL: CRC Press.

Taboada, M., & de, F. (1954). Pulmonary acariasis in Spain. An illustrative case report. *British Medical Journal, 4859*, 437–438.

Tamura, A., Ohahsi, N., Urakami, H., & Miyamura, S. (1995). Classification of *Rickettsia tsutsugamushi* in a new genus, *Orientia* gen. nov., as *Orientia tsutsugamushi* comb. nov. *International Journal of Systematic and Evolutionary Microbiology, 45*, 589–591.

Tandon, N., Chatterjee, H., Gupta, S. K., & Hati, A. K. (1988). Some observations on house dust mites in relation to naso-bronchial asthma in Calcutta, India. In G. P. ChannaBasavanna, & C. A. Viraktamath (Eds.), *Progress in acarology* (Vol. 1, pp. 163–168). Leiden, Netherlands: E. J. Brill.

Tenorio, J. M. (1974). A new species of *Lynxacarus* (Acarina: Astigmata: Listrophoridae) from *Felis catus* in the Hawaiian islands. *Journal of Medical Entomology, 11*, 599–604.

Theiler, M., & Downes, W. G. (1973). *The arthropod-borne viruses of vertebrates*. New Haven: Yale University Press, 578 pp.

Tomalski, M. D., & Miller, L. K. (1991). Insect paralysis by baculovirus-mediated expression of a mite neurotoxin gene. *Nature, 352*, 82–85 *(Pyemotes tritici)*.

Traub, R., Hertig, M., Lawrence, W. H., & Harris, T. T. (1954). Potential vectors and reservoirs of hemorrhagic fever in Korea. *American Journal of Hygiene, 59*, 291.

Traub, R., & Wisseman, C. L., Jr. (1974). The ecology of chigger-borne rickettsiosis (scrub typhus). *Journal of Medical Entomology, 11*, 237–303.

Traver, J. R. (1951). Unusual scalp dermatitis in humans caused by the mite *Dermatophagoides* (Acarina: Epidermoptidae). In *Proceedings of the Entomological Society of Washington, 53* (p. 1).

van Bronswijk, J. E. M. H., & Sinha, R. N. (1971). Pyroglyphid mites (Acari) and house dust allergy: A review. *The Journal of Allergy and Clinical Immunology, 47*, 31–52.

van Bronswijk, J. E., & De Kreek, E. J. (1976). *Cheyletiella* (Acari: Cheyletiellidae) of dog, cat and domesticated rabbit, a review. *Journal of Medical Entomology, 13*, 315–327.

van der Hammen, L. (1972). A revised classification of the mites (Arachnidea, Acarida) with diagnoses, a key, and notes on phylogeny. *Zoologische Mededelingen, 47*, 273–292.

Walker, E. D., & Landis, D. A. (1994). Straw itch mite, *Pyemotes tritici*, infestation in brome seed related to acute dermatitis in Michigan granary workers. *Great Lakes Entomologist, 27*, 125–128.

Wall, R., & Kolbe, K. (2006). Taxonomic priority in *Psoroptes* mange mites: *P. o*vis or *P. equi*? *Experimental and Applied Acarology, 39*, 159–162.

Wharton, G. W., Jr. (1976). House dust mites. *Journal of Medical Entomology, 12*, 577–621.

Wharton, G. W., Jr., & Fuller, H. S. (1952). A manual of the Chiggers: The biology, classification, distribution, and importance to man of the larvae of the family Trombiculidae (Acarina). In *Memoirs of the entomological society of Washington, No. 4*. Washington, D.C. 185 pp.

Whitaker, J. O., Jr. (1982). Ectoparasites of mammals of Indiana. In *Indiana Academy of Science, Monograph, 4*, 240 pp.

Wiedman, F. D. (1916). *Cytoleichus penrosei,* a new arachnoid parasite found in the diseased lungs of a prairie dog, *Cynomys ludovicianus*. *The Journal of Parasitology, 3*, 82–89 (Pneumocoptidae).

Williams, J. F., & Williams, C. S. (1978). Psoroptic ear mites in dairy goats. *Journal of the American Veterinary Medical Association, 173*, 1582–1583.

Williams, J. F., & Williams, C. S. (1982). Demodicosis in dairy goats. *Journal of the American Veterinary Medical Association, 180*, 168–169.

Wilson-Hanson, S., & Prescott, C. W. (1985). Trombidiosis in cats. *Australian Veterinary Journal, 62*, 202–203.

Wisniewski, H. M., Sigurdarson, S., Rubenstein, R., Kascsak, R. J., & Carp, R. I. (1996). Mites as vectors of scrapie. *Lancet, 347*, 1114.

Woolley, T. A. (1988). *Acarology: Mites and human welfare*. New York: John Wiley & Sons, 484 pp.

Wraith, D. G., Cunnington, A. M., & Seymour, W. M. (1979). The role and allergenic importance of storage mites in house dust and other environments. *Clinical & Experimental Allergy, 9*, 545–561.

Wright, F. C., Guillot, F. S., & Meleney, W. P. (1981). Transmission of psoroptic mites from bighorn sheep (*Ovis canadensis mexicana*) to domestic sheep, cattle and rabbits. *Journal of Wildlife Diseases, 17*, 381–386.

Wright, F. C., & Glaze, R. L. (1988). Blackbuck antelope (*Antilope cervicapra*), a new host for *Psoroptes cuniculi* (Acari: Psoroptidae). *Journal of Wildlife Diseases, 24*, 168–169.

Wright, F. C., Riner, J. C., & Fisher, W. F. (1984). Comparison of lengths of outer opisthosomal setae of male psoroptic mites collected from various hosts. *The Journal of Parasitology, 70*, 141–143.

Wright, F. C., Riner, J. C., & Guillot, F. S. (1983). Cross-mating studies with *Psoroptes ovis* (hering) and *Psoroptes cuniculi* Delafond (Acarina: Psoroptidae). *The Journal of Parasitology, 69*, 696–700.

Yashikawa, M., Hanaoka, Y., Yamada, Y., et al. (1983). Experimental proof of itching papules caused by *Cheyletus malaccensis* Oudemans. *Annual Report of Tokyo Metropolitan Research Laboratory of Public Health, 34* (pp. 264–276).

Yeatts, J. W. G. (1994). Rabbit mite infestation. *Veterinary Record, 134*, 359–360 *(Psoroptes cuniculi)*.

Yeruham, I., Rosen, S., & Hadani, A. (1986). Sheep demodicosis (*Demodex ovis* Railliet, 1895) in Israel. *Revue d Elevage et de Medecine Veterinaire des Pays Tropicaux, 39*, 363–365.

Yunker, C. E. (1973). Mites, Chapter 15. In R. J. Flynn (Ed.), *Parasites of laboratory animals* (pp. 426–492). Ames: Iowa State Univ. Press, 884 pp.

Yunker, C. E., Binninger, C. E., Keirans, J. E., Beecham, J., & Schlegel, M. (1980). Clinical mange of the black bear, *Ursus americanus*, associated with *Ursicoptes americanus* (Acari: Audycoptidae). *The Journal of Wildlife Diseases, 16*, 347–356.

Zahler, M., Hendrikx, W. M. L., Essig, A., Rinder, H., & Gothe, R. (2000). Species of the genus *Psoroptes* (Acari: Psoroptidae): A taxonomic consideration. *Experimental and Applied Acarology, 24*, 213–225.

Zhang, Z.-Q. (Ed.). (2011), *Zootaxa: Vol. 3148. Animal biodiversity: An outline of higher-level classification and survey of taxonomic richness* (pp. 1–237).

Zenoble, R. D., & Greve, J. H. (1980). Sarcoptid mite infestation in a colony of Guinea pigs. *Journal of the American Veterinary Medical Association, 177*, 903–908.

Zuchowska, E. (1991). Swierzb ssakow w ogrodach zoologiczynch [Scabies in zoo mammals]. *Wiadomosci Parazytologiczne, 37*, 123–125.

Chapter 27

Ticks (Ixodida)

William L. Nicholson[1], Daniel E. Sonenshine[2], Bruce H. Noden[3] and Richard N. Brown[4]

[1]*Rickettsial Zoonoses Branch, Division of Vector-Borne Diseases, National Center for Emerging and Zoonotic Infectious Diseases, Centers for Disease Control and Prevention, Atlanta, GA, United States;* [2]*Department of Biological Sciences, Old Dominion University, Norfolk, VA, United States;* [3]*Department of Entomology and Plant Pathology, Oklahoma State University, Stillwater, OK, United States;* [4]*Department of Wildlife, Humboldt State University, Arcata, CA, United States*

Ticks are well-known vectors of human and veterinary pathogens. They transmit a greater variety of infectious organisms than any other group of blood-sucking arthropods. Worldwide, they are the most important vectors in the veterinary field and are second only to mosquitoes in terms of their public health importance. Ticks transmit numerous protozoan, viral, bacterial (including rickettsial), and fungal pathogens. In humans, thousands of cases of tick-borne diseases caused by these agents occur annually, and the incidence of human disease is increasing globally. In addition, the bites of ticks can cause toxic reactions, allergic responses, and even fatal paralysis, while the wounds that they produce can create entry sites for secondary microbial infections and diminish the value of livestock by damaging their hides. Tick-borne diseases such as babesiosis, anaplasmosis, theileriosis, heartwater, and many others result in economic loss to those who raise livestock in many tropical and subtropical regions of the world. Although difficult to measure precisely, the global economic impact of ticks and tick-borne diseases is estimated to be in the multiple billions of dollars (U.S.) (Jongejan and Uilenberg, 2004). The study of ticks has contributed greatly to our ability to understand and control the spread of infectious diseases.

This chapter reviews the remarkable adaptations and behavior of ticks that facilitate their success as blood-feeding parasites and the diverse tick-host pathogen interactions that contribute to their role as vectors of human and animal disease agents. For more detailed information about the biology of ticks, the reader is referred to Sonenshine and Roe (Vol. 1, 2014). For detailed information on human tick-borne diseases, the reader is referred to Bowman and Nuttall (2008), Goodman et al. (2005), Sonenshine and Roe (Vol. 2, 2014), and Eisen et al. (2017). For more detailed information on veterinary tick-borne diseases, the reader should consult Uilenberg (1995), Jongejan and Uilenberg (2004), and Sonenshine and Roe (Vol. 2, 2014).

TAXONOMY

Ticks constitute the Suborder Ixodida, of the acarine Order Parasitiformes, and are exclusively parasitic. The Ixodida contain three extant families: the **Ixodidae**, **Argasidae**, and **Nuttalliellidae** (Table 27.1). The Ixodidae are subdivided

TABLE 27.1 Families and Genera of Ticks

Family	Subfamily (Subgroup)	Genera
Ixodidae	Ixodinae (Prostriata)	*Ixodes*
	Amblyomminae (Metastriata)	*Amblyomma*
	Bothriocrotoninae (Metastriata)	*Bothriocroton*
	Haemaphysalinae (Metastriata)	*Haemaphysalis*
	Hyalomminae (Metastriata)	*Hyalomma*
	Rhipicephalinae (Metastriata)	*Anomalohimalaya, Cosmioma, Dermacentor, Margaropus, Nosoma, Rhipicentor, Rhipicephalus*
Argasidae	Argasinae	*Argas*
	Ornithodorinae	*Ornithodoros, Carios*
	Otobinae	*Otobius*
Nuttalliellidae		*Nuttalliella*

After Horak et al. (2002); Murrell and Barker (2003).

Medical and Veterinary Entomology. https://doi.org/10.1016/B978-0-12-814043-7.00027-3

into the Prostriata, represented by the single genus *Ixodes* with 244 species, and the Metastriata with 459 species, comprising the remaining 14 genera (Guglielmone et al., 2010, 2014). There are 707 recognized species in this family, representing about 78% of all tick species that have been described (Table 27.1). The Argasidae contain four genera and about 190 species (Guglielmone et al., 2010). The **Nuttalliellidae** is a monospecific family, represented by only one species, *Nuttalliella namaqua*.

Ixodidae, also known as the hard ticks, are grouped into the Prostriata and the Metastriata based on morphologic characters. Prostriate ticks have a prominent anal groove located anterior to the anus and extending to the posterior body margins. Metastriate ticks have a small slit-like anal groove that is located posterior to the anus and does not extend to the body margin (Table 27.1).

For additional information on taxonomy of the Ixodida, please review Filippova (1966, 1967), Roberts (1970), Keirans (1992), Klompen and Oliver (1993), Durden and Keirans (1996), Klompen et al. (2000), Murrell et al. (2000), Barker and Murrell (2008), Walker et al. (2000), Beati and Keirans (2001), Barker and Murrell (2002), Horak et al. (2002), and Guglielmone et al. (2014).

The following subsections provide descriptions of some of the more important tick genera and species of particular importance as vectors of human or domestic animal disease-causing agents.

Family Ixodidae (Hard Ticks)

Genus *Ixodes*

This is the largest tick genus, with an estimated 244 species. *Ixodes* species are known as the **Prostriata**, characterized by a distinctive anal groove that encircles the anus anteriorly. They also lack eyes and other light-sensitive organs on the dorsum. Males have sclerotized ventral plates, which are absent in males of other genera. The genus is worldwide in distribution, including Antarctica. Four species are particularly important as vectors of microbial agents to humans: the **blacklegged tick** (*Ixodes scapularis*) in eastern North America; the **castor bean tick**, or **sheep tick** (*I. ricinus*) in Europe and western Asia; the **taiga tick** (*I. persulcatus*) in northeastern Europe and northern Asia; and the **western blacklegged tick** (*I. pacificus*) in the far western United States. Other, non-human-biting *Ixodes* spp. serve as enzootic (maintenance) vectors of important tick-borne disease agents, such as *I. dentatus, I. affinis,* and *I. spinipalpis* in North America, and *I. ovatus* in northern Asia and Japan.

Genus *Dermacentor*

This is one of the more important genera of metastriate ticks, with 35 species. The basis capituli appears rectangular when viewed dorsally. A pair of medially directed spurs occurs on the first pair of coxae. The palps are short and thick, and the scutum is almost always ornamented. Most *Dermacentor* spp. are three-host ticks that feed on diverse mammals. Adults attack medium-sized or large mammals, whereas the immatures feed on small mammals and lagomorphs. *Dermacentor* species are found mostly in Europe, Asia, Africa, and North and Central America. In North America, important species are the **American dog tick** (*D. variabilis*), the **Rocky Mountain wood tick** (*D. andersoni*), the **Pacific Coast tick** (*D. occidentalis*), and the **winter tick** (*D. albipictus*). In Central and South America and some Caribbean islands, an important species is the **tropical horse tick**, *D. nitens* (designated previously as *Anocentor nitens*). In Europe, two important species are *D. reticulatus* and *D. marginatus.*

Genus *Rhipicephalus*

Ticks of the genus *Rhipicephalus* are recognized by the hexagonal shape of the basis capituli when viewed dorsally. Important species include the **brown dog tick** (*R. sanguineus*) and the **brown ear tick** (*R. appendiculatus*). *Rhipicephalus* ticks mainly parasitize mammals, and only rarely are they found as larvae or nymphs on birds and reptiles. Representative species are found throughout the world. *Rhipicephalus sanguineus* is cosmopolitan in distribution, although more recent studies have indicated that the complex has both temperate and tropical lineages, which may be later assigned to separate species. Among the more important are the five species of the subgenus *Boophilus*, formerly considered as a separate genus (Murrel and Barker, 2003). Subgenus *Boophilus* ticks are small and lack ornamentation. The basis capituli is short and broad, with rounded lateral margins. These ticks are one-host parasites of ungulates. Subgenus *Boophilus* ticks are found in most tropical and subtropical regions of the world. Important species include the **cattle tick** (*Rhipicephalus [B.] annulatus*) and the **tropical fever tick** or **southern cattle tick** (*Rhipicephalus [B.] microplus*). The genus *Rhipicephalus* contains 84 described species.

Genus *Haemaphysalis*

This is the second largest tick genus, which is recognized by the pronounced lateral projection of palpal segment 2 in most species (including all three North American species), which extends well beyond the basis capituli. These small

ticks lack eyes. *Haemaphysalis* spp. parasitize birds and mammals in most regions of the world. An important species is the rabbit tick *H. leporispalustris*, widespread throughout much of North America. Several species in the Old World are important pests and/or vectors of animal and human disease agents, such as *H. longicornis* in Asia and the Pacific region (including Australia), *H. punctata* in Europe, and *H. spinigera* in India. The genus contains about 167 species.

Genus *Hyalomma*

This is a relatively small genus of ~30 species of medium-sized to large Old World ticks. They are characterized by their elongated palps, which are at least twice as long as wide. The distinct eyes are located in sockets adjacent to the posterolateral edges of the scutum. *Hyalomma* ticks are unornamented. Most species live in xeric environments where they parasitize small and medium-sized wild mammals and livestock. Some species parasitize birds or reptiles. The distribution of *Hyalomma* spp. is limited to the Old World, primarily in arid or semiarid habitats. An important subspecies is *H. marginatum marginatum*, a vector of Crimean-Congo hemorrhagic fever (CCHF) virus. Other important species are *H. truncatum* in Africa, *H. asiaticum* in central Asia, and *H. detritum* in Asia and the Mediterranean basin. *Hyalomma detritum* is of major veterinary importance as a vector of the agent of bovine tropical theileriosis.

Genus *Amblyomma*

Adults of most species in this genus are medium or large in size. The palps are long with segment 2 at least twice as long as segment 3. The scutum is usually ornamented with varying-colored iridescent patterns. Eyes are present in most species (absent in species formerly assigned to *Aponomma*) but are not situated in sockets. Virtually all terrestrial vertebrate species serve as hosts, although amphibians are rarely attacked. The distribution is worldwide, primarily in humid tropical or subtropical regions. Examples of important species include: the **Gulf Coast tick** (*A. maculatum*) and **lone star tick** (*A. americanum*) in North America; the **tropical bont tick** (*A. variegatum*) in Africa and on some Caribbean islands; and the **bont tick** (*A. hebraeum*) in Africa. The genus contains about 130 species (including 20 species formerly assigned to the genus *Aponomma*, now in part, a synonym of *Amblyomma*).

The remaining genera of the Ixodidae contain relatively few species, none of which are known to be important in pathogen transmission. These include *Anomalohimalaya, Bothriocroton, Cosmiomma, Nosoma, Margaropus,* and *Rhipicentor*. A genus previously designated as *Anocentor* was invalidated and its single species transferred to the genus *Dermacentor*. Similarly, the genus *Aponomma* is no longer considered valid and its species were transferred to the genus *Amblyomma* or to the new genus *Bothriocroton.*

Family Argasidae (Soft Ticks)

Genus *Argas*

Argas ticks have a flattened body margin, a lateral sutural line, and a leathery, folded cuticle. The many small integumental folds usually have a button-like appearance, each with a pit on its top. Most species parasitize bats or birds. The genus is worldwide in distribution, mostly in xeric environments or dry caves in otherwise humid environments. Examples of important species are the **fowl tick** (*A. persicus*) and the **pigeon tick** (*A. reflexus*). About 57 species have been described.

Genus *Carios*

These ticks are similar to those of the genus *Argas*, but differ in the structure of their Haller's organ on the tarsus of the foreleg. In most *Carios* the setiform seta is replaced by a second serrate seta. The roof of Haller's organ in both subgenera is solid, lacking perforations The host range includes mammals (mainly bats) and birds. Approximately 10 species formerly classified in the genus *Antricola*, now a synonym of *Carios* according to some authors, possess a tuberculated cuticle. The females have a distinctive, scooplike hypostome; the hypostome is vestigial in the males. The *Antricola* species are parasites of New World bats. Thus far, none has been implicated in the transmission of microbial disease agents. A species formerly classified in the genus *Nothoaspis*, now a synonym of *Carios* according to some authors, is similar to *Antricola*, but the anterior dorsal surface bears a smooth shield-like structure, the pseudoscutum. The current classification of the genus *Carios* is based on Horak et al. (2002) and Klompen and Oliver (1993). One important species is *Carios capensis*, found on seabirds. The bat tick, *Carios kelleyi,* has been implicated as a potential vector of rickettsiae and borreliae (Reeves et al., 2006). The genus contains approximately 87 species.

Genus *Ornithodoros*

Nymphs and adults have a leathery cuticle with innumerable tiny wrinkles and small protuberances (mammillae) and a rounded body margin; they lack a lateral, sutural line. Mammillae are smaller and more numerous than those found in *Argas*. The host range is diverse and includes reptiles, birds, and mammals. The genus is worldwide in distribution. Examples of important species include the **African tampan**

(*O. moubata*) and the **cave tick** (*O. tholozani*). In North America, several species (e.g., *O. hermsi*, etc.) are important as vectors of relapsing fever spirochetes to humans and animals. The genus contains about 38 species.

Genus *Otobius*

The integument of the nymphs is spinose, whereas that of the adults is granulated. There are just two nymphal instars. The adults do not feed, and the hypostome is vestigial. *Otobius* ticks are found in North America, Africa, and Asia, having been inadvertently introduced onto the latter two continents. The genus contains two species: the **spinose ear tick** (*O. megnini*) and *O. lagophilus*.

Family Nuttalliellidae

The only known species in this family, *Nuttalliella namaqua*, occurs in eastern and southern Africa. It shares features with both the Argasidae and the Ixodidae but also has several unique morphological traits. This tick has ball-and-socket joints that articulate the leg segments, a small, dorsal pseudoscutum, and a highly wrinkled cuticle with numerous pits and elevated rosettes. While considered a generalist feeder, it has been collected from the nests of rock hyraxes and swallows in South Africa, Namibia and Tanzania (Mans et al., 2011; Latif et al., 2012). Although rare and of no known medical or veterinary importance, recent studies of the biology and ecology of this species have provided new insights into the evolution of ticks (Mans et al., 2011, 2012).

MORPHOLOGY

External Anatomy

The major external regions of ticks are the **capitulum** (gnathosoma), **idiosoma**, and the legs (Figs. 27.1–27.3). The capitulum (Figs. 2.3C, 27.3) consists of the **basis capituli**, which articulates with the body; the segmented **palps**, the **chelicerae**, and the toothed **hypostome**. The capitulum of ixodid ticks is located at the anterior end of

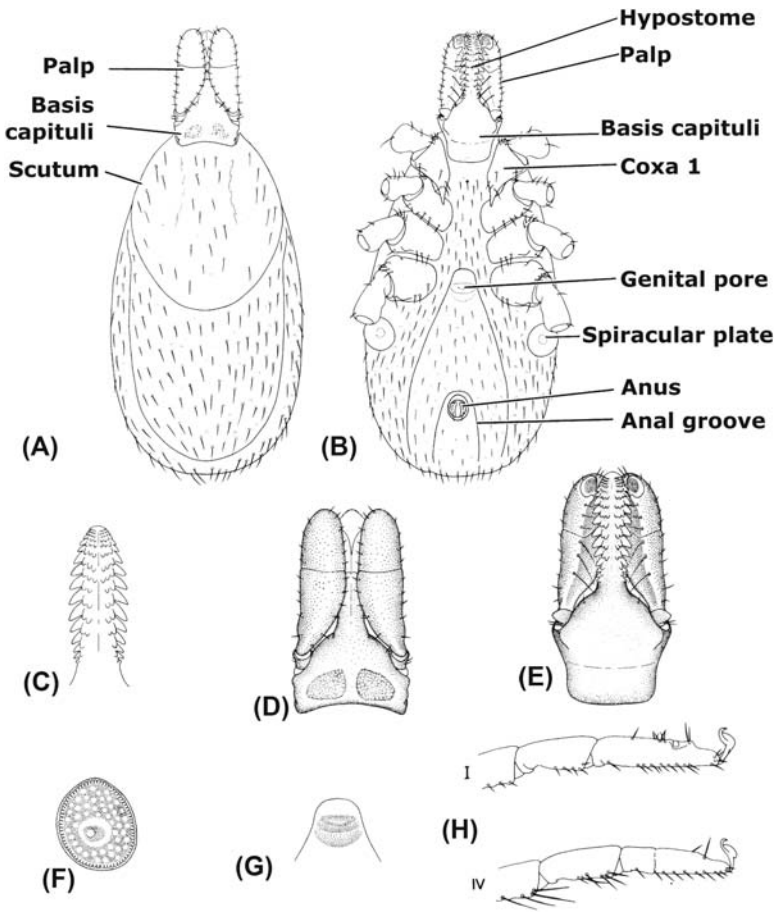


FIGURE 27.1 External morphology of representative female ixodid tick (*Ixodes pacificus*). (A) Dorsal view. (B) Ventral view. (C) Hypostome. (D) Capitulum, dorsal view. (E) Capitulum, ventral view. (F) Spiracular plate. (G) Genital pore. (H) Legs I and IV. *Modified from Sonenshine (1991), with permission of Oxford University Press.*

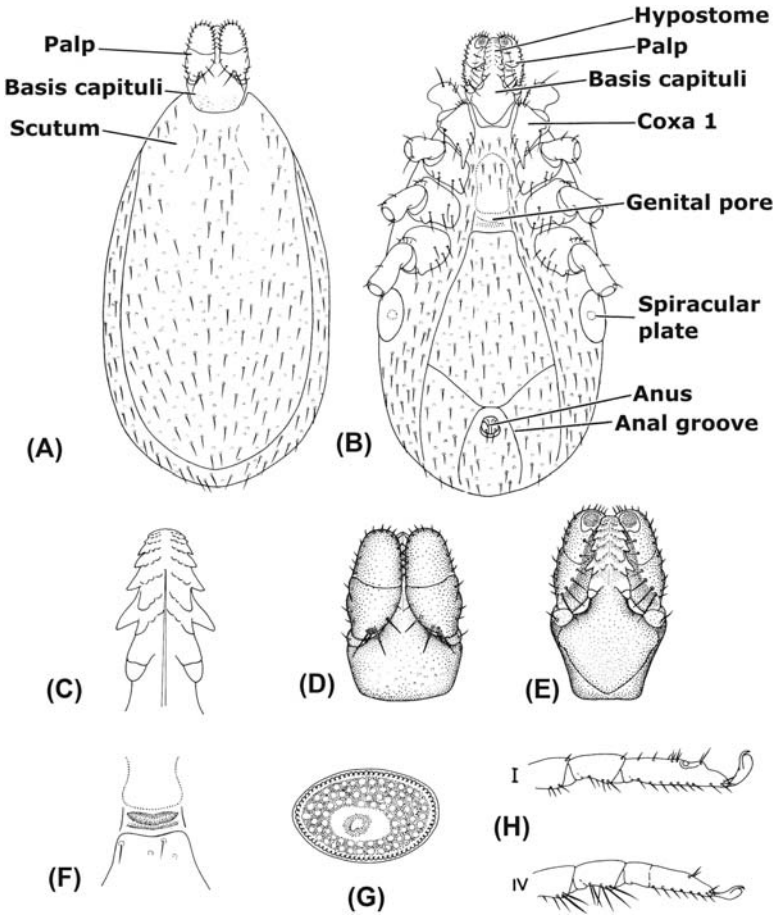


FIGURE 27.2 External morphology of representative male ixodid tick (*Ixodes pacificus*). (A) Dorsal view; (B) Ventral view; (C) Hypostome; (D) Capitulum, dorsal view; (E) Capitulum, ventral view; (F) Genital pore; (G) Spiracular plate; (H) Legs I and IV. *Modified from Sonenshine (1991), with permission of Oxford University Press.*

the body. Females bear paired clusters of pores, the **porose areas**, located dorsally on the **basis capituli**. The porose areas secrete antioxidants that inhibit degradation of the waxy compounds in the secretions of **Gené's organ**, which coat the eggs as they are laid. The **chelicerae** are located on the dorsal aspect of the capitulum. Their shafts, surrounded by spinose sheaths, lie between the palps and often extend even farther anteriorly than the palps. Each chelicera bears two digits distally. The larger, medial digit can be moved laterally; the smaller outer digit resides in a cavity of the medial digit and moves with it. Both digits have sharp denticles. The chelicerae are used to cut host tissues during attachment. The hypostome is a prominent, ventrally located structure that bears rows of recurved teeth on its ventral surface; teeth are absent in some nonfeeding males. A narrow food canal is located on the mid-dorsal surface. The palps consist of four distinct segments. In nymphs and adults of most ixodid species, the small terminal (fourth) segment is recessed in a cavity in segment 3 and bears numerous fine setae at its tip.

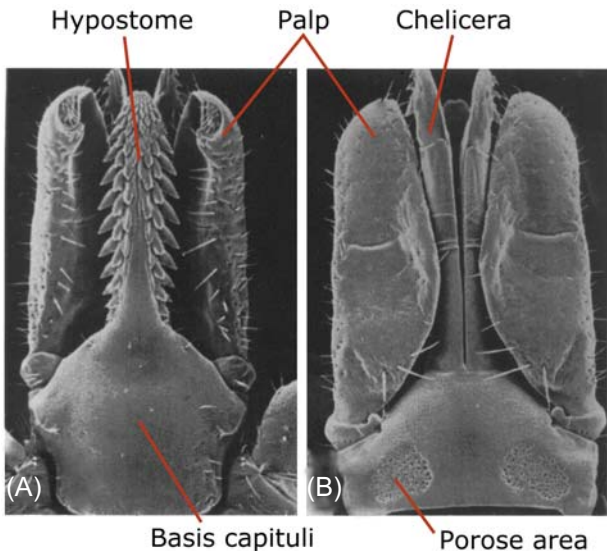


FIGURE 27.3 Capitulum of a representative ixodid tick (*Ixodes scapularis*), scanning electron micrographs. (A) Ventral view. (B) Dorsal view. *Modified from Sonenshine (1991), with permission of Oxford University Press.*

The capitulum of adult and nymphal argasids is similar. However, it is situated just below an anteriorly protruding body extension, or **hood**, and is not visible dorsally in nymphs or adults (Figs. 27.4 and 27.5). The four palpal segments are about equal in size. Small flaps, the cheeks, occur alongside the capitulum in many species and can be folded to cover the delicate mouthparts. In argasid larvae, the mouthparts protrude anteriorly, as in ixodids.

The body, exclusive of the capitulum, is the **idiosoma**. It is divided into two parts: the anterior podosoma that bears the legs and the genital pore, and the posterior opisthosoma, the region behind the coxae that bears the spiracles and the anal aperture. The cuticle is relatively tough with sclerotized plates (sclerites) in certain locations. It serves as the site of muscle attachment and protects the animal from desiccation and injury. The cuticle bears numerous sensory setae as well as various pores representing the openings of dermal glands or sensilla.

The legs are jointed and articulate with the body via the **coxae**. Larvae are easily recognized by the presence of only three pairs of legs, whereas nymphs and adults have four pairs of legs. The structure of the legs is similar in the Ixodidae and Argasidae. Each leg is divided into six segments: the coxa, trochanter, femur, patella (=genu), tibia, and tarsus. The coxae are inserted ventrally and allow limited rotation in the anteroventral and dorsoventral planes. The other segments can be flexed, so that the legs can be either folded against the ventral body surface for protection or extended for walking. A pair of claws and a padlike pulvillus are present on each tarsus of most species. The pulvillus is absent in argasid nymphs and adults. An odor-detecting sensory apparatus, **Haller's organ** (Fig. 27.6), is evident on the dorsal surface of the tarsus of leg I in all stages. This organ consists of an anterior pit and a posterior capsule. Olfactory and mechanosensory, but not gustatory functions also have been associated with this organ (Nuss et al., 2016). The tick's Haller's organ uses novel molecular processes for chemosensation different from those found in insects. Haller's organ also functions as an infrared receptor (Mitchell et al., 2017). Variations in the structure of Haller's organ are useful for distinguishing genera and species.

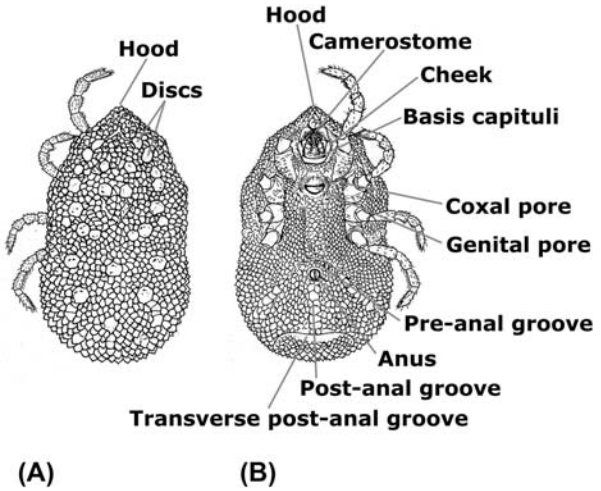


FIGURE 27.4 External morphology of a generalized argasid tick (*Ornithodoros*). (A) Dorsal view. (B) Ventral view.

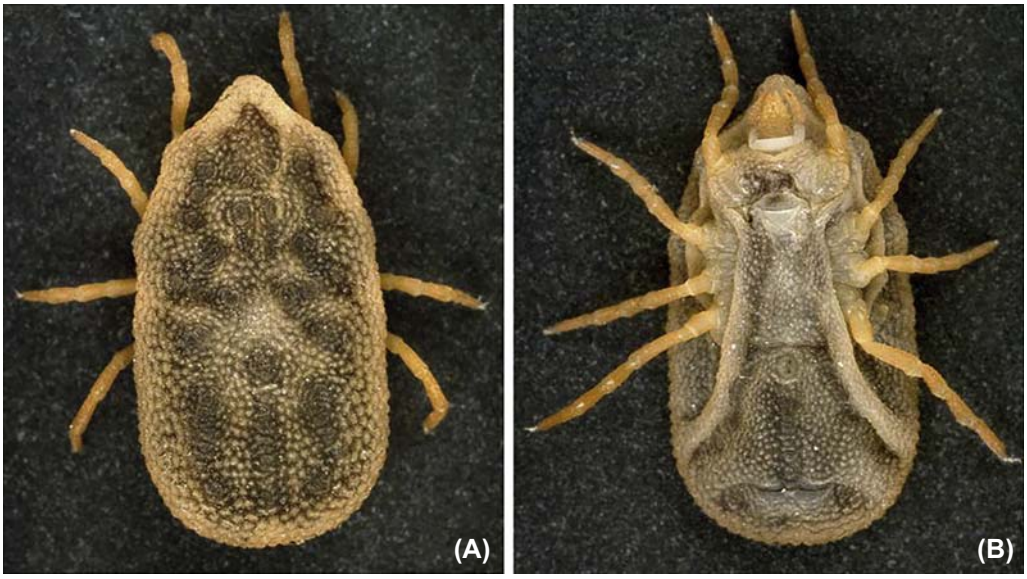


FIGURE 27.5 A representative argasid tick (*Carios kelleyi*). (A) Female, dorsal view. (B) Female, ventral view. *Courtesy of Jim Gathany, Centers for Disease Control and Prevention.*

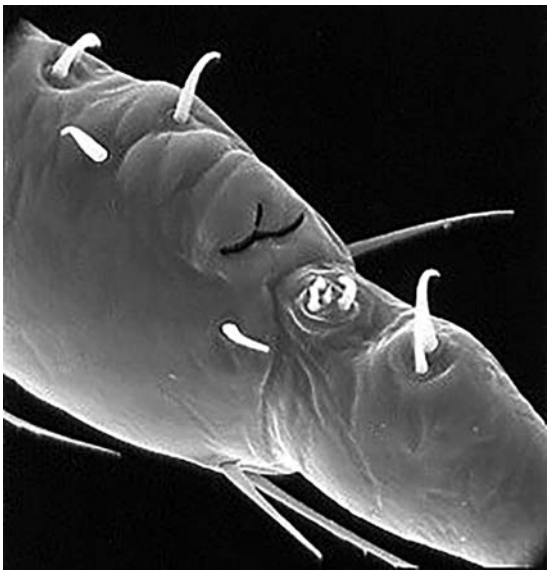


FIGURE 27.6 Haller's organ of the larval stage of *Amblyomma rotundatum*, a parasite of reptiles and amphibians in the Americas; scanning electron micrograph. *Courtesy of the U.S. National Tick Collection, Georgia Southern University.*

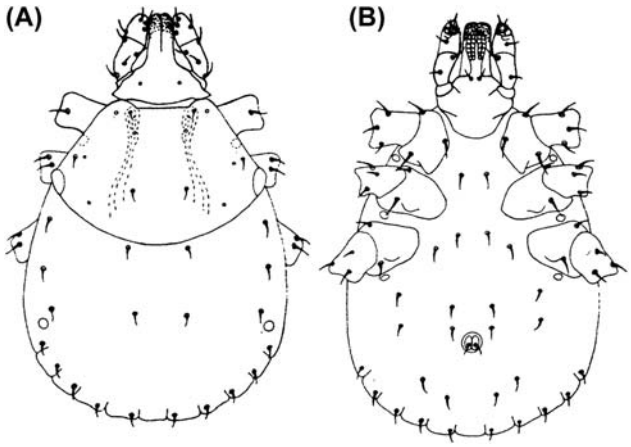


FIGURE 27.7 Larva of representative ixodid tick (*Dermacentor variabilis*), with legs beyond coxae and trochanters not shown. (A) Dorsal view. (B) Ventral view. *From Clifford et al. (1961).*

Ixodidae

Ixodid ticks, also called **hard ticks**, are illustrated in Figs. 27.1–27.3. Females have a hard cuticular plate or scutum on the anterior half of the dorsal body surface (Fig. 27.1A). In males, the scutum occupies virtually the entire dorsal surface (Fig. 27.2A). Elsewhere, the cuticle contains tiny surface folds, which give it a fingerprint-like appearance when viewed at high magnification. The body of the female posterior to the scutum expands enormously during feeding as new cuticle is synthesized to accommodate the bloodmeal. In males, however, the larger scutum limits expansion. The scutum bears setae and tiny pores termed **sensilla auriformia**. The latter are believed to serve as proprioceptive organs. When present, a simple eye occurs along each posterolateral margin of the scutum.

Immediately posterior to the scutum in the females are paired **foveal pores** (absent in *Ixodes)* from which a volatile sex pheromone, 2,6-dichlorophenol, is emitted. The dorsal body surface posterior to the scutum, the **alloscutum**, has innumerable fine folds. In females, a paired protrusible organ, Gené's organ, lies in the dorsal foramen between the scutum and the capitulum (capitular foramen). The ends of this organ protrude during oviposition and apply wax to each egg as it is deposited. In *Ixodes* males, hard sclerotized plates cover the ventral body surface (Fig. 27.2B). In females, the genital pore is a U- or V-shaped opening, with prominent marginal folds (Fig. 27.1G), but in males it is covered by a movable plate (Fig. 27.2F). Other ventral structures include: paired spiracular plates behind coxae IV in adults and nymphs (absent in larvae), each with a small ostium that opens to the respiratory system; and the anal aperture, located near the posterior margin. The entire body is covered by numerous setae and the porelike sensilla auriformia. Larvae possess few setae, although their number and relative placement provide valuable taxonomic characters for generic and subgeneric differentiation (Fig. 27.7).

Argasidae

The major external body features of argasid ticks, also known as **soft ticks**, are illustrated in Figs. 27.4 and 27.5. The body margins are rounded in most species. In *Argas*, however, they are flattened and covered by small marginal discs. Eyes, when present, occur on folds lateral to the coxae. A tiny coxal pore, the opening of the duct from the paired coxal glands, occurs bilaterally between the coxae of legs I and II. The spiracular plates, located between coxae III and IV, are relatively small and inconspicuous. In females, the genital pore appears as a horizontal slit surrounded by a prominent fold. In males, the pore is subtriangular or suboval, without a genital apron. There are no foveal pores.

Internal Anatomy

The internal organs of a typical tick are illustrated in Fig. 27.8. The organs are bathed in a circulating fluid, the hemolymph. The hemolymph is an aqueous medium rich in salts, amino acids, soluble proteins, and other dissolved substances. In addition, it contains several types of hemocytes, the most prominent of which are the plasmatocytes and granulocytes. These cells often function in phagocytosis of invading microbes, as well

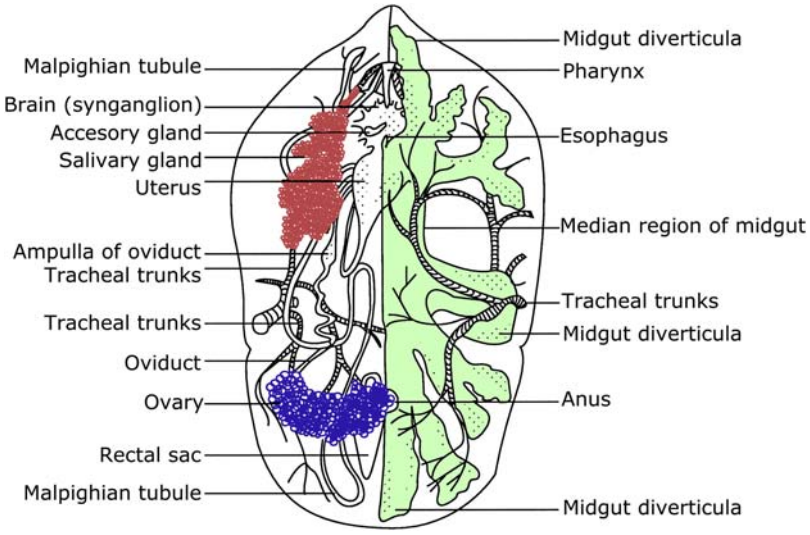


FIGURE 27.8 Internal anatomy of typical argasid tick, female; midgut shown on right side; midgut removed on left side to reveal underlying organs. *Modified from Balashov (1972), with permission from Copyright Agency of Russia.*

as other functions (Matsuo et al., 2009). Four major categories of cell types are generally recognized in tick hemolymph, namely, prohemocytes, nongranular plasmatocytes, granulocytes (type I and II), and spherulocytes (Borovickova and Hypša, 2005). A simple heart, situated mid-dorsally, filters and circulates this vital body fluid. Muscles extend from the dorsal and ventral cuticular surfaces to the inner surfaces of the coxae, chelicerae, and other structures.

The most prominent internal organ is the midgut, a large saclike structure with numerous lateral diverticula. The shape of the midgut depends on the state of engorgement. In unfed ticks, the diverticula are narrow, tubelike structures. In fed ticks, they enlarge and obscure most of the other organs as they fill with blood. Branches of the tracheal system ramify over the surfaces of the diverticula and surround the other internal organs. Ticks respire through these innumerable tiny air tubes, which open to the exterior via the paired spiracles.

Paired salivary glands are situated anterolaterally. These large glands, which resemble clusters of grapes, are connected via the salivary ducts to the mouthparts. Their salivary secretions empty into the salivarium located between the chelicerae and the hypostome. Tick saliva contains pharmacologically active compounds that facilitate attachment and suppress host inflammatory responses. The salivary glands eliminate excess water from the bloodmeal. In ixodid ticks, most water in the bloodmeal is extracted by specialized salivary-gland cells and excreted into the host as the tick feeds.

Other prominent internal structures are the reproductive organs. In males, these include the testes, the tubular vasa deferentia, the seminal vesicle, and the ejaculatory duct that is connected to the genital pore. The ejaculatory duct is obscured by the large, multilobed accessory gland that secretes the components of the spermatophore. In females, the reproductive organs include the ovary, paired oviducts, uterus, vagina, and the seminal receptacle. The ovary is small and inconspicuous in unfed ticks but expands enormously during feeding and especially after mating. In gravid females, the ovary is distended with large, amber-colored eggs.

In argasid and ixodid ticks, excretion is accomplished by the **Malpighian tubules**, a pair of long, coiled structures that empty into the rectal sac. Nitrogenous wastes are excreted in the form of guanine. In argasid ticks, paired **coxal glands** adjacent to the coxae of leg I extract excess water and salts accumulated during feeding and excrete this watery waste via the coxal pores. Each gland consists of a membranous sac that serves as a filtration chamber and a coiled tubule that selectively reabsorbs small, soluble molecules and ions. Relapsing fever spirochetes may be transmitted to vertebrate hosts via the **coxal fluid** of infected ticks.

The central nervous system in ticks is fused to form the **synganglion**, located anteroventrally above the genital pore. The synganglion, which regulates the function of the structures described above, is the fused central nervous system. Large pedal nerves extend from the synganglion to the legs; smaller nerves innervate the palps, chelicerae, cuticular sensilla, and the internal organs. Transcriptomes of the synganglion of different tick species have described expression of neuropeptides, neuropeptide receptors, and neurotransmitter receptors and their roles in regulating their physiological processes (Bissinger et al., 2011; Sonenshine et al., 2014; Egekwu et al., 2016). Remarkably, almost all

of these molecules occur in the synganglia of the different tick species examined, including ixodid and an argasid tick species. However, statistically significant differences were observed in gene expression of genes that regulate patterns of blood-feeding, water elimination, pharyngeal pump action, cuticle synthesis, and reproductive activity; differences that help explain the major differences in feeding, development, and reproduction between ixodid and argasid ticks.

LIFE HISTORY

The life cycle includes four stages: the egg, larva, nymph, and adult. Ixodid ticks have only one nymphal instar, whereas argasid ticks have two or more nymphal instars. All ticks feed on blood during some or all stages in their life cycle; therefore, they are obligate ectoparasites. Larvae attack hosts, feed, detach, and develop in sheltered microenvironments where they molt to nymphs. Nymphs seek hosts, feed, drop, and molt to adults (except in argasid ticks, which molt into later nymphal instars). Adult ticks seek hosts, feed, and, in the case of engorged ixodid females, drop off to lay their eggs (Fig. 27.9).

In contrast to most other hematophagous arthropods, ticks can be remarkably long-lived. Many can survive for one or more years without feeding. Their life cycles vary greatly, with the greatest differences evident between the Ixodidae and Argasidae.

Ixodid Life Cycles

Immature and adult ticks each take a bloodmeal, except for the nonfeeding males of some species (especially members of the genus *Ixodes*). Following contact with the host, a tick uses its chelicerae to puncture the skin and its hypostome to securely anchor itself. In many species, attachment is known to be reinforced by secretion of cementing substances with the saliva into and around the wound site. Females feed only once. Following mating, females ingest blood rapidly (24–48 h) and swell enormously. Replete, mated females drop from their hosts, find a sheltered location, and subsequently oviposit hundreds to thousands of eggs (Fig. 27.9). For *D. variabilis*, the average egg production is 5,400. For *Hyalomma impeltatum*, the reported average is nearly 10,700 eggs per female. The greatest number ever recorded was produced by an *Amblyomma nuttalli* female that produced close to 23,000 eggs. The eggs are deposited in a single, continual mass over many days or weeks. The female dies on completion of egg laying.

FIGURE 27.9 Lone star tick (*Amblyomma americanum*), female, that has just finished depositing an egg mass of about 4,000 eggs. *Photograph by Gary R. Mullen.*

Males swell only slightly during feeding. They usually remain on their hosts, feed repeatedly, and inseminate several females. Mating typically occurs on the host. Certain species of *Ixodes,* however, mate on their hosts, in nests, or in vegetation. Many *Ixodes* males have vestigial hypostomes, and these species invariably mate off the host. Except for *Ixodes* species, males and females require a bloodmeal to stimulate oogenesis and spermatogenesis. More than 90% of the life cycle is spent off the host. Molting usually occurs in some sheltered microhabitat such as soil and leaf litter, or in host nests. After molting, nymphal and adult ticks must seek another host and feed. When host seeking and feeding occur in all three parasitic stages, the pattern is termed a **three-host life cycle** (Fig. 27.10). This is characteristic of more than 90% of ixodid species.

In tropical climates with frequent rainfall, developmental times are relatively short, and several generations may occur each year. In regions with alternating dry and rainy seasons, the life cycle is longer because ticks cease host seeking during the driest period. In colder temperate or subarctic regions, development is slower, and ticks commonly undergo diapause during the coldest months. As a result, the life cycle may take two or more years. An example of a diapausing species is *Dermacentor variabilis*. Larvae feed on mice or other small mammals, mostly in spring. Fed larvae drop off and molt to nymphs that again attack small mammals. Fed nymphs drop off and molt within a few weeks. If the adults feed and reproduce in the same year, the entire life cycle can be completed within several months. Thus, under favorable conditions in nature, the typical three-host life cycle can be completed in less than 1 year. However, adverse environmental conditions can prolong the life cycle to 2 or more years. The life cycle of *Ixodes ricinus* may require up to 4 years in the northern parts of its range in Europe

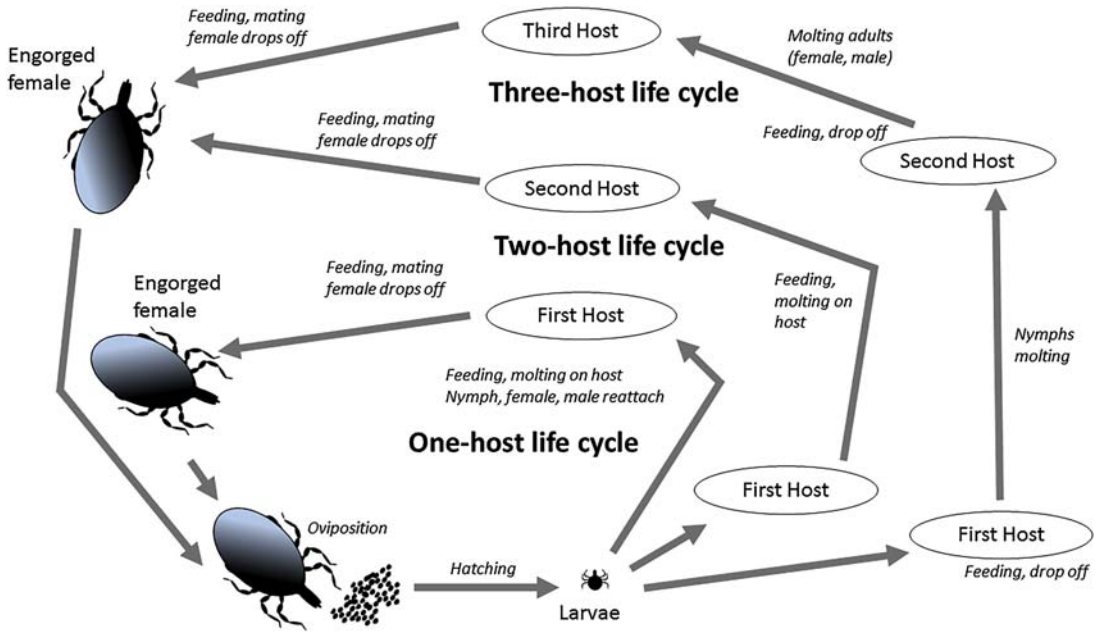


FIGURE 27.10 Three basic life cycles of ixodid ticks: (1) one-host ticks (inner circle) in which the larva, nymph, and adult all attach to, and develop on, a single host (e.g., *Rhipicephalus (Boophilus) annulatus*). (2) Two-host ticks (middle circle) in which larva and nymph feed on one host and the adult attaches and feeds on a second host (e.g., *Hyalomma dromedarii*). (3) Three-host ticks (outer circle) in which larva, nymph, and adult each parasitize a different host (typical of most ixodid ticks). Most argasid ticks have a multihost life cycle involving more than three hosts; with several nymphal instars, each potentially feeding on a different host. *Courtesy of W. L. Nicholson.*

(Hoogstraal, 1985). In Ireland, the life cycle takes 3 years, with each stage requiring approximately 1 year before developing to the next (Gray, 1991).

A few ixodid species exhibit a **two-host** or **one-host life cycle** (Fig. 27.10). For example, in the two-host camel tick (*Hyalomma dromedarii*), fed larvae molt on their hosts, and the unfed nymphs reattach soon after emergence. Following engorgement, the nymphs drop off the host, molt, and feed again as adults on a second host. In the one-host cattle tick *R.* (*B.*) *annulatus*, and in other species of the subgenus *Boophilus*, all stages feed and molt on the same host. Mating also occurs on this host. Replete, fertilized females drop off the host and oviposit in soil.

FIGURE 27.11 Argasid tick (*Ornithodoros turicata*), female, depositing small batches of eggs. *Photograph by Jerry F. Butler.*

Argasid Life Cycles

In contrast to the ixodids, most argasids have two or more nymphal instars in their life cycle, each of which must consume a bloodmeal. This pattern is termed the multihost life cycle. Molting occurs off the host in cracks, crevices, or beneath debris in or near the nest. Argasid females take repeated small bloodmeals and lay small batches of eggs (Fig. 27.11), typically fewer than 500 eggs/batch after each feeding. These are termed multiple gonotrophic cycles, and as many as six gonotrophic cycles have been reported in some species. The interval between feedings is typically several months. Mating usually occurs off the host. Because of the multiple nymphal instars that may number six or even seven in some species, argasid ticks often live for many years. In addition, these ticks are highly resistant to starvation, which can extend their longevity even further. In some species that feed on migratory bats or birds, diapause serves to delay oviposition or development during the periods when hosts are absent.

The larvae of most *Ornithodoros* and *Carios* spp. that parasitize bats and birds remain attached to their hosts for many days, just as do ixodid ticks. Following the larval bloodmeal, they molt twice without additional feeding. Thereafter, the life cycle is similar to the typical argasid pattern. Another species with an unusual life cycle is *Otobius megnini*. This tick exhibits a high degree of host and body-site predilection specificity that regulates its feeding and development. Females do not feed and are autogenous (i.e., oviposit without feeding).

BEHAVIOR AND ECOLOGY

Feeding behavior, even on preferred hosts, is not a uniform process. Blood-feeding begins soon after contact and acceptance of recognition features that determine that the animal is a suitable host. In ixodids, a tick may crawl about the host for several hours in search of a suitable feeding site. Once a site has been selected, the tick cuts into the skin with its cheliceral digits and inserts its hypostome to initiate the attachment process. Shortly after they attach, most ixodid ticks secrete cement during the first 1−2 days to secure themselves at the wound site. Subsequently, the tick begins salivating into the developing hematoma and sucking blood; salivating and blood sucking alternate, often for extended periods of time for each process. The feeding lesion enlarges as the tick injects anticoagulant and antihemostatic compounds into the wound; recruitment of host leucocytes to the wound site also contributes to tissue lysis and fluid influx around the tick's mouthparts. Successful blood feeding depends upon the secretion of an extensive array of antihemostatic, anti-inflammatory, and immunomodulatory proteins and lipids in the tick saliva so as to suppress the host's ability to reject the feeding tick. Of particular importance in tick saliva are antagonists of the intrinsic pathway factor X (Xa) and factor V (Va), which converts prothrombin to thrombin. Thrombin in turn converts plasma fibrinogen to fibrin and clots the blood. Tick saliva blocks blood coagulation by inhibiting factor Xa. In addition, many species also secrete proteins that inhibit thrombin directly or inhibit the conversion of prothrombin to thrombin by inhibiting factor V. Other salivary proteins prevent platelet aggregation, also important for blood coagulation, and proteins that bind, antagonize, or degrade important host mediators of pain, itching, and inflammation, particularly the host's histamine, serotonin, and bradykinin. Nevertheless, ticks feeding on the same host (i.e., tick-sensitized individuals) often encounter significant resistance and are unable to engorge, drop off, or even die, a phenomenon known as acquired resistance to tick bite (Wikel, 2014). Our knowledge of the tick's ability to antagonize the host's hemostatic mechanisms is still incomplete (Ribeiro et al., 2017), but more information has become available with new advances using next-generation sequencing, genomics, proteomics, and metabolomics of tick salivary glands (e.g., Ribeiro et al., 2006; Kazimírová and Štibrániová, 2013; Chmelař et al., 2016).

Digestion of the bloodmeal takes place in the midgut. Erythrocytes and other blood cells are lysed soon after ingestion. Hemoglobin from lysed cells binds to receptors on the midgut epithelial cells and is incorporated by a process known as receptor-mediated endocytosis into tiny vacuoles in the digestive cells (Coons et al., 1986). The vacuoles fuse with lysosomes, forming specialized phagolysosomes, wherein hydrolytic enzymes released into these acidic vacuoles carry out digestion of the hemoglobin (Gough and Kemp, 1995; Mendiola et al., 1996). Subsequently, a cascade of proteolytic enzymes digest the hemoglobin, liberating dipeptides and free amino acids for transport out of the cell and into the hemolymph (Horn et al., 2009). Most of the heme released following hemoglobin digestion is detoxified to hematin, which accumulates in specialized "hemosomes" (Lara et al., 2003), while the amino acids liberated from the globin moieties are passed into the hemolymph. The latter provides the primary nourishment derived from the tick's bloodmeal. Although some other proteins, various lipids, and carbohydrates are also digested, most proteins ingested with the bloodmeal remain in the midgut lumen.

Ixodid ticks feed gradually prior to mating because first they must create new cuticle to accommodate the massive bloodmeal. Typical attachment periods range from as few as 2 days for larvae to as long as 13 days for females. When feeding is completed, the weight of blood and other fluids consumed ranges from 11 to 17 times the tick's prefeeding body weight in ixodid larvae and from 60 to 120 times the tick's prefeeding body weight in ixodid females. Measurements of blood volume consumed range from 0.7 mL to as high 8.9 mL per female in some ixodid species. Nymphal and adult argasid ticks attach for only brief periods. This can be as little as 35−70 min for adults. These ticks do not secrete cement during attachment; instead, they attach solely with the mouthparts, especially the hypostome. Argasid ticks swell to the extent that their cuticles can stretch. New cuticle is not secreted during feeding as it is in ixodid ticks.

In ticks, mating can occur either during feeding or off the host. In the metastriate Ixodidae, mating occurs while the adults are attached and feeding. Unfed adults are sexually immature and require a bloodmeal to stimulate gametogenesis. Mating is usually regulated by sex pheromones and follows a complex, hierarchical pattern

of responses. Feeding females secrete a volatile **sex-attractant** pheromone, usually **2,6-dichlorophenol**, that excites males feeding nearby on the same host. The males detach and seek the females that they recognize by means of the mounting sex pheromone (a mixture of cholesteryl esters) on the female's body surface. The male climbs onto the dorsum of the female, then moves to her ventral surface and searches for her gonopore. Once a male locates the gonopore, he probes the opening with his chelicerae. Spermatophores, containing spermatozoa, are produced in the large accessory gland of the sexually mature male during the mating process. At this time, the spermatophore emerges from the male's genital pore, whereupon the male seizes it with his mouthparts and inserts it into the female's vulva. Copulation is essential to initiate rapid engorgement by the blood-feeding females. In most ixodid ticks, the attached females do not fully engorge unless inseminated by a conspecific male, and the ovary remains in the nonvitellogenic state; however, parthenogenesis is known to occur in some species. Full engorgement by the female leads to a remarkable sequence of molecular and physiological changes that result in vitellogenin production, ovarian development, and oviposition (Lomas et al., 1997; Mitchell et al., 2007; Thompson et al., 2005, 2007).

In prostriate Ixodidae and in argasid ticks, the adults become sexually mature soon after the nymphal molt. These ticks usually mate in the nests or in vegetation, although ticks of some prostriate species also may mate on the host. There is evidence that mating in argasid ticks is regulated by one or more sex pheromones, although no specific compounds have been identified to date.

A preovipositional period of up to several weeks precedes egg laying in ixodid ticks. During oviposition, the cuticle of the vulva softens and evaginates as the eggs pass through it, thereby serving as an ovipositor. The emerging eggs are waxed by secretions from **Gené's organ**. The process of oviposition continues for several weeks. Typically, about 50%−60% of the female's body weight at the time of drop-off is converted to eggs. The number of eggs deposited is directly proportional to the size of the engorged female. At the completion of oviposition, the spent female dies. Thus, there is only a single gonotrophic cycle among ixodids. In the Argasidae, however, mated females commence oviposition soon after feeding, but deposit small clutches containing only a few hundred eggs. Following oviposition, the females remain active and seek hosts again. These ticks feed and lay eggs after each meal; they do not need to mate again. The number of gonotrophic cycles varies but rarely exceeds six.

Most tick species live in forests, savannahs, second-growth areas of scrub and brush, and grassy meadows. Others remain buried in sand or sandy soils, under stones, in crevices, or in the litter, duff, and rotting vegetation at the floor of woods and grasslands. In contrast, almost all argasids and some ixodids, especially males and immatures of several species of the genus *Ixodes*, are **nidicolous**. They live in the nests, burrows, caves, or other shelters used by their hosts.

Non-nidicolous ticks are active during certain periods of the year when climatic conditions favor development and reproduction. During such periods they engage in host-seeking behavior. In temperate and subpolar regions, seasonal activity is regulated by ambient temperature, changing photoperiod, and incident solar energy. In tropical regions, where day length or temperature varies only slightly throughout the year, tick activity is often controlled by the transition from the dry to the rainy season. Host-seeking ticks exhibit at least two strategies for locating potential targets for their bloodmeals. Ambush ticks climb onto weeds, grasses, bushes, or other leafy vegetation to wait for passing hosts. When stimulated by the presence of a host, they extend their forelegs anterolaterally in what is called **questing behavior** (shown in Fig. 27.12A) and quickly grasp the hair, feathers, or clothing of a passing host. Hunter ticks emerge from their refuges in the soil, sand, or duff when excited by host odors and run rapidly across the ground to attack hosts.

After contacting a potential vertebrate host, the tick must perceive appropriate host-recognition cues that enable it to determine whether to attach and feed, or to drop off and resume host-seeking. Odors, radiant heat, visual images, or vibrations stimulate the tick and enable it to recognize its prospective host. Odors are probably the most important stimuli. Electrophysiological studies have shown that larvae of *Rhipicephalus (B.) microplus* respond to odors from extracts of cattle skin but not to dry air. Human breath also elicits a response but not as vigorously as that caused by cattle extracts. Among the more important attractants emitted by hosts are carbon dioxide in animal breath, and ammonia in urine and other animal wastes. Other odors that attract ticks are butyric acid and lactic acid, which occur commonly in sweat and other body fluids. Acetone and 1-octen-3-ol have also been shown to attract ticks (Carr et al., 2013). Little is known about the molecular biology of chemoreception in ticks. Using next-generation sequencing and comparative transcriptomics, Carr et al. (2017) found that the chemosensory function of the tick Haller's organ is olfactory but not gustatory. The olfactory mechanism is different from insects, lacking odorant binding peptides, and apparently using an olfactory G protein-coupled receptor (GPCR) signal cascade unique to the Haller's organ for odor detection and the terminator protein β-arrestin to terminate

FIGURE 27.12 The brown dog tick (*Rhipicephalus sanguineus*). (A) Female, dorsal view. (B) Male dorsal view. *Photograph courtesy of Centers for Disease Control and Prevention.*

neuronal responses (Carr et al., 2017). Small increases in radiant heat excite ticks and act synergistically with host odors. Visual cues may be important, especially in certain hunter ticks that can discriminate dark shapes against the bright background of the sky. Host-seeking ticks of many species respond to shadows, resulting in extension of their legs to facilitate contact. Remarkably, the Haller's organ of ixodid ticks can also function as an infrared detector (Mitchell et al., 2017) enabling the ticks to respond to distant warm-blooded hosts, especially at night.

Other stimuli that elicit questing behavior include vibrations, sound, and tactile cues. Vibrating the grass stems on which ticks are perched can elicit questing behavior almost immediately. *Rhipicephalus (B.) microplus* larvae respond to sounds in the 80−800 Hz range, typical of the frequencies produced by feeding cattle, whereas the sounds produced by barking dogs reportedly attract *Rhipicephalus sanguineus* (Waladde and Rice, 1982). Tactile stimuli perceived when ticks contact their hosts, in combination with short-range attractants such as heat and odor, help to determine the selection of suitable feeding sites. Ticks will not attach to a host unless the appropriate stimuli are received in a particular sequence.

The timing of drop-off from the host offers important ecological advantages. For non-nidicolous ticks, such drop-off rhythms are synchronized with host behavioral patterns. This tends to disperse fed ticks in optimal habitats where they can develop and reproduce. Photoperiod appears to be the dominant exogenous factor affecting drop-off patterns. The daily light:dark cycle induces a regular rhythm of feeding and dropping off. This effect, termed **photoperiodic entrainment** is partially reversible. In a series of elegant experiments with *H. leporispalustris,* the existence of an endogenous drop-off rhythm entrained by the scotophase or dark period was shown. The rhythm was affected only partially by changing the photoperiodic regime and was maintained even when hosts were held in constant darkness. Detachment may occur while the hosts are inactive in their nests or burrows or, alternatively, it may be coordinated with the period of maximum host activity. In *D. variabilis*, fed larvae and nymphs drop soon after night begins. In contrast, immatures of *H. leporispalustris* drop off during daylight hours when their lagomorph hosts are confined in their burrows or warrens.

Ticks exhibit varying degrees of host specificity. More than 85% of argasid and ixodid ticks exhibit relatively strict host specificity. However, the evolutionary significance of this phenomenon is uncertain. Others have debated that most of the existing tick-host-association patterns may be explained as artifacts of biogeography and ecological specificity as well as incomplete sampling (McCoy et al., 2013). Regardless of how host specificity evolved, it is clear many species are specialists. Examples include certain species of argasids (e.g., some *Carios* spp.) that infest only bats, and the ixodids *Dermacentor*

albipictus, R. (*B.*) *annulatus,* and *R.* (*B.*) *microplus* that feed only on large ruminants. Many of the nidicolous ticks are highly host-specific. Examples include *Ixodes marxi* that feeds almost exclusively on squirrels, *Amblyomma tuberculatum* that, in most stages and primarily as adults, parasitizes the gopher tortoise, and *Argas arboreus* that attacks herons.

At the opposite extreme are ticks that are opportunistic species with catholic feeding habits. Examples include *Ixodes ricinus* and *I. scapularis.* Larvae and nymphs of these species feed readily on lizards, birds, small mammals, and larger hosts like sheep and humans. Adults feed on larger mammals, especially ungulates, and also attack humans. More than 300 species of vertebrates have been recorded as hosts for *I. ricinus,* and more than 120 have been recorded for *I. scapularis*.

Host specificity is influenced by evolutionary history, ecological and physiologic factors, and the ability of the ticks to avoid host rejection. Many species belonging to the less specialized and phylogenetically primitive genus *Amblyomma* and all species of the former *Aponomma* (now included in *Amblyomma* or *Bothriocroton*) feed on reptiles or primitive mammals. Ticks adapted to a specific habitat type (e.g., grassland) encounter only those vertebrates adapted to the same habitat. Questing height also is important. Ticks questing on or near the ground are exposed mostly to small animals, while those questing higher in the vegetation are more likely to encounter larger animals.

The extent to which different hosts are used depends on host behavior and opportunities for contact, such as foraging range, time of day and time spent foraging, habitats visited, and other factors. White-footed mice, which forage extensively on the ground, acquire numerous *I. scapularis* immatures, whereas flying squirrels, which spend much less time on the ground, are rarely infested. A similar relationship exists among migratory birds. In the United States, ground-feeding birds that forage in habitats shared with cottontail rabbits are heavily infested with immatures of the rabbit tick *H. leporispalustris*. Birds that forage above ground in bushes or trees rarely encounter these ticks. Acceptance of a vertebrate animal also is dependent on physiological factors and the ability of the ticks to recognize it as a host.

Host utilization may be influenced by the ability of ticks to evade or suppress host homeostatic systems and avoid rejection. This was first reported in a landmark paper by Trager (1939), who noted that the feeding success of *D. variabilis* on guinea pigs declined with frequent re-exposures. The guinea pig is a South American rodent and an unnatural host for this North American tick. When fed on its natural hosts (e.g., white-footed mice), *D. variabilis* experiences little if any rejection when feeding on tick-naïve hosts, but increasing rejection may occur when ticks attempt to feed on tick-sensitized hosts (Krause et al., 2009). Similarly, *I. scapularis* saliva contains pharmacologically active compounds that suppress host mediators of edema and inflammation such as anaphylatoxins, bradykinin, and other kinins, but lacks compounds to suppress histamine. Because histamine-induced edema does not occur in the white-footed mouse, this does not deter tick feeding on this host. However, recent research indicates that white-footed mice do develop a strong, granulomatous inflammation in response to repeated tick challenge by *I. scapularis,* but the dermal architecture is largely unaffected, allowing the ticks to remain attached (Anderson et al., 2017). However, histamine-containing basophils are very abundant in guinea pigs, and cutaneous basophil hypersensitivity develops rapidly even after a single feeding by these ticks (Ribeiro, 1989). *Ixodes scapularis* saliva also contains an enzyme that destroys complement, thereby facilitating the survival of pathogens such as *Borrelia burgdorferi* ingested during blood-feeding (Valenzuela et al., 2000).

Ticks occur in many terrestrial habitats ranging from cool, arboreal northern forests to hot, arid deserts. Each species, however, has become adapted to specific types of habitats where generally it is found in greater abundance. Typical habitat associations of non-nidicolous ixodid ticks include forests, meadows and other clearings, grasslands, savannahs, and semi-desert or desert areas. At one end of the spectrum are species that have very limited resistance to desiccation and occur in cool, moist forests (e.g., *I. scapularis* and *I. ricinus*). In the middle are the many species that can survive at least brief periods of desiccation during host seeking or development (e.g., *Dermacentor variabilis, Amblyomma maculatum,* and *A. americanum*). At the other end of the spectrum are the desiccation-tolerant species adapted to survive in arid steppes, semi-deserts, and other xeric environments (e.g., *Hyalomma asiaticum* and *Ornithodoros savignyi*).

Water balance is a critical determinant of a tick's ability to survive while waiting for hosts, sometimes requiring weeks or months. When they begin to desiccate, they retreat to more sheltered, humid microenvironments such as the rotting vegetation in a meadow or damp leaf litter on the forest floor. They secrete a hygroscopic salivary secretion onto their mouthparts that collects atmospheric water (direct sorption). After repeated cycles of secretion and drinking the condensed water, the rehydrated ticks are able to resume host-seeking. Some ticks are able to remain in the questing position for many days without rehydration, while others must return to their humid microenvironments each day.

Ixodes scapularis is an example of a tick with very limited desiccation tolerance. Consequently, it is most abundant in dense, humid, forest habitat or in dense shrub-

dominant habitats adjacent to large rivers, bays, or the Atlantic Ocean. Another desiccation-intolerant species is *Ixodes ricinus*. This tick is widespread in the British Isles, Continental Europe, and western Asia, where it frequents woodlands, damp meadows, pastures, and ecotones.

Dermacentor variabilis is an example of a species exhibiting greater tolerance to desiccation. It flourishes in the ecotone between secondary growth deciduous forests and lush, grassy meadows, as well as along secondary roads and trails in forested habitats. The dense ecotonal vegetation provides shade, increased moisture, protection from intense solar radiation, and food plants that support the tick's mammalian hosts. This type of environment is ideal for the immature stages of *D. variabilis*. Adults, with their greater resistance to desiccation and greater mobility, venture further afield to quest in sunlit meadows or along roads and trails.

The camel tick, *Hyalomma dromedarii*, is an example of a desiccation-tolerant species. This desert tick is common in the steppes and semi-desert habitats in large areas of North Africa and the Middle East. Larvae and nymphs generally live in rodent burrows. Adults bury themselves in sand and duff near their hosts, especially around caravansaries and similar locations where camels and other livestock are kept.

Nidicolous ticks living in or near the nests of their hosts are adapted to highly specialized environments. Normally the temperature and relative humidity in a burrow, cave, or similar type of shelter are more uniform throughout the year than in the external macroenvironment. The higher relative humidity in such microenvironments is due in part to the presence of hosts, their wastes, and plant materials used to construct or line their nests. Nidicolous ticks exhibit behavioral patterns that restrict their distribution to these sheltered locations. They avoid bright sunlight and low humidity, the type of conditions prevalent at the entrances of burrows or caves. Confined within these cryptic, restricted locations, nidicolous ticks become active when hosts are present. However, when hosts are absent, they may wait for up to several years for hosts to return or until they die of starvation.

Seasonal activity refers to the period of the year when ticks actively seek hosts. For example, *D. variabilis* larvae emerge from winter **diapause** in spring to feed on small mammals, especially mice and voles. Activity accelerates rapidly as increasing numbers of larvae emerge from overwintering sites to attack hosts, culminating in the seasonal peak within a few weeks. Thereafter, activity continues unabated, with larval abundance declining as more individuals find hosts, desiccate, or die of starvation. Nymphal and adult ticks also feed during the warm spring and early summer months. In the southern parts of its range, overwintering *D. variabilis* adults emerge early and soon overlap with those that develop from nymphs fed in the spring. Thus, the seasonal peak for adults occurs in early summer. As a result, most females oviposit in July and August, and the newly hatched larvae enter diapause as day length diminishes. In the southeastern United States, the entire life cycle is completed in 1 year. Occasionally, a small secondary peak of *D. variabilis* larval activity occurs in the fall. In the northern part of its range, however, tick activity is delayed due to cooler spring temperatures and shorter day lengths. As a result, although larvae and nymphs feed in the late spring and summer, adults emerge too late in the summer to commence questing activity. These adults undergo diapause and emerge the following spring. This pattern of feeding and diapause results in a 2-year life cycle.

Ixodes scapularis also exhibits distinct seasonal activity periods. In the northern part of its range, larval activity does not occur until middle or late summer and the nymphs that molt from the fed larvae diapause until the following spring. Nymphs that feed in the spring molt in summer, but the young adults delay host-seeking until fall. This pattern results in a 2-year life cycle. In the southernmost part of its range, *I. scapularis* activity occurs earlier in the year. These southern populations may complete their life cycle in just 1 year.

A few tick species are active during the cooler months of the year, especially fall and winter. Larvae of the winter tick (*Dermacentor albipictus*), a 1-host tick that feeds on horses, deer, elk, moose, and other large ungulates, commence host-seeking activity in late summer or early fall. Larvae and nymphs feed and molt on the same hosts and the resulting adults reattach, feed, and mate. Replete females drop off the host and oviposit in the soil. In the northern-most parts of its range, adults usually do not appear until late winter, with peak occurrence in April. Subsequent oviposition and hatching occur in late spring. At this time, larvae undergo diapause, presumably in response to increasing daylight, and do not commence host seeking until after an extended period of declining photoperiod. Development proceeds faster farther south. On stanchioned bovines at Kerrville, Texas, in the southern United States, the entire process of feeding, molting, and production of engorged females is completed in 21–36 days. Engorged females that drop off their hosts in winter do not oviposit until the following spring.

In tropical regions, where day length is nearly uniform throughout the year and where there is no prolonged dry season, the seasonal activity of many tick species (e.g., *Rhipicephalus appendiculatus*) is often influenced by the distribution of rainfall. Farther from the equator, particularly in colder regions in southern Africa, *R. appendiculatus* diapauses during the dry season.

In contrast, most argasid ticks do not exhibit patterns of seasonal activity. This is especially true for ticks infesting

the nests or burrows of nonmigratory hosts such as rodents and carnivores. However, nidicolous ticks that parasitize migratory birds and bats tend to delay oviposition so that hatching occurs at about the time the hosts return.

Diapause is an important behavior that enables ticks to survive adverse environmental conditions and conserve energy until conditions improve. Diapausing ticks become inactive, reduce their metabolic rates, and do not feed on hosts even when given the opportunity. Newly emerged larvae, freshly molted nymphs, and adults of many species enter diapause before seeking hosts, particularly if they emerge during periods of declining photoperiod. This is termed host-seeking diapause. Diapause enables *D. variabilis* larvae and adults to survive the cold winters that occur throughout most of the tick's range. It also determines the length of the life cycle, 1 year in the south but 2 years in the northern part of its range. Diapause delays activity of *I. scapularis* nymphs at more northern latitudes so that they do not commence host seeking until spring or early summer. It also may delay adult activity that usually begins in fall, often several months after molting (Gray et al., 2016).

Another type of diapause is morphogenetic diapause, in which development or oviposition is delayed. In *Dermacentor marginatus*, oviposition rather than hatching is delayed. Thus, females that feed in spring or early summer lay eggs immediately, but those that feed in late summer or early fall oviposit the following spring. A remarkable example of morphogenetic diapause occurs in certain argasid ticks that inhabit the nests of birds or the roosts of bats. Females of the bat tick *Carios kelleyi* (Fig. 27.5) oviposit immediately after feeding in spring. However, those that feed in fall delay oviposition until the following spring. Because the bats migrate to cold caves or caverns far from the tick's normal habitats, this ovipositional delay avoids the risk that larvae will emerge at a time when no hosts are available.

TICK SPECIES OF MEDICAL-VETERINARY IMPORTANCE

The following ticks are important as household pests, species that transmit disease agents to humans, and species that are injurious to livestock or transmit disease agents to animals. The more important tick-borne diseases of humans and other animals are listed in Tables 27.2 and 27.3.

The **brown dog tick** (*Rhipicephalus sanguineus*) exists currently as a complex of species (Fig. 27.12) and is a common household pest throughout most of the world (Nava et al., 2014). The primary host for all life stages is the domestic dog, which can become heavily infested. However, in many areas bordering the Mediterranean Sea, western Asia, and Africa, this tick also feeds readily on a wide range of wildlife (especially small mammals) and also attacks humans. Recent work has demonstrated tropical and temperate lineages that may exhibit biological differences. It often infests kennels, houses, and peridomestic areas, especially when dogs are kept indoors, which can produce considerable distress when the owners encounter thousands of these ticks. Seasonal activity peaks in summer, although activity peaks can occur throughout the year when ticks inhabit heated homes. This species is the primary vector of *Rickettsia conorii*, which causes **Mediterranean spotted fever** or **Boutonneuse fever** in many Mediterranean countries. It has been implicated as the vector for *Rickettsia rickettsii* in the southwestern United States, Mexico, and parts of Central and South America. In dogs and some other animals, this tick is a vector of the agents of cyclic thrombocytopenia (*Anaplasma platys*), canine ehrlichiosis (*Ehrlichia canis*), canine hepatozoonsis (*Hepatozoon canis*), and canine babesiosis (*Babesia vogeli* and a small *B. gibsoni*-like species).

The **brown ear tick** (*Rhipicephalus appendiculatus*) and related species are major pests of livestock in eastern, central, and southern Africa. Hosts include most domestic ruminants and many wildlife species, to which it attaches predominantly in and around the ears. It is the vector of the agent of **East Coast fever**, a protozoan disease that afflicts ruminant livestock within its range. Many other species of *Rhipicephalus* are important livestock pests and vectors of pathogens (e.g., *R. bursa* is a vector of the agents of equine, bovine, and small ruminant babesiosis and bovine and ovine anaplasmosis in the Medterranean area). *Rhipicephalus evertsi* and *R. turanicus* are two other species injurious to livestock.

The genus *Haemaphysalis* contains numerous species that attack mammals and birds. In North America, an important species is the widespread **rabbit tick** (*H. leporispalustris*). Larvae and nymphs attack ground-feeding birds as well as lagomorphs, while adults feed only on lagomorphs. Larvae and nymphs are active in the late summer and fall, while adults feed in the spring. This species contributes to the maintenance of **Rocky Mountain spotted fever** (*Rickettsia rickettsii* infection) among wildlife. In India, an important species of this genus is *H. spinigera* that occurs in dense forest habitat. Larvae feed on small mammals and ground-feeding birds, but nymphs and adults attack larger animals, including monkeys, cattle, and even humans. Larvae are active during October and November, nymphs from November to June, and adults mostly in July and August. This tick species is the principal vector of the virus that causes **Kyasanur Forest disease**.

In Europe, *H. punctata* may transmit mild forms of the agents of bovine babesiosis (*B. major*) and theileriosis (*T. buffeli* and *T. orientalis*) or babesiosis of small ruminants (*B. motasi*). *Haemaphysalis longicornis* infests

TABLE 27.2 Representative Tick-Borne Diseases of Public Health Importance and Associated Characteristics (All Tick-Borne Diseases Have Not Been Included)

Disease	Causative Agent	Primary Tick Vector Species	Animal Host(s) Beyond Humans
Human babesiosis	*B. microti,*	*Ixodes scapularis*	Rodents, cattle
	B. divergens	*Ixodes ricinus*	
	B. duncani (WA1, CA5)	Unknown	
	"B. ventorum" (EU-1)	*Ixodes ricinus*	
Tick-borne encephalitis	*Flavivirus*[a]	*I. ricinus, I. persulcatus*	Rodents, insectivores, carnivores, etc.
Kyasanur Forest disease	*Flavivirus*[a]	*Haemaphysalis spinigera*	Monkeys, small mammals, carnivores, birds, cattle
Powassan encephalitis	*Flavivirus*[a]	*Ixodes, Dermacentor,* and *Haemaphysalis* spp.	Rodents, hares, carnivores
Colorado tick fever	*Coltivirus*[b]	*Dermacentor andersoni*	Rodents, carnivores, domestic animals
Heartland virus	*Phlebovirus*[c]	*Amblyomma americanum*	Possibly raccoons and deer
Severe fever with thrombocytopenia syndrome virus	*Phlebovirus*[c]	*Haemaphysalis longicornis*	Goats, wild animals
Bourbon virus	*Thogotovirus*[d]	*Amblyomma americanum*	Deer, raccoons
Crimean- Congo hemorrhagic fever	*Nairovirus*[c]	*Hyalomma m. marginatum, H. m. rufipes,* others	Hares, hedgehogs, small mammals
Rocky Mountain spotted fever	*Rickettsia rickettsii*	*Dermacentor variabilis, D. andersoni, A. cajennense, Rhipicephalus sanguineus,* others	Small mammals, carnivores, dogs, rabbits, others
Boutonneuse fever[e]	*Rickettsia conorii*	*R. sanguineus, D. marginatus, D. reticulatus,* others	Small mammals, hedgehogs, dogs
African tick-bite fever	*Rickettsia africae*	*Amblyomma* spp.	Mammals, including humans
Rickettsia parkeri rickettsiosis	*Rickettsia parkeri*	*Amblyomma maculatum* group ticks	Cotton rats and others, cotton mice, dogs
Pacific Coast fever	"*Rickettsia philipii*" (364D)	*Dermacentor occidentalis*	Unknown, likely rodents
Human ehrlichiosis	*Ehrlichia chaffeensis*	*Amblyomma americanum,*	Deer, dogs
Human ehrlichiosis	*Ehrlichia ewingii*	*Amblyomma americanum*	Dogs, deer
Ehrlichiosis	*Ehrlichia muris eauclairensis*	*Ixodes scapularis*	*Peromyscus leucopus,* dogs
Human anaplasmosis	*Anaplasma phagocytophilum*	*Ixodes scapularis, I. pacificus I. ricinus, I. persulcatus*	Rodents, deer, dogs
Human anaplasmosis	*Anaplasma platys*	*Rhipicephalus sanguineus*	Dogs
Human anaplasmosis	*"Anaplasma capra"*	*Ixodes persulcatus*	Goats, sheep
Human anaplasmosis	*Anaplasma ovis*	*Rhipicephalus* spp., *Dermacentor* spp.	Sheep
Neoehrlichiosis	*Neoehrlichia mikurensis*	*Ixodes ricinus, I. persucatus*	Rodents, canines, badger, fox
Q fever	*Coxiella burnetii*	Many tick species	Large domestic livestock
Lyme disease	*Borrelia burgdorferi B. afzelii, B. garinii B. bissettii*	*Ixodes scapularis, I. ricinus, I. pacificus, I. persulcatus,* others	Mammals, birds

Continued

TABLE 27.2 Representative Tick-Borne Diseases of Public Health Importance and Associated Characteristics (All Tick-Borne Diseases Have Not Been Included)—cont'd

Disease	Causative Agent	Primary Tick Vector Species	Animal Host(s) Beyond Humans
Tick-borne relapsing fever	*Borrelia* spp.	*Ornithodoros* spp.	Various mammals
Tularemia	*Francisella tularensis*	*Haemaphysalis leporispalustris*, others	Lagomorphs, rodents, carnivores
Tick paralysis	Tick proteins	*I. holocyclus, I. rubicundus, D. variabilis, D. andersoni,*	Cattle, sheep, dogs, other mammals, birds, others
Tick-bite allergies	Tick proteins	*Argas reflexus, Ornithodoros coriaceus, Ixodes pacificus*, etc.	Humans

[a]*Family Flaviviridae.*
[b]*Family Reoviridae.*
[c]*Family Bunyaviridae.*
[d]*Family Orthomyxoviridae.*
[e]*Also known as Mediterranean spotted fever.*

cattle and other large mammals in eastern Asia and the Pacific area, and was recently discovered in the United States (New Jersey and at least eight other states). This tick transmits the agent of bovine babesiosis (*B. ovata*) and a more pathogenic version of *T. buffeli/T. orientalis* in eastern Asia. Ticks of the *H. leachi* group are vectors of the agent of a severe canine babesiosis (*B. rossi*) in Africa.

The **American dog tick** (*Dermacentor variabilis*) (Fig. 27.13) is a major pest of people and domestic animals throughout much of the eastern and southcentral United States as well as some areas of southeastern Canada. Tick populations generally decline west of the Mississippi River basin, although *D. variabilis* may be locally abundant in some parts of the Midwestern and far western United States. Larvae and nymphs feed on small mammals and birds, but adults attack dogs, other medium-sized mammals, livestock, and humans. Larvae and nymphs are active in late winter and spring, while adults are most abundant in late spring and early summer. This species is the major vector of *R. rickettsii*, the agent of **Rocky Mountain spotted fever**, in the eastern United States. It also transmits the agents of **tularemia** (*Francisella tularensis*) and **anaplasmosis** (*Anaplasma marginale*) and can cause tick paralysis in dogs and humans. In western North America, the closely related **Rocky Mountain wood tick** (*Dermacentor andersoni*) (Fig. 27.14) is an important pest attacking humans, livestock, and wildlife. Adults and nymphs of this tick attack almost any medium-sized or large mammal, whereas larvae feed on small mammals. Adults and nymphs are active in late spring and early summer, while larvae are most abundant in the summer. *Dermacentor andersoni* is the primary vector of *R. rickettsii* and **Colorado tick fever** virus in this region. It also transmits *Anaplasma marginale*, which causes **anaplasmosis** in domestic ruminants. In the Pacific Northwest, *D. andersoni* is an important cause of **tick paralysis**.

The **Pacific Coast tick**, *Dermacentor occidentalis*, has been associated with spotted fever group rickettsiosis in northern California. A rickettsial species, 364D, provisionally designated "*Rickettsia philipii*" found in these ticks, was shown to be the cause of a febrile illness with eschar formation at the bite site (Padgett et al., 2016).

Dermacentor spp. are also important in Eurasia. *Dermacentor reticulatus* is the main vector of the agents of European canine babesiosis (*Babesia canis*) and equine babesiosis (*B. caballi*); it can also transmit the agent of bovine anaplasmosis. *Dermacentor marginatus* is also an important pest of sheep and may be involved in the epidemiology of Q fever.

Dermacentor nitens (until recently, placed in a separate genus, *Anocentor*), the tropical horse tick, is an important pest of livestock in tropical Central and South America and parts of the Caribbean. It typically infests the ears and is a vector of the agent of equine babesiosis/therileriosis (*Theileria equi* or *B. caballi*).

The **blacklegged tick** (*Ixodes scapularis*) (Fig. 27.15A) is widespread throughout large areas of the eastern, southcentral, and midwestern United States. The species appears to be divided into northern and southern clades, which differ genetically and in behavior. The immature stages usually feed on small mammals, lizards, and birds, while adults are most common on white-tailed deer. All stages of the northern clade of *I. scapularis* will bite humans. Nymphal ticks, the stage most likely to transmit **Lyme disease** spirochetes to people, are active in late spring and early summer. Adults are active in the fall and

TABLE 27.3 Representative Tick-Borne Diseases of Veterinary Importance

Disease	Causative Agent	Primary Tick Vector Species	Affected Host(s)
Bovine babesiosis	*Babesia bigemina* *B. bovis*	*R.* (*Boophilus*) *annulatus* *R.* (*B.*) *microplus,* others	Cattle
Canine babesiosis	*B. canis, B. rossi, B. vogeli, B. gibsoni*	*R. sanguineus, Haemaphysalis leachi*	Domestic dogs
East Coast fever	*Theileria parva*	*Rhipicephalus appendiculatus*	Cattle, Cape buffalo
Tropical theileriosis	*T. annulata*	*Hyalomma* spp.	Cattle, water buffalo
Malignant theileriosis	*T. lestoquardi*	*Hyalomma anatolicum*, other tick species	Sheep
Feline cytauxzoonosis	*Cytauxzoon felis*	*Amblyomma americanum, Dermacentor variabilis*	Domestic and wild cats
Louping ill	*Flavivirus*	*Ixodes ricinus*	Sheep, grouse, others
African swine fever	*Iridovirus*	*Ornithodoros porcinus, Ornithodorus erraticus*	Domestic and wild pigs, warthogs
Tick-borne fever	*Anaplasma phagocytophilum*	*I. ricinus, I. scapularis, I. pacificus, I. persulcatus*	Domestic and wild ruminants, horses, dogs, humans
Canine ehrlichiosis	*Ehrlichia canis E. ewingii E. chaffeensis*	*R. sanguineus, I. ricinus A. americanum,* others	Dogs
	E. muris eauclairensis	*Ixodes scapularis*	Dogs
Heartwater	*Ehrlichia ruminantium*	*Amblyomma hebraeum, A. variegatum, Rhipicephalus (B.) microplus*	Ruminants
Bovine ehrlichiosis	*Ehrlichia minasensis* (*Ehrlichia* sp. UFMG-EV)	*Rhipicephalus (B.) microplus*	Cattle
Anaplasmosis	*Anaplasma marginale, A. centrale, A. ovis*	*Dermacentor* spp., *R.* (*Boophilus*) spp., *Hyalomma* spp., *Rhipicephalus* spp.	Cattle, sheep, other ruminants
Borrelioses	*Borrelia burgdorferi*	*Ixodes scapularis, I. ricinus* *I. pacificus, I. persulcatus*	Dogs, cats, cattle, horses, others
Avian spirochetosis	*Borrelia anserina*	*Argas persicus*	Turkeys, chickens, other birds
Epizootic bovine abortion	*Pajaroellobacter abortibovis*	*Ornithodoros coriaceus*	Cattle, deer
Tularemia	*Francisella tularensis*	*D. andersoni, A. americanum, D. variabilis*, others	Sheep, horses, rabbits, game birds
Q fever	*Coxiella burnetii*	Many tick spp.	Most domestic animals
Tick paralysis	Tick proteins	*Ixodes brunneus, I. rubicundus, Rhipicephalus evertsi, D. andersoni D. variabilis, Argas walkerae*	Ruminants, other mammals, dogs, wild birds, chickens,
Tick toxicoses	Tick proteins	*Ornithodoros savigny, O. lahorensis, A. persicus*	Cattle, sheep, birds
Sweating sickness	Tick proteins	*Hyalomma truncatum*	Cattle, sheep, other ruminants, dogs

FIGURE 27.13 The American dog tick (*Dermacentor variabilis*): (A) female, dorsal view, and (B) male dorsal view. *Photograph courtesy of Centers for Disease Control and Prevention.*

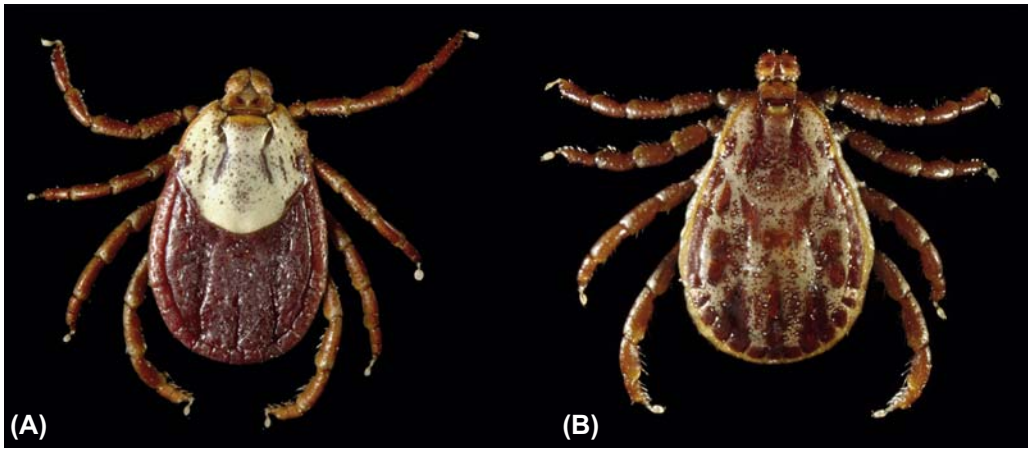


FIGURE 27.14 The Rocky Mountain wood tick (*Dermacentor andersoni*): (A) female, dorsal view, and (B) male dorsal view. *Photograph courtesy of Centers for Disease Control and Prevention.*

FIGURE 27.15 Important *Ixodes* ticks: (A) the blacklegged tick, *Ixodes scapularis*, female, dorsal view, and (B) the western blacklegged tick, *Ixodes pacificus*, female dorsal view. *Photograph courtesy of Centers for Disease Control and Prevention.*

early spring (and winter in southern latitudes). Larvae are most abundant in the summer. *Ixodes scapularis* is the primary vector of the Lyme disease spirochete *Borrelia burgdorferi*, and of *B. miyamotoi*, *B. mayonii*, the protozoan *Babesia microti* that causes **human babesiosis,** and *Anaplasma phagocytophilum*, the agent of **human granulocytic anaplasmosis**. More recently, it has been shown to be an important vector of **Powassan virus** (Flaviviridae), sometimes reported as deer tick virus, which causes a severe and occasionally fatal severe encephalitis in humans (Hermance and Thangamani, 2017). It is the only tick recorded to be infected with *Ehrlichia muris eauclairensis*, an emerging cause of human ehrlichiosis in the upper midwestern United States (Johnson et al., 2015; Karpathy et al., 2016).

There is increasing evidence that *I. scapularis* in the United States consists of at least two genetic populations, which may account for the significant differences in behaviors. It has long been noted that northern populations of *I. scapularis* feed on rodents, particularly white-footed mice, from which they obtain their *B. burgdorferi* infections. Larval and nymphal *I. scapularis* in the southern United States are mainly found on lizards and are not usually encountered through routine methods used to collect northern *I. scapularis*. While it is common to encounter northern nymphs actively seeking hosts on leaves and twigs above the litter surface, host-seeking nymphs in southern *Ixodes scapularis* populations remain below the leaf litter surface and are rarely encountered during their most active seasons. These behavioral differences potentially result in decreased *I. scapularis* collections using flagging or dragging in southern areas, decreased tick contact with humans, and fewer cases of *I. scapularis*-associated diseases. The **western blacklegged tick**, *Ixodes pacificus* (Fig. 27.15B), is an important vector for *B. burgdorferi* in the western United States. The infection prevalence is generally lower than that seen in *I. scapularis*. Adult *I. pacificus* are primarily active in fall and winter.

In Europe, the **castor bean tick,** or **sheep tick** (*I. ricinus*), is a major pest of livestock and humans. This tick ranges from Ireland, Britain, and Scandinavia across continental Europe to Iran and southward to the Mediterranean Sea. In Britain and Ireland, it is commonly found in overgrown sheep pastures that contain dense mats of moist, rotting vegetation ideal for tick development and survival. On the European continent, *I. ricinus* abounds in mixed hardwood-pine forests and shrubs but rarely in grassy meadows. Larvae and nymphs attack mostly small mammals, insectivores, birds, and lizards. Adults are found most commonly on sheep, other domestic ruminants, and deer. However, this tick may attack virtually any vertebrate, including humans. Seasonal activity varies greatly in different regions throughout the tick's range. *Ixodes ricinus* transmits the agents of **Lyme disease**, which, in Europe, include *Borrelia burgdorferi*, *B. garinii*, and *B. afzelii*. In addition, *I. ricinus* is the major vector of the virus which causes **tick-borne encephalitis**, and of *Anaplasma phagocytophilum*. In Ireland, Britain, and some other areas of western Europe, *I. ricinus* also transmits the virus, which causes **louping ill** in sheep, and the bacterium, *Staphylococcus aureus*, which causes **tick pyemia** in sheep. It is also a vector of an agent of human babesiosis and is of importance to livestock as the vector of *B. divergens,* which causes bovine babesiosis. Further east it is replaced by the aggressive **taiga tic***k*, *Ixodes persulcatus*, another major vector of human pathogens.

In Australia, an important species is the **Australian paralysis tick** (*Ixodes holocyclus*). This tick is found along the eastern coast of Queensland and Victoria provinces. It feeds on most wild mammals, domestic animals, and humans. *Ixodes holocyclus* is notorious as the cause of **tick paralysis** in Australia. In contrast to other diseases caused by an infectious microbe, tick paralysis is caused by a proteinaceous material, **holocyclotoxin** in the case of *I. holocyclus*, secreted in the tick's saliva. Even the bite of a single tick may be sufficient to cause a fatal paralysis.

Many species of *Hyalomma* are vectors of *Theileria annulata,* the agent of bovine tropical theileriosis, a major disease of cattle and domestic buffalo in much of Asia, including the Middle East, the Mediterranean basin, parts of southern Europe, and some parts of northern sub-Saharan Africa. The agent of theileriosis virulent to small ruminants, *Theileria recondita,* is also transmitted by *Hyalomma* spp. Important vectors include *H. detritum*, *H. anatolicum*, *H. asiaticum*, and *H. lusitanicum*. In the Mediterranean basin and parts of the former Soviet Union (the Crimea and adjacent areas of the former USSR), an important tick is *Hyalomma marginatum marginatum*. Larvae and nymphs attack hares, hedgehogs, and birds. Adults attack larger mammals, including domestic ruminants and humans. This tick is one of the most important vectors of **Crimean-Congo hemorrhagic fever** virus. In the adult stage, *Hyalomma* spp. infest particularly the perianal area, the perineum, or the tail switch, where they escape visual detection.

The **lone star tick** (*Amblyomma americanum*) (Fig. 27.16) is one of the most notorious tick pest species in the United States. It is found along the Atlantic coast from New York to Florida and west into Texas and Oklahoma. *Amblyomma americanum* larvae, nymphs, and adults readily attack humans and companion animals, as well as livestock and wildlife. Virtually any mammal or ground-feeding bird may be infested. It is often abundant in areas with large populations of deer, which serve as the primary hosts for the adult ticks. In the southeastern United States, nymphs and adults emerge from their winter diapause and commence host-seeking activity in late spring. Larvae generally appear in late summer. Seasonal

FIGURE 27.16 The lone star tick (*Amblyomma americanum*): (left to right) female, dorsal view; male dorsal view; nymph, dorsal view; and larva, dorsal view. *Photograph courtesy of Centers for Disease Control and Prevention.*

activity may be delayed farther north. *Amblyomma americanum* has been implicated as a vector of the agents that cause **human ehrlichiosis**, that is, *Ehrlichia chaffeensis* and *E. ewingii*, as well as tulararemia (*Francisella tularensis*), *R. rickettsii*, and an endosymbiotic spotted fever group rickettsial species, *R. amblyommatis* (previously "*Candidatus* R. amblyommii").

Another important species in the United States is the **Gulf Coast tick** (*Amblyomma maculatum*) (Fig. 27.17), which is found in the southeastern and southcentral United States and Central America. Larvae and nymphs attack a wide range of birds and small mammals, but adults feed largely on ruminants. These ticks feed mainly on the head and ears. *Amblyomma maculatum* can cause severe injury to the skin of cattle and other livestock, often rendering the hides useless from the bites of these ticks or from secondary infections and predisposing to screwworm and severe dermatophilosis. It is an efficient experimental vector of *Ehrlichia ruminantium* that causes heartwater, a major African disease of ruminants, which has been imported into the Caribbean area. *Amblyomma maculatum* group ticks transmit *Rickettsia parkeri* to humans in the eastern and in the southwestern United States (Allerdice et al., 2017).

In Africa, the **bont tick** (*Amblyomma hebraeum*) (Fig. 27.18) and the **tropical bont tick** (*A. variegatum*) attack livestock as well as wild ruminants. In addition, *A. variegatum* larvae and nymphs will parasitize ground-feeding birds, including herons and other migratory birds, and small mammals. *Amblyomma hebraeum* is restricted to southern Africa, but *A. variegatum* ranges throughout most of sub-Saharan Africa, Madagascar and several islands in

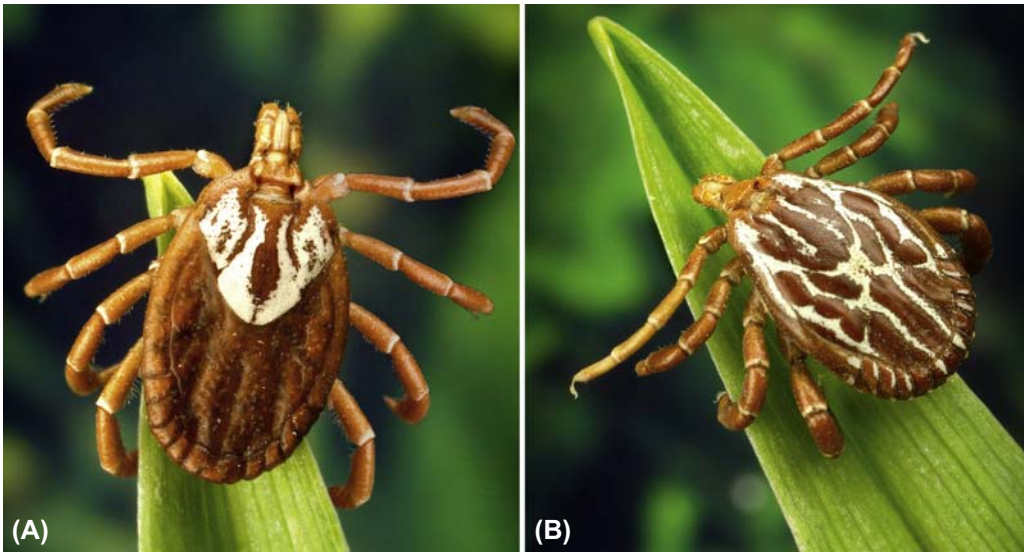


FIGURE 27.17 The Gulf Coast tick (*Amblyomma maculatum*): (A) female, dorsal view, and (B) male dorsal view. *Photograph courtesy of James Gathany, Centers for Disease Control and Prevention.*

FIGURE 27.18 The bont tick (*Amblyomma hebraeum*): (A) female, dorsal view, and (B) male dorsal view. *Photograph courtesy of Centers for Disease Control and Prevention.*

the Caribbean. These ticks are the major vectors of *Ehrlichia* (formerly *Cowdria*) *ruminantium,* which causes **heartwater** in ruminants, whereas *A. variegatum* is associated with severe forms of ruminant **dermatophilosis** (see later). They are also vectors of *Rickettsia africae,* the causative agent of an African human disease, which has been inadvertently introduced into the Caribbean.

Arguably the most important livestock tick on a global scale is the one-host **southern cattle fever tick**, *Rhipicephalus* (*Boophilus*) *microplus*. This species, and other members of the subgenus *Boophilus*, can cause large infestations in cattle and other ungulates (Fig. 27.19). The southern cattle fever tick originated in southern Asia but is now also established in Australia, the Pacific area, Mexico, Central and tropical South America, the Antilles, Madagascar, and large parts of eastern and southern Africa, where it has replaced the indigenous tick *Rhipicephalus* (*B.*) *decoloratus*. When present in large numbers, it may cause retarded growth and weight loss. It is even more important as the main vector of *Babesia bovis* and *B. bigemina*, agents of bovine babesiosis, and of *Anaplasma marginale,* which causes anaplasmosis. Other important species are *R. decoloratus* (although it is not a vector of *Babesia bovis*), present in much of sub-Saharan Africa, and *Rhipicephalus* (*B.*) *geigyi*, which has replaced it in West Africa. Another species of major importance is the **cattle fever tick,** *Rhipicephalus* (*B.*) *annulatus*, ranging throughout large areas of North and sub-Saharan Africa north of the equator, parts of southern Europe and western Asia, and parts of North America, Central America, and South America. Only intensive surveillance has prevented its reintroduction, as well as that of *R. microplus*, into the United States from tick-infested herds in Mexico. *Rhipicephalus* (*B.*) *annulatus* is active throughout the year in the tropics. This one-host tick feeds almost exclusively on cattle, but it also infests white-tailed and other species of deer. It is a major pest of cattle, causing reduced weight gains and milk production in heavily infested animals. It is best known for its role in the transmission of the protozoan *Babesia bigemina*, which causes **Texas cattle fever**, and as a vector of *B. bovis* and *Anaplasma marginale.*

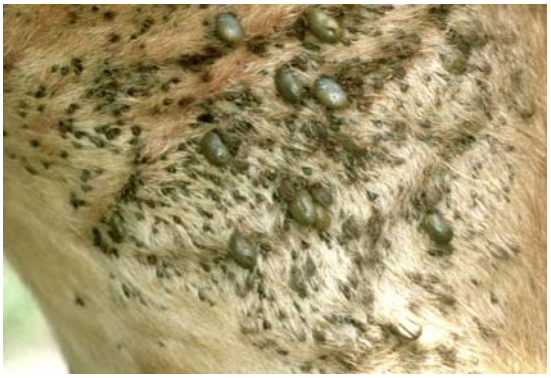


FIGURE 27.19 White-tailed deer heavily infested with ticks, *Rhipicephalus (Boophilus)* sp. *Courtesy of U.S. Department of Agriculture, Animal Research Service, Kerrville, TX.*

Among the Argasidae, the **fowl tick** (*Argas persicus*) and related *Argas* spp. are important parasites of poultry in the Old World. All life stages feed on these birds. Populations of this tick can reach enormous numbers in poultry barns and can cause high mortality due to exsanguination. This tick is the vector of the rickettsia *Aegyptianella pullorum* that causes **fowl disease** in domestic fowl. In the Mediterranean region, it is a vector of *Borrelia anserina*, the agent of **fowl spirochetosis**, an important poultry disease. In the New World, a complex of three species (*A. radiatus, A. sanchezi*, and *A. miniatus*) supplements the introduced and now established (yet rare)

A. persicus. The Old World diseases are also now known in the New World.

The genus *Ornithodoros* includes several species that live in animal burrows and poorly maintained homes or shelters, where they hide in cracks and crevices of walls, ceilings, and attics. In the western United States, *O. hermsi* often infests mountain cabins and other dwellings. Although rodents that infest dwellings are the principal hosts of *O. hermsi*, humans may be attacked when they enter such dwellings if rodents have been killed or driven out. This tick is notorious as a vector of the **relapsing fever** spirochete *Borrelia hermsii*.

In eastern and southern Africa, the human-biting **African tampan** (*Ornithodoros moubata*) and related species, such as *O. porcinus*, coexist with people and animals in mud huts where the tick hides in the walls. These species are the major vectors of the **relapsing fever** spirochete *Borrelia duttoni*. *Ornithodoros porcinus* may be involved in maintaining the virus of **African swine fever,** cycling between warthogs and ticks. The virus becomes directly contagious when it spreads to domestic pigs, and has subsequently been temporarily introduced to other continents. African swine fever is of major international importance. It has been difficult to eradicate from Spain and Portugal, where it has become established in a local species, *Ornithodoros erraticus*. O*rnithodoros porcinus* also occurs on Madagascar. Another species, *O. sonrai*, is thought to be implicated in the epidemiology of African swine fever in West Africa.

Ornithodoros savignyi is a major pest of camels, other domestic animals, and humans in the drier parts of Africa and southern Asia. Although it is not known to transmit pathogenic organisms, it often occurs in large numbers in the sand of sites where animals and humans congregate (e.g., resting sites, wells, etc.) and may be responsible for loss of blood and bites, which remain painful and itching for long periods.

Another important argasid in western North America is the **spinose ear tick** (*Otobius megnini*). It has become established in India, Madagascar, Kenya, and Turkey. It frequently infests livestock, especially cattle and horses, and most domestic ruminants. *Otobius megnini* also attacks wild ruminants, especially deer, antelope, mountain sheep, and may even bite humans. The larvae and second-stage nymphs feed, whereas the adults do not. This tick feeds in the ears, causing injury to the auditory canal (the nymphs are covered with spines) and secondary infections.

In the central and eastern United States, the bat tick *Carios kelleyi* (Fig. 27.5) has been shown to feed occasionally on human blood and to attack people in bat-infested houses. An erythematous skin rash, presumably due to a reaction to the bite, may occur (Gill et al., 2004).

PUBLIC HEALTH IMPORTANCE

Ticks are of public health significance mainly because of the zoonotic animal disease agents transmitted by them, which include an increasing array of bacterial, viral, and protozoan disease agents (Harwood and James, 1979; Sonenshine, 1993; Goodman et al., 2005). They also are important because their attachments can cause various kinds of dermatoses or skin disorders, such as inflammation, pain, and swelling. Rarely, they invade the auditory canal producing a condition known as **otoacariasis**. Certain species of ticks may cause a flaccid, ascending and sometimes fatal paralysis known as **tick paralysis**. Individuals bitten repeatedly by some ticks may develop allergic or even anaphylactic reactions (Van Wye et al., 1991).

Among the biological factors that contribute to the high vector potential of ticks are their persistent blood-sucking habit, longevity, high reproductive potential, relative freedom from natural enemies, and highly sclerotized bodies that protect them from environmental stresses. Further, the slow feeding behavior of ixodid ticks permits wide dispersal and increases their likelihood of acquiring pathogens during attachment to a host. **Transstadial passage** of microbial disease agents from larva to nymph or nymph to adult commonly occurs in vector ticks; transovarial transmission of many agents occurs in some ticks; and both phenomena contribute to the maintenance and spread of certain tick-borne agents.

Several other biological attributes of ticks also enhance their vector potential. First, pharmacologically active substances present in the saliva of ticks may promote feeding success and aid transmission of microbial agents. For example, the saliva of *I. scapularis* has antiedema, antihemostatic, and immunosuppressive properties. Second, ticks imbibe large quantities of blood during each feeding period. Indeed, certain species may increase their body weight by 100-fold or more. This is actually an underestimate of the amount ingested because feeding ticks concentrate the bloodmeal by secreting copious amounts of host-derived fluid back into the host. Third, ticks take multiple bloodmeals during their lifetimes. Those individuals that attain adulthood and that successfully feed as adults feed three (ixodids) or more (argasids) times.

It should be noted that ticks are far more efficient than insects in maintaining microbial agents in their bodies. In ticks, most internal tissues change gradually during development and transstadial survival of pathogens occurs frequently. In holometabolous insects, however, the extensive internal changes that occur during molting seem to have a harmful effect on most microorganisms that cause human disease.

As reviewed by Lane (1994) and Nuttall and Labuda (1994), ticks transmit microbes via several routes,

including salivary secretions (e.g., Lyme disease spirochete, Colorado tick fever virus, the agent of heartwater, and spotted fever group rickettsiae), coxal fluids (certain species of relapsing fever spirochetes), regurgitation (e.g., possibly the spirochetes that cause Lyme disease), and feces (Q fever organisms). A novel type of transmission, **saliva-activated transmission**, occurs in the case of some tick-borne arboviruses (Jones et al., 1992). In this model, one or more proteins secreted in tick saliva potentiate virus transmission. Moreover, this phenomenon seems to be the mechanism underlying "**nonviremic transmission**," whereby arboviruses are transmitted from infected to uninfected ticks feeding simultaneously on a vertebrate host having no or very low levels of viremia (Nuttall and Jones, 1991; Nuttall and Labuda, 1994). Transmission between co-feeding infected and uninfected ticks, which also has been demonstrated for the Lyme disease spirochete, *Borrelia burgdorferi*, is important epidemiologically for two reasons (Randolph et al., 1996). First, some vertebrates that do not develop systemic infections still can serve as competent hosts for infecting vector ticks, and, second, it adds yet another transmission route for certain tick-borne pathogens. Although some tick-borne agents may be transmitted via two routes (e.g., transmission of certain relapsing fever spirochetes via coxal fluid secretions and by saliva), only one route is usually significant.

The more important tick-borne diseases of public health concern are summarized in Table 27.2. The causative agent, clinical manifestations, ecology, and epidemiology of each of these diseases are discussed next.

Human Babesiosis

Human babesiosis is an emerging disease caused by several species of protozoans in the genus *Babesia.* This genus also contains species of major veterinary importance, as do the related protozoan genera *Cytauxzoon* and *Theileria*. Species in all three genera belong to the family Babesiidae, order Piroplasmorida, and phylum Apicomplexa. They are often referred to as **piroplasms** because they possess pear-shaped, intraerythrocytic merozoites in the vertebrate host. *Babesia* spp. resemble malarial parasites (*Plasmodium* spp.) and other blood-infecting protozoans, especially in regard to their developmental cycles. More information about these important parasites can be found in Schetters and Brown (2006), Suarez and Noh (2011), Beugnet and Moreau (2015), and Solano-Gallego and Sainz (2016). Wilson and Chowning in 1908 were the first to incriminate babesial parasites as a probable cause of human infection among patients with Rocky Mountain spotted fever in the western United States. However, the first definitive case of babesiosis was not described until 1957 in a splenectomized Yugoslavian cattle farmer who died of a babesial infection following an 8-day illness. In the United States, the disease was initially recognized in a California resident in 1968. To date, less than 100 cases of human babesiosis have been reported from Europe, while cases in the United States have reached over 1,700 cases annually. Sporadic cases occur elsewhere. In the United States, human babesiosis occurs principally along the Eastern Seaboard, especially on Nantucket Island, Massachusetts, and Long Island, New York, where the etiologic agent is *Babesia microti*. Other endemic foci of *B. microti* occur in Connecticut, Minnesota, and Wisconsin. The incidence appears to be increasing in Wisconsin, where 72% of the 32 cases reported from 1996 to 2005 occurred in 2004–2005. In the far-western United States, *Babesia duncani* has been identified in nine patients since 1991 (Conrad et al., 2006). Besides the index case, four of the patients previously had their spleen removed (one died), and two each were blood donors or blood recipients. This piroplasm, formerly designated as the WA1-type *Babesia* in the literature, lies in a distinct clade separable from *Babesia* sensu stricto, *B. microti*, and *Theileria* spp.

At least 70% of the cases in Europe are associated with the cattle piroplasm *B. divergens* (Genchi, 2007). Intriguingly, *Babesia* parasites similar to, but not identical with, *B. divergens* have been detected in three asplenic men in Missouri (1992), Kentucky (2001), and Washington State (2002) (Herwaldt et al., 2004). Serosurveys suggest that a low percentage of Europeans ($\leq$3.4%) from several countries may be infected with *B. microti*, particularly individuals who engage in higher-risk outdoor activities like forestry. A newer *Babesia* species, *Babesia* sp. EU1, first described in 2003 based on isolates obtained from two asplenic men in Austria and Italy, is an emerging zoonosis (Herwaldt et al., 2003). Phylogenetically, this organism is most closely related to *B. odocoilei*, a parasite of white-tailed deer in North America.

In humans, *Babesia* spp. may produce a malarial-like disease without the periodicity that often accompanies the human malarias. Following an incubation period of 1–4 weeks, the clinical course varies according to the etiologic agent and ranges from subclinical infection to a severe disease with sudden onset. Splenectomized persons infected with either *B. divergens* or *B. microti*, or elderly persons infected with *B. microti*, tend to develop severe or sometimes fatal illnesses. Signs and symptoms at onset include fever, chills, profuse sweating, headache, and generalized muscle aches. Joint pain, nausea, vomiting, and prostration may occur. Parasitemia and the resultant clinical course may persist for several months with severe anemia, jaundice, and hemoglobinuria. In many individuals, however, babesiosis is a mild, self-limited disease that requires only supportive therapy. Clindamycin and quinine in combination are the current drugs of choice, but azithromycin and atovaquone in combination are equally

effective in treating babesiosis patients with fewer adverse effects.

With few exceptions, *Babesia* species develop entirely within circulating red blood cells. Sporozoites are introduced via the saliva of a *Babesia*-infected tick during feeding. Once they gain entry into the bloodstream, most parasites develop asexually within red blood cells. Occasionally, *Babesia* invade lymphocytes, and only subsequent generations develop in the erythrocytes. Within the host cell, the parasites develop into **trophozoites** termed **meronts**, which multiply asexually by binary fission to produce **merozoites**. Some of the merozoites escape from the disintegrating host cells to invade other erythrocytes and continue the cycle. Other merozoites develop into gametocytes called **piroplasms** after entering previously uninfected erythrocytes. The gametocytes remain in an arrested state of development until they are ingested by a feeding tick.

When *Babesia*-infected blood is ingested by ticks, the gametocytes commence development, but the asexual stages are destroyed. The gametocytes escape from the dying host cells and transform into gamete-forming cells called gamonts, which develop structures (rays and spines) that are subsequently used to penetrate cells. Following gametic fusion, the resulting zygotes invade the tick's digestive epithelium, develop into motile **kinetes**, and migrate to other internal organs. Some *Babesia* spp., such as *B. divergens* and *Babesia* sp. EU1, invade the female tick's ovaries and are transmitted transovarially to the next generation, whereas others (e.g., *B. microti*) are not passed via the eggs. Instead, immature ticks are infected while feeding on a parasitemic host; the parasites invade the salivary glands, multiply, and are passed transstadially to the next stage.

In the northeastern and upper midwestern United States, *B. microti* is maintained in a transmission cycle involving the blacklegged tick (*I. scapularis*) and the white-footed mouse (*Peromyscus leucopus*). Meadow voles (*Microtus pennsylvanicus*) also are efficient reservoir hosts. Most people who acquire the infection are bitten by nymphal ticks. In the far-western United States the primary tick vector(s) and reservoir host(s) of *B. duncani* have not been identified, but the close similarity of babesial isolates from mule deer with *B. duncani* isolates from humans suggests that large ungulates might serve as reservoirs. In Europe, *I. ricinus* is the primary vector of both *B. divergens* and *Babesia* sp. EU1, whereas *Ixodes trianguliceps* transmits *B. microti* among small mammals (Randolph, 1991, 1994, 1995). The apparent paucity of human *B. microti* infections in Europe may be attributable to the fact that *I. trianguliceps* is a nidicolous tick that seldom attaches to people.

Babesia spp. may be transmitted via two other routes besides tick-feeding: blood transfusion and transplacentally. More than 50 cases of transfusion-associated *B. microti* infections have been reported in North America, and 10 cases of neonatal babesiosis have been published. *Babesia* ranks second only to *Plasmodium* among blood transfusion-acquired parasitic infections. Estimates of the percentages of *Babesia*-infected blood products in certain endemic settings in the United States (e.g., Connecticut, Massachusetts) range between 0.17% and 3.7%.

Tick-Borne Encephalitis Complex

Tick-borne encephalitis complex (TBE) is one of at least 12 related, but distinguishable, serotypes of tick-borne flaviviruses (Family Flaviviridae) that constitute the **TBE complex.** It includes such viruses as **Louping ill**, **Kyasanur Forest disease**, **Omsk hemorrhagic fever**, and **Powassan encephalitis**. Each of these viruses produces a clinically distinctive disease.

First described as **Russian spring-summer encephalitis** (RSSE) in 1932 from the far-eastern region of the former Soviet Union, TBE was recognized after World War II in central Europe, where it was termed central-European encephalitis (CEE). RSSE and CEE are now considered to represent a single entity, TBE, which has been classified into three subtypes (European, Siberian, and far-eastern) that vary in virulence for humans. In 2007, a novel variant of the far-eastern subtype was isolated from the brain of a 15-year-old boy in Primorsky District, Russia, who succumbed to the infection. Grard et al. (2007) proposed that TBE viruses be divided into four types: Western, Eastern, Turkish sheep, and Louping ill.

TBE is endemic in nearly 30 areas of Europe and northern Asian countries, and the incidence is estimated to be as high as 14,000 cases per year, with about 11,000 of them occurring in Russia. However, the number of risk areas across Europe and parts of Asia have increased (Petri et al., 2010). Few arthropod-borne zoonotic agents have received as much scientific scrutiny. In Russia, for instance, researchers published approximately 5,000−6,000 articles and 40−50 monographs on various aspects of TBE during the first 60 years following its discovery (Korenberg and Kovalevskii, 1999). In that regard, it has been estimated that 20,000−30,000 autonomous natural foci, ranging in size from a few square kilometers to several hundred kilometers, exist in Russia.

Illness in humans is accompanied by high, often biphasic, fever and headache, followed soon afterward by inflammation of the brain (encephalitis) and meninges (meningitis). Some patients develop muscle weakness or paralysis, especially in the right shoulder muscles.

Case-fatality rates average about 1%−2% for European strains, 20%−60% for far-eastern strains, and rarely exceed 6%−8% for Siberian strains (Charrel et al., 2004). Although Siberian strains are less lethal than far-eastern strains, they nevertheless tend to cause chronic or prolonged infections.

Fortunately, there are several highly effective, safe, and well-tolerated vaccines commercially available against TBE viruses in Europe and Russia (Petri et al., 2010). The two highly purified, formalin-inactivated, whole-virus vaccines developed in Europe have an overall efficacy of 99% when used in accordance with the recommended vaccination schedule. Notwithstanding, the number of reported cases of TBE increased an astounding 400% in Europe between 1974 and 2003 due to the complex interaction of ecological, economic, social, political, and climatic factors (Kunze et al., 2007). A notable exception has been Austria, which has experienced a dramatic decline in clinical cases as a result of increased vaccination coverage, from approximately 6% in 1980 to 88% of the entire population in 2006. The widespread use of vaccines in Austria from 2000 to 2006 is estimated to have prevented approximately 2,800 cases and 20 deaths from TBE (Heinz et al., 2007). In stark contrast, vaccination coverage in TBE-endemic countries bordering Austria is meager: 11% in the Czech Republic and 13% each in Germany and Switzerland.

Climatic changes may contribute to the geographic expansion or resurgence of some vector-borne diseases. They alone, however, cannot explain the recent upsurge in the incidence of TBE or the pronounced spatiotemporal heterogeneity of the virus in Central Europe and the Baltic Region (Rogers and Randolph, 2006; Randolph and Sumilo, 2007). Anthropogenic impacts on the landscape have allowed tick populations to expand and multiply; changes in human behavior may have resulted in a greater degree of contact with virus-laden ticks; and migrating birds can disperse TBE virus-infected *I. ricinus* ticks (Randolph, 2001; Waldenström et al., 2007). In Estonia, Latvia, and Lithuania, environmental changes resulting from political upheaval and socioeconomic transitional factors following the end of Soviet rule that presumably elevated human contact with infected ticks have been posited to play an important role in the increased incidence of TBE. Among Latvians, harvesting mushrooms and berries, or working in forests has been associated with unemployment, lower incomes, increased forest visitation, and a higher than average risk of being tick-bitten the previous year (Randolph and Sumilo, 2007). Weather conditions also may influence the frequency of forest visits and therefore the degree of tick exposure in this region.

The primary vectors of TBE viruses are *Ixodes ricinus* (European subtype) and *I. persulcatus* (Siberian and far-eastern subtypes). Other tick species that have been found infected naturally (*Ixodes arboricola, I. hexagonus, I. trianguliceps*) may amplify viral infection. Although most mammals are susceptible to TBE virus, rodents, especially the bank vole (*Clethrionomys glareolus*), field mice (*Apodemus* spp.), and insectivores are the chief reservoir hosts. Viral amplification and enzootic maintenance occur by means of the seasonally synchronized co-feeding of virus-infected nymphs and large numbers of uninfected larvae during brief periods (2−3 days) of nonviremic infectivity within primary vertebrate hosts (Randolph et al., 1999; Randolph and Sumilo, 2007). Thus, transstadially infected nymphs transmit the virus horizontally to uninfected larvae, which molt up to a year later to produce infected nymphs. This nonviremic route of transmission between co-feeding ticks can even occur in rodents that are immune to TBE virus (Labuda et al., 1997). The principal environmental driver for synchronizing the springtime feeding of larvae and nymphs in TBE foci initially was thought to be a rapid rate of cooling in autumn, corrected for mid-summer maximum temperatures (Randolph et al., 2000). More recent evidence suggests that the rate of spring warming, corrected by January minimum temperatures, is more important in synchronizing larval and nymphal feeding activities than is the rate of autumnal cooling (Randolph and Sumilio, 2007).

Kyasanur Forest disease (Family Flaviviridae, Genus *Flavivirus*) was identified in 1957 from a sick monkey in Kyasanur Forest in Karnataka state, India (Holbrook, 2012). The virus is transmitted by ixodid ticks, especially *Haemaphysalis spinigera*. Once infected, the tick remains infected for life and can transmit the virus both transstadially and transovarially. Mammalian hosts for the tick and virus are rodents, shrews, and monkeys. Infection may cause epizootics with high fatality rates in primates. Exposure to sick or dead animals may also be a risk factor for human infection; person-to-person transmission is not known to occur. After an incubation period of 3−8 days, chills, fever, and headache occur. Severe muscle pain, gastrointestinal distress, vomituing, and bleeding may occur 3−4 days after initial symptoms. Patients may recover after one to weeks of illness, but a subset of patients may have additional fever, severe headache, mental disturbances, tremors, and vision deficits. The estimated case-fatality rate ranges from 3% to 5%. The disease is found in southern India, with about 500 cases per year. More recently, cases have been identified from additional states in the south, west, and east of the country. Similar viruses have been discovered in Saudi Arabia (Alkhurma hemorrhagic fever virus) and China (Nanjianyin virus).

Powassan virus, named after the town in Ontario, Canada, where it was originally isolated from a 5-year-old boy who succumbed to the infection, is a *Flavivirus* (family

Flaviviridae) related to TBE viruses. It was first recognized in scattered localities in the United States, Canada, and in eastern parts of the former Soviet Union. The virus causes a disease known as **Powassan encephalitis** that is characterized in its acute stage by encephalitis, severe headache, and fever. Nausea, labored breathing, and neurologic disorders, including partial paralysis, occur frequently. As many as 50% of recovered patients may suffer permanent nerve damage due to neuronal loss and necrosis and more severe infections can have a case-fatality rate of about 10%. Thirty-one cases were reported from the northeastern United States and Canada from 1958 to July 2001, and incidence has been increasing to approximately 1.9 cases per year between 1999 and 2007 (Ebel, 2010). Cases also have been recorded in Russia (Charrel et al., 2004).

Tick vectors of Powassan virus belong to the genera *Ixodes, Dermacentor,* and *Haemaphysalis*. In the United States, isolates of the virus have been obtained from *Ixodes cookei* and *Ixodes marxi* in the east and *Dermacentor andersoni* and *Ixodes spinipalpis* in the west. *Ixodes cookei* feeds on various wild and domestic animals and (rarely) on humans. Marmots (woodchucks) are important hosts of *I. cookei* and are excellent reservoirs of the virus. Similarly, the snowshoe hare amplifies populations of vector ticks and the virus. The virus has been isolated twice from naturally infected foxes, a red squirrel, a white-footed mouse, and a spotted skunk, but the reservoir competence of these species remains to be determined. Antibodies to the virus have been detected in 38 wild and five domestic mammalian species. *Dermacentor andersoni* is the most important vector in the western United States and Canada. In the former Soviet Union, the virus has been isolated from *Haemaphysalis neumanni, I. persulcatus*, and *Dermacentor silvarum* and from mosquitoes. *Apodemus* spp. mice and *Microtus* spp. voles are the primary vertebrate hosts in the Eastern Hemisphere (Charrel et al., 2004).

In the northeastern United States, a genotype of Powassan virus (known as lineage II) identified during the 1990s is also called **deer tick virus** (DTV). This lineage is maintained in white-footed mice in the northeastern and upper midwestern United States and is transmitted by *Ixodes scapularis*. In the laboratory, 90% of *I. scapularis* larvae acquired DTV from needle-inoculated mice; the efficiency of transstadial passage was 22%; and the resultant nymphs transmitted the infection to naïve mice after having been attached for as few as 15 min. Clinical disease in humans attributable to DTV was reported in 2001. Clinical features included fever, fatigue, double vision, and weakness, with progressive neurological involvement. Two deaths have been reported in the literature (Ebel, 2010).

Colorado Tick Fever

Colorado tick fever (CTF) is caused by a *Coltivirus* in the family Reoviridae (Fig. 27.20A). Coltiviruses were formerly divided into two subgroups, A and B, based on their genetic relatedness. North American and European

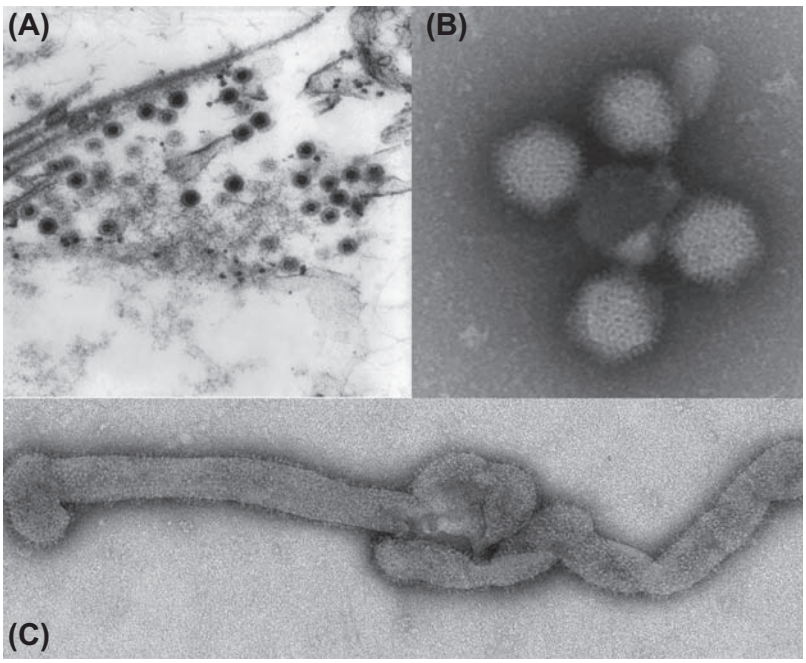


FIGURE 27.20 Electron micrographs of some tick-borne viruses: (A) Colorado tick fever virus, genus *Coltivirus*; (B) Heartland virus, genus *Phlebovirus*; and (C) Bourbon virus, genus *Thogotovirus*. *Courtesy of Centers for Disease Control and Prevention.*

species were placed in subgroup A, and Asian species in subgroup B (Marfin and Campbell, 2005). In 2000, researchers proposed that subgroup B coltiviruses were sufficiently distinct to warrant inclusion in a separate genus, *Seadornavirus*. Subgroup A coltiviruses currently comprise four antigenically related viruses: CTF virus in western North America; Salmon River virus in Idaho, USA; **Eyach virus** in the Czech Republic, France, and Germany; and "California hare coltivirus" in the United States (Attoui et al., 2005). The latter virus was isolated first from a western gray squirrel in 1965, and a similar if not identical virus was isolated 11 years later from a black-tailed jack rabbit (Lane et al., 1982). Notably, these isolates originated in either northwestern or westcentral California, far outside the distributional ranges of the primary mammalian hosts of CTF virus and of the Rocky Mountain wood tick (*Dermacentor andersoni*), the primary bridge vector to humans. Furthermore, this virus is the only one of the subgroup A coltiviruses that has not been associated definitively with human illness.

Symptoms of CTF usually appear within 4 days (range, 1−14 days) following the attachment of an infected tick. The disease is characterized by a biphasic fever, chills, headache, generalized musculoskeletal aches, and malaise. Some patients experience eye pain, intolerance of light, chills, sore throat, and nausea. The virus develops in most internal organs and may spread to the brain or bone marrow. Although CTF sometimes is depicted as a mild febrile illness, acutely ill patients usually are bedridden, and as many as 14% require hospitalization (Marfin and Campbell, 2005). Convalescence may be prolonged, with some patients taking several weeks to recover. Case-fatality rates are very low, usually less than 0.2%, and all reported deaths have involved children. Early in the course of disease, CTF may be mistaken for Rocky Mountain spotted fever because up to 12% of CTF patients develop a maculopapular or petechial rash.

In endemic regions, people engaged in outdoor activities in mountainous or highland areas from about 4,000 ft to over 10,000 ft (1,219−3,048 m) are at risk of exposure to virus-infected ticks. In Rocky Mountain National Park, Colorado (USA), natural foci occur on south-facing slopes covered with open stands of pine and shrubs on dry, rocky surfaces. Cases are reported from March to November, but most occur in the spring and early summer when adult and nymphal ticks are active. The distribution of CTF approximates that of *D. andersoni* in western North America. The virus has been isolated from ticks, humans, or both from parts of the United States and Canada. In the United States, 476 (61%) of 777 cases reported to 12 state health departments between 1987 and 2001 were contracted in Colorado, with Utah (n = 122) and Montana (n = 106) ranking second and third (Marfin and Campbell, 2005). Risk factors for the disease include being male, 10−49 years of age, and occupational or recreational exposure at higher elevations (1,200−3,000 m) in the Rocky Mountains or other endemic mountainous areas of the western United States. Transfusion-associated CTF has been reported.

CTF virus is passed efficiently from stage-to-stage in *D. andersoni* ticks, but transovarial passage does not occur. Therefore, the virus is maintained horizontally as host-seeking nymphs, previously infected while feeding as larvae on viremic hosts, attach to and infect susceptible small mammalian hosts. The virus has been isolated from *D. albipictus, D. occidentalis, D. parumapertus, Haemaphysalis leporispalustris, Ixodes sculptus, I. spinipalpis,* and *Otobius lagophilus.* Larvae and nymphs of *D. andersoni* feed on small mammals, especially ground squirrels, mice, and rabbits. Nymphs, which quest higher in vegetation than larvae, also attack larger mammals such as small carnivores and occasionally humans. Important hosts of the immatures include golden-mantled ground squirrels, deer mice, bushy-tailed woodrats, chipmunks, and rabbits. Adults parasitize larger mammals, such as porcupines, elk, deer, antelope, carnivores, and humans. Competent reservoir hosts include the golden-mantled ground squirrel, least chipmunk, deer mouse, bushy-tailed woodrat, and porcupine. Viremia in amplifying hosts may persist for weeks or months, and possibly even longer in hibernating mammals. In the latter case, overwintering hosts may serve as a source of infection for uninfected immature ticks the following spring (Marfin and Campbell, 2005).

Eyach virus, which was isolated for the first time from *I. ricinus* ticks in Germany in 1976, is antigenically related to, but distinct from, CTF virus (Charrel et al., 2004). Additional strains of the virus were isolated from *I. ricinus* and *I. ventalloi* ticks in France in 1981. Serologic surveys demonstrated that Eyach virus occasionally infects people in France and the former Czechoslovakia and may cause encephalitis and polyradiculoneuritis in some patients. The transmission cycle has not been defined, but the primary reservoir is believed to be the European rabbit (Charrel et al., 2004).

Crimean-Congo Hemorrhagic Fever

Crimean-Congo hemorrhagic fever (CCHF) is caused by a negative-stranded RNA virus in the genus *Nairovirus*, Family Bunyaviridae (Bente et al., 2013; ICTV, 2017). The disease was first identified in 1944 and later in 1969, as the two locations led to the hyphenated geographical name. *Hyalomma* spp., especially *H. marginatum,* serve as

reservoirs and vectors for the CCHF virus. Ticks maintain the virus transstadially and transovarially. Additional ixodid tick species within the genera *Amblyomma*, *Dermacentor*, *Haemaphysalis*, *Ixodes*, and *Rhipicephalus* have been found infected naturally or experimentally, but *Hyalomma* spp. ticks are the main vectors to humans. Domestic livestock can serve as amplifying hosts, while hares and hedgehogs may serve as wildlife hosts for immature ticks. Interestingly, there is no evidence that the virus causes illness in non-human animals. The disease is found in about 30 countries in eastern and southern Europe, the Mediterranean region, China, central Asia, the Middle East, India, and Africa. More recently, large numbers of cases have been reported from Turkey. People at risk include livestock workers, animal handlers, and slaughterhouse workers, and case reporting has increased. Healthcare workers in endemic areas can also become infected through contact with body fluids. Clinical illness includes headache, high fever, back pain, joint pain, stomach pain and vomiting. Changes in mood and sensory perception may be seen in more severe cases. Petechiae in the palate, severe bruising, uncontrolled bleeding and nosebleed may begin on about the fourth day of illness and last for up to 2 weeks. Case fatality rates of 9%−50% have been observed in hospitalized patients. Treatment is mainly supportive, although ribavirin may provide additional benefit.

Severe Fever and Thrombocytopenia Syndrome Virus

Severe fever and thrombocytopenia syndrome virus (SFTSV) was first identified as a cause of severe illness and death in China in 2007 (Mansfield et al., 2017). The virus is a member of the genus *Phlebovirus*, Family Bunyaviridae (ICTV, 2017). Isolation of the virus from patients confirmed the etiology in patients and additional cases began to accumulate. Clinical signs include fever, vomiting, diarrhea, thrombocytopenia, leukopenia, and multiple organ failure. The case-fatality of SFTSV infections has been estimated to be 6%−30% (Liu et al., 2014). Since its first recognition, the disease has been identified in other provinces of China, Japan, and South Korea. Ecological studies have shown that *Haemaphysalis longicornis* is the primary vector, with infection rates of 2%−5%. Goats, cattle, dogs, pigs, and chickens are naturally exposed to the virus, but goats and other livestock may be important amplifying hosts contributing to human epidemiology. Many wildlife species have shown serological evidence of exposure, and certain rodents have been found to be infected.

Heartland Virus

Heartland virus was first isolated from two male patients in northwestern Missouri (McMullan et al., 2010). This virus is also in the genus *Phlebovirus*, Family Bunyaviridae (Fig. 27.20B). They had been bitten by lone star ticks, *Amblyomma americanum*, from which the virus was subsequently isolated (Savage et al., 2013, 2017). Fever, headache, fatigue, and mental stupor were seen in both patients. Both men recovered with supportive care, as have other people shown to be infected by laboratory testing. Three patients (from Oklahoma, Tennessee, and Georgia) died. The virus has been found in a low number of nymphal and in lower numbers of adult *A. americanum* ticks. Raccoons and white-tailed deer are suspected amplifying hosts based on serology (Bosco-Lauth et al., 2015; Riemersma and Komar, 2015). The virus is the first phlebovirus identified from the Western Hemisphere and is most closely related to SFTSV. Additional epidemiologic investigations are needed to better characterize the clinical spectrum of disease.

Bourbon Virus Disease

During laboratory studies of the Heartland virus, plaque neutralization assays showed abnormal plaques from patients suspected of having Heartland viral disease (Kosoy et al., 2015). Subsequent genetic characterization identified a new virus, designated Bourbon virus (from the locality of Bourbon Co., Kansas, USA). This virus belongs to the genus *Thogotovirus* in the family Othromyxoviridae (Fig. 27.20C). Unfortunately, the index case patient died of the infection. Studies are underway to determine the prevalence of this new disease, but it appears to be uncommon. In 2017, a death in eastern Missouri was confirmed to be a consequence of Bourbon virus. The virus has been identified from *A. americanum* ticks collected in another part of Missouri, so the full range of this virus is still to be determined (Savage et al., 2017).

Rocky Mountain Spotted Fever

This disease was first recognized in the Bitterroot Valley of western Montana in 1872. Rocky Mountain spotted fever (RMSF) is widely distributed throughout most of the United States and, to a lesser extent, in Canada and Central and South America. In the United States, the disease was recognized only in the West until the 1930s when cases were detected for the first time in the east. Since 1985, about 600−800 cases of RMSF have been reported yearly with an annual national incidence ranging from 0.24 to 0.32 per 100,000 population. Most cases now occur east of the

Mississippi River in the southcentral and southeastern states, especially along the Atlantic coast (Fig. 27.21). Cases tend to occur in foci in rural areas and suburban communities near major population centers. In the southeastern states, the seasonal peak of reported cases typically occurs in July, coincident with, or shortly after, the period of peak abundance of adult *D. variabilis*. In the northeastern states, the peak is usually in May or early June, although a bimodal pattern may occur in this region.

RMSF is caused by a rickettsia of the spotted fever group, *Rickettsia rickettsii*, an intracellular bacterium that multiplies freely in the cytoplasm and occasionally in the nuclei of host cells. It can cause a severe disease of the circulatory system with significant mortality in untreated or inappropriately treated cases. Rickettsiae multiply in the endothelial linings of capillaries, smooth muscle of arterioles, and in other blood vessels (Fig. 27.22B). After an incubation period of about 7 days, patients develop fever, intense headaches, joint pain, muscle aches, nausea, and other symptoms. While dermatologic features may not appear until later in the disease course, a characteristic **maculopapular rash** (Fig. 27.22A) occurs in most patients several days after onset of symptoms. It consists of many tiny, pink or reddish spots, some of which may coalesce. The rash first appears on the hands and feet, gradually spreads to cover the entire body, and may persist for a week or longer. This particular pattern of progression is an important clinical feature of RMSF and helps to distinguish it from rashes produced by other vector-borne disease agents, such as epidemic typhus and allergic reactions. However, the extent of the rash indicates further progression of the disease and increasing severity. Severe cases may culminate in delirium or coma. Death can occur at any time during the acute clinical phase as a result of renal failure, clotting within blood vessels, shock, or encephalitis. Currently, 2%–5% of RMSF patients in the United States may die despite the availability of effective antibiotic therapy. Even treated patients may die, primarily due to delayed or inappropriate treatment.

Rickettsia rickettsii is transmitted by the bite of ixodid ticks. In the United States and Canada, *D. variabilis* (Fig. 27.13) and *D. andersoni* (Fig. 27.14) are the primary vectors. The elegant, pioneering work of **Dr. Howard T. Ricketts** at the turn of the 20th century elucidated the role of *D. andersoni* and its vertebrate hosts in the transmission cycle of *R. rickettsii*. Ricketts detected the agent in wild-caught ticks, and demonstrated experimentally that *D. andersoni* could transmit it to susceptible laboratory and

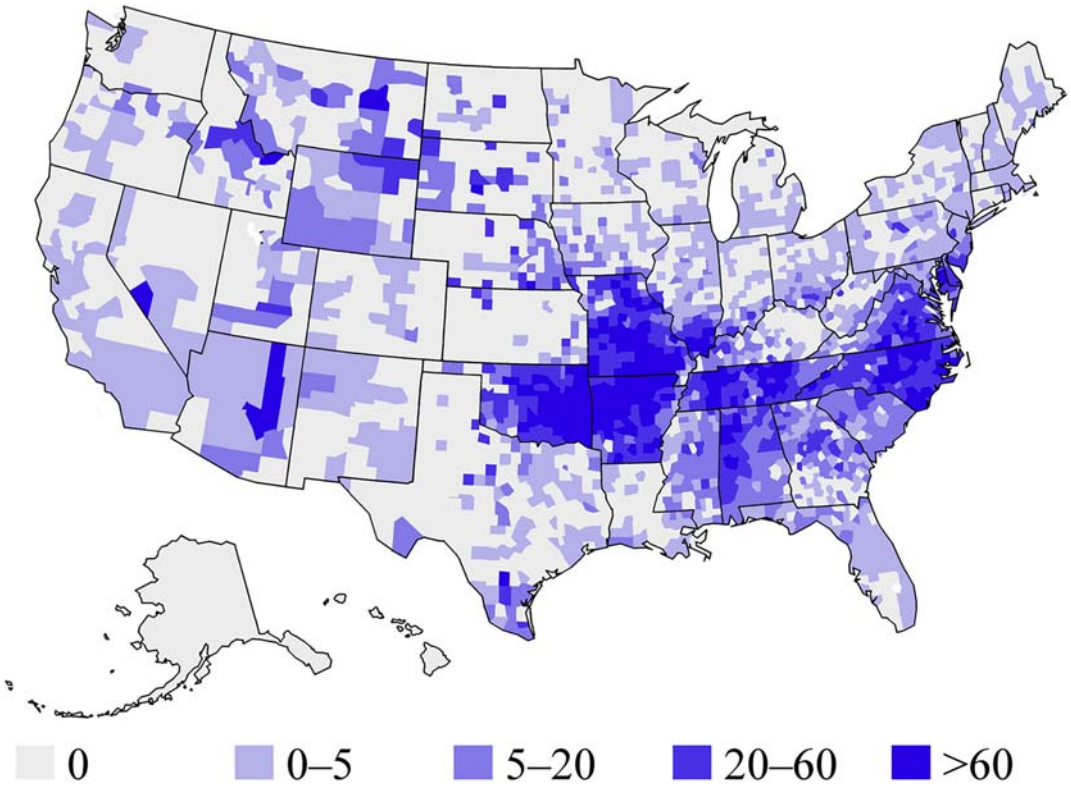


FIGURE 27.21 Reported incidence rate of spotted fever rickettsiosis, by county—United States, 2000–2013. *Per 1,000,000 persons per year. *Courtesy Rickettsial Zoonoses Branch, Centers for Disease Control and Prevention.*

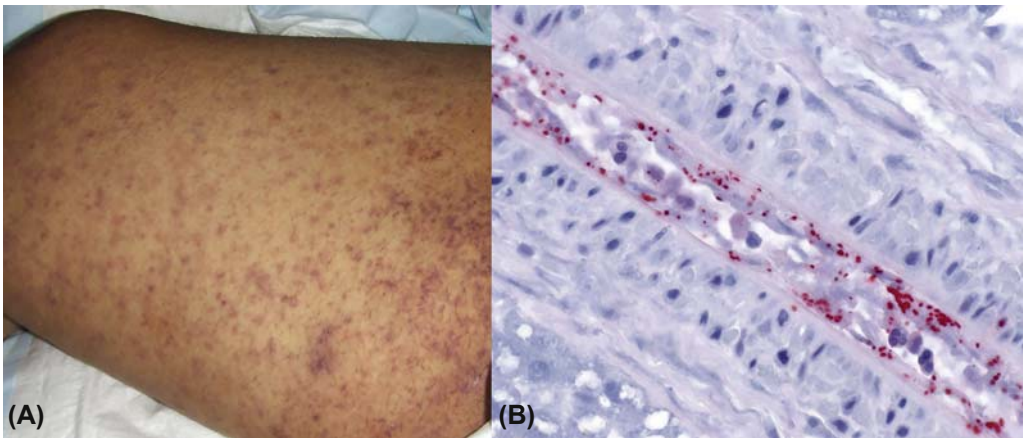


FIGURE 27.22 Rocky Mountain spotted fever (RMSF): (A) maculopapular rash with central petechiae associated with RMSF and (B) *Rickettsia rickettsii*, stained red by immunohistochemistry, in blood vessel endothelial cells. *Courtesy of Christopher D. Paddock, Centers for Disease Control and Prevention.*

wild rodents by the bite. Further, he showed that *R. rickettsii* is passed transstadially and transmitted transovarially within populations of ticks. His contributions laid the foundation for subsequent studies of tick-borne zoonotic agents in the United States and abroad.

Dermacentor variabilis is abundant throughout eastern North America, but it has a much more limited distribution in the West where it is not known to transmit *R. rickettsii*. In contrast, *D. andersoni* is restricted to western North America. The immature stages of both tick species feed on rodents and other small mammals, while the adults attack larger mammals, including people.

In 2003, cases of RMSF were reported from southeastern Arizona, which had previously reported few cases. Subsequent investigation showed an annual human incidence 300 times that anywhere else in the United States. The brown dog tick (*Rhipicephalus sanguineus*) (Fig. 27.12) was implicated as the vector (Demma et al., 2005b). The nymphal stage was identified as the likely vector to humans. *Rickettsia rickettsii* infection rates in the ticks reached 10% at the home of a fatal case and was 4% overall. High numbers of ticks were documented in the peridomestic environment but were rarely found in dwellings. The tick numbers were associated with the lack of control measures and an excessive stray or free-roaming dog population in the affected communities. Since then, cases continue to be reported from Arizona (Drexler et al., 2017). By the 2000s, RMSF cases were reported from northern Mexico and now number hundreds of cases, with fatalities primarily in children (Alverez-Hernandez et al., 2017). In retrospect, Parker and coworkers (1933) knew that this species had been shown to transmit this pathogen to humans in Mexico, and conducted studies to demonstrate that *R. sanguineus* is a very efficient vector in experimental infections. Other tick species that help to maintain *R. rickettsii* in nature include *H. leporispalustris*, which feeds on birds and rabbits, and *Ixodes texanus*, which feeds on raccoons. The lone star tick, *A. americanum* (Fig. 27.16), has been found infected naturally at high rates with **spotted fever group rickettsiae**, *R. amblyommatis,* which were believed to be nonpathogenic for humans. However, recent studies have suggested that a mild illness may result from bites of this tick and that serologic evidence can develop after such bites. Although *A. americanum* is suspected as a vector of RMSF rickettsiae and is capable of laboratory transmission (Parker et al., 1933; Levin et al., 2017), especially in endemic areas outside the distribution of *D. variabilis*, it is not considered to be a primary vector of *R. rickettsii*. In Central America and South America, various species of *Amblyomma* (*A. cajennense, A. mixtum, A. sculptum, A. patinoi, A. aureolatus, A. tonelliae,* and *A. tenellum*) have been implicated as vectors of *R. rickettsii* or closely related rickettsiae.

Larval and nymphal ticks maintain the infection from year-to-year and infect susceptible rodents when the ticks emerge to feed in the spring. Infected ticks must remain attached for at least 10 h before transmission can occur; this is known as the **reactivation phenomenon**. The delay in transmission is due to the fact that *R. rickettsii* seems to be in an avirulent state in unfed ticks and the rickettsiae become virulent only after prolonged attachment of the tick to its host or following ingestion of blood by ticks.

Most humans who contract RMSF are infected by the bite of adult ticks in late spring or summer, although nymphs occasionally transmit the infection. Only about 1%−3% of adult *Dermacentor* ticks in most foci are infected with spotted fever group rickettsiae, a small proportion of which are *R. rickettsii*. As previously noted, *Rhipicephalus* may show much higher prevalences of specific *R. rickettsii* infection. Ticks can be assayed for evidence of rickettsial infection by examination of their

hemolymph using immunofluorescence assays (IFAs). However, precise estimates of tick-infection prevalences with *R. rickettsii* are complicated by the potential presence of nonpathogenic spotted fever group rickettsiae, such as *Rickettsia montanensis, R. bellii,* and *R. rhipicephali*. IFA tests that employ species-specific monoclonal antibodies can resolve these. However, polymerase chain reaction (PCR) assays and nucleotide sequencing have provided more reliable means for determining tick infection prevalences and have largely replaced other methods.

Culture isolations of *R. rickettsii* have been made from numerous small and medium-sized wild mammals. Species that have been implicated as natural reservoirs include meadow voles and deer mice. In these animals, there are few if any obvious signs of clinical disease during infection. Young dogs in certain areas may also serve as short-term reservoirs for the pathogen and contribute infected ticks to the environment.

The period when rickettsiae are present in the blood of reservoir hosts is usually brief, often less than a week. Ticks feeding on infected animals may acquire rickettsiae, which produce generalized infections in tick tissues. In western North America, people normally become infected when they enter tick-infested habitats while engaged in outdoor activities in rural areas. In eastern North America, humans acquire their infections both in rural and peridomestic settings because dogs, which are significant hosts of adult *D. variabilis*, carry infected ticks into the home environment. Dog ownership is often noted as a risk factor in human cases.

Boutonneuse Fever

Boutonneuse fever, also known as Indian tick typhus, Kenya tick typhus, Crimean tick typhus, Marseilles fever, Mediterranean spotted fever, and Mediterranean tick fever, shares many features with Rocky Mountain spotted fever. However, the causative agent, *Rickettsia conorii*, does not occur in the Americas. It has an extensive range in southern Africa, India, central Asia, the Middle East, Europe, and North Africa. Patients with Boutonneuse fever develop fever, chills, severe headaches, and a rash. In addition, a button-like (=boutonneuse) ulcer called an **eschar** or a **tache noir** usually forms at or near the site of tick attachment (Fig. 27.23). The disease is generally milder than Rocky Mountain spotted fever, and most patients recover without antibiotic treatment. However, strains vary in virulence, and one that occurs in Israel has caused severe illness and several deaths. In temperate regions, cases of Boutonneuse fever are most common in late spring and summer, coincident with the seasonal activity of the primary tick vectors.

Rickettsia conorii is transmitted by several species of ixodid ticks in six genera (*Amblyomma, Dermacentor, Haemaphysalis, Hyalomma, Ixodes, Rhipicephalus*). In Europe, *Dermacentor reticulatus, D. marginatus*, and *I. ricinus* are important vectors. *Rhipicephalus sanguineus* is the principal vector in southern Europe, the Middle East, and North Africa, especially in countries bordering the Mediterranean Sea. Larvae and nymphs of *R. sanguineus* feed on small mammals, especially rodents and hedgehogs, whereas the adults feed mainly on larger mammals including humans. Lagomorphs, rodents, and possibly birds can serve as reservoir hosts. Dogs are susceptible to infection and transport vector ticks into and around human domiciles. Development of *R. conorii* within populations of ticks is similar to that of *R. rickettsii* in *D. andersoni* and *D. variabilis.*

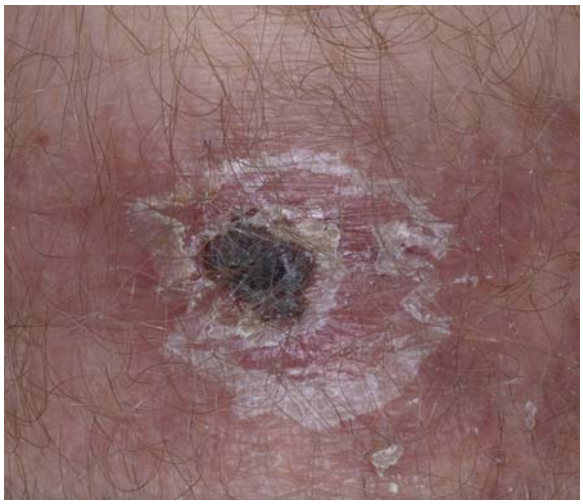


FIGURE 27.23 An eschar or tache noir lesion, may develop at the site of tick bite with infection by certain spotted fever group rickettsiae. *Courtesy of Christopher D. Paddock, Centers for Disease Control and Prevention.*

Separate subspecies of *R. conorii* have been shown to cause human disease in Israel, Sicily, and Portugal (Israeli spotted fever) and areas surrounding the Caspian Sea and in Chad (Astrakhan spotted fever) (Parola et al., 2005).

Other Spotted Fever Group Rickettsiae

African tick bite fever (also called South African tick typhus) is caused by *R. africae*. The clinical features are similar to those of Boutonneuse fever, but multiple eschars are more likely with this infection. This species is responsible for much of the imported spotted fever group rickettsial infections from sub-Saharan Africa and has been identified as established in the French West Indies. The pathogen is transmitted by ticks of the genus *Amblyomma*.

Additional rickettsioses are summarized by Parola et al. (2005) and will not be discussed at length here. *Rickettsia sibirica* may cause Siberian spotted fever and certain subspecies may cause lymphangitis-associated rickettsiosis or

tick-borne lymphadenitis (also known as *Dermacentor*-borne necrosis—erythema—lymphadenopathy). *Rickettsia aeschlimannii* has infected patients in Morocco in South Africa, *R. massiliae* causes illness in Spain, Italy, Canary Islands, and Argentina, while *R. helvetica* has been identified in humans from France and Sweden.

Queensland tick typhus is caused by *R. australis* and is found along the eastern coast of Australia. *Rickettsia honei* is found on Flinders Island near Tasmania (Flinders Island spotted fever), Thailand, and elsewhere. **Japanese spotted fever** is caused by *R. japonica* in southwestern Japan.

***Rickettsia parkeri* rickettsiosis** (also called tidewater fever) is caused by *Rickettsia parkeri*. Although the organism had been identified over 60 years earlier, *R. parkeri* was first reported as a human infection in 2004 (Paddock, 2005). Human infection is characterized by clinical findings similar to, and possibly confused with, RMSF. However, an eschar at the bite site with a maculopapular rash provides evidence of possible *R. parkeri* infection. Cases of the infection have been confirmed in the eastern United States inland to Virginia and, with newly identified cases from the southwestern United States (Allerdice et al., 2017). Infection by *R. parkeri* is associated with bites of the Gulf Coast tick, *Amblyomma maculatum* (Fig. 27.17), in which field studies have demonstrated infection by the rickettsiae.

Pacific Coast tick fever, caused by a novel rickettsial organism (provisionally designated 364D and later as "*Rickettsia philipii*"), was first described from California patients in 2008. The disease is characterized by one or multiple eschars, fever, and headache. It is transmitted to humans by the tick *Dermacentor occidentalis*. Larval, nymphal, and adult ticks have been found infected in nature, but the roles of various wildlife hosts for the tick have not been determined (Padgett et al., 2016).

Human Ehrlichiosis

Ehrlichiae are obligate intracellular organisms in the Family Anaplasmataceae that invade the cells of the vertebrate hematopoietic system. Several species in the genus *Ehrlichia* are important to human and veterinary health and grow within cytoplasmic vacuoles of monocytes, granulocytes, lymphocytes, or platelets. On infection of a cell, the ehrlichiae divide by binary fission to form microcolonies, known as **morulae**; cells may contain one or many morulae (Fig. 27.24). Although they are not commonly detected in routine examination of stained peripheral blood smears, the presence of morulae is helpful in presumptive diagnosis. Specific PCR assays and cell culture provide useful diagnostic methods.

Human infection by Anaplasmataceae was first described in Japan in the 1950s. The etiologic agent, *Neorickettsia sennetsu*, is transmitted to humans by an unknown mechanism, but ingestion of infected fish parasites is suspected. In the United States, human ehrlichiosis was first recognized as a febrile illness following a tick bite. Investigations of the disease in different parts of the world have found multiple species causing human disease. These organisms were reclassified into the family Anaplasmataceae in 1999, so that currently members of three genera and one proposed genus are recognized as human pathogens.

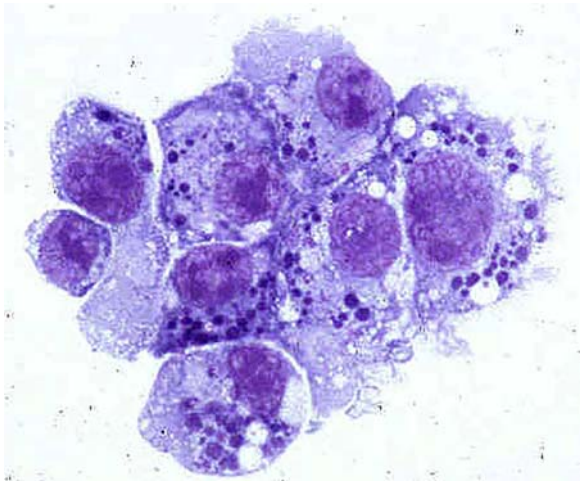


FIGURE 27.24 *Ehrlichia chaffeensis* growing as microcolonies, or morulae, in a cultured canine monocytic cell line (DH82), Wright—Giemsa stain. *Courtesy of Rickettsial Zoonoses Branch, Centers for Disease Control and Prevention.*

Ehrlichia chaffeensis is primarily found in the southeastern and southcentral United States, although there is evidence of wider distribution of this or similar agents. This species primarily invades the monocytic leukocytes (Fig. 27.24). The disease, originally called **human monocytic ehrlichiosis**, manifests as an acute illness with high fever, severe headaches, aching muscles and joints, and other nonspecific signs and symptoms. A rash is not common but may occur in about 20%—30% of younger patients. The disease can be mild, although many patients may require hospitalization. Severe cases can occur and may result in death, especially in those with compromised immune systems. Many cases of *E. chaffeensis* infection have been reported since its recognition, with 4,613 cases during the period 2008—2012 (Nichols Heitman et al., 2016). The primary vector for *E. chaffeensis* is *Amblyomma americanum* (Paddock and Childs, 2003; Childs and Paddock, 2003). This tick species feeds readily on many animals, including white-tailed deer, which serve as a reservoir for the ehrlichiae and an important host for the tick. Other wild and domestic animals have been identified as potential reservoirs based on serologic, cultural, and molecular studies.

Ehrlichia ewingii was first detected in granulocytes of human patients from Missouri in 1999 (Buller et al., 1999). Since then, additional cases have been reported, primarily in immunocompromised patients (Nichols Heitman et al., 2016). The etiologic agent has been recorded from several southern states where it was known to be a cause of illness in dogs. Experimental and field studies have determined that *Amblyomma americanum* is the primary vector of this organism. The reservoir for the pathogen is not known, but the organism has been identified in white-tailed deer. Other reservoirs may include wild or domestic canines.

Recently, *E. muris eauclairensis* (also known as the *Ehrlichia muris*-like agent), was detected in both people and dogs in the upper midwestern United States. The blacklegged tick, *I. scapularis*, was identified as the primary vector in experimentally infected and field-collected ticks. The organism has been detected in white-footed mice from endemic areas. More than 70 patients have been reported, and all resided in or traveled to Minnesota or Wisconsin (Johnson et al., 2015). Ticks removed from military personnel in other parts of the country have not been infected (Stromdahl et al., 2015). The main symptoms are fever and malaise, and successful treatment may be achieved with doxycycline.

Human Anaplasmosis

Human granulocytic anaplasmosis is caused by *Anaplasma phagocytophilum*, and this infection is widely distributed in temperate areas of North America, Europe, and Asia (Nicholson, 2018; Goodman et al., 2005). The pathogen resides in vacuoles in the cytoplasm of infected granulocytic cells (neutrophils and eosinophils) and replicates as microcolonies known as **morulae**. The number of cases reported in the United States has increased each year, with 8,896 cases reported in the period 2008−2012 (Dahlgren et al., 2015). The number of human cases in Europe or Asia has not been as high, but reports are increasing there as well.

Human disease manifests as fever, headache, chills, malaise, myalgia, and nausea in most patients. Vomiting, diarrhea, cough, arthralgia, and confusion are less common features of the infection. Rash is infrequently noted. Leukopenia, thrombocytopenia, and elevated hepatic enzymes may be found in clinical laboratory studies. Anaplasmosis cases reported to national surveillance systems have been increasing over the last several years. Severe complications are uncommon, but may be more frequent in elderly patients or patients with compromised immune systems. Complications due to delayed or inappropriate treatment may occur in patients coinfected with other tick-borne pathogens. More than a third of anaplasmosis patients require hospitalization, and <1% can die. These infections respond well to doxycycline treatment.

In the United States, the pathogen is transmitted to humans and domestic animals by the blacklegged tick *Ixodes scapularis* in the eastern and upper midwestern states and by *Ixodes pacificus* in northern California and other far western states. Local enzootic transmission is also known to occur among small mammals in certain areas by host-specific enzootic vectors (e.g., *Ixodes spinipalpis* among woodrats). In Europe, *Ixodes ricinus* serves as the primary tick vector, while *I. persulcatus* is the main vector in Asia. The pathogen is passed transstadially, but not transovarially in the ticks. A wide range of wildlife hosts in each geographic area provide bloodmeals to the ticks and may serve as reservoirs for the bacteria. In the United States, *Peromyscus leucopus* mice, *P. maniculatus* mice, *Neotoma* spp. woodrats, and various squirrel species have been demonstrated as reservoirs, but the maintenance in nature may be more complex as multiple wildlife species have been found to be infected in various parts of the world (Nieto and Foley, 2008). Domestic animals may also be infected and show clinical signs. *Anaplasma phagocytophilum* infection in ruminants was known for many years as tick-borne fever in Europe.

Other *Anaplasma* spp. have been found to infect humans with similar clinical features in various geographic regions. Their impact on public health will require additional attention as limited epidemiological studies have been conducted. *Anaplasma platys* was described from febrile patients in Venezuela and in the United States and is likely transmitted by the brown dog tick. *Anaplasma ovis* has been found in Europe, Asia, Africa, and North America with human cases in Cyprus and Iran. *Rhipicephalus* and *Dermacentor* ticks that feed on ruminants appear to be the vectors. A new species, designated "*Anaplasma capra,*" has been recently identified in patients in China and has been associated with goats and sheep. The pathogen has been detected in *Ixodes persulcatus* ticks (Li et al., 2015). Further study will be need to determine the full spectrum of illness and the eco-epidemiological features of these and other novel *Anaplasma* infections.

Human Neoehrlichiosis

Infection by the bacterial agent informally named "*Candidatus* Neoehrlichia mikurensis" is an emerging disease of humans in Europe and Asia (Silaghi et al., 2016). The pathogen represents a distinct taxon that has not yet been formally named, and thus was given a candidate designation. The organism appears to be widespread among certain species of small rodents in Europe and Asia. Voles have been found to be the most frequently

infected, and the vectors are *Ixodes ricinus* and *I. persulcatus* in endemic areas. Related pathogens have been identified in foxes and badgers in Europe and *I. holocyclus* ticks in Australia. Human cases have not been numerous, occur mainly in elderly adults, and are often associated with immune suppressive therapy or immunocompromised immune systems. Clinical features include fever, localized pain in joints or muscles, vascular and thromboembolic events, and transitory ischemic attacks. Leuocytosis with neutrophilia and anemia may be seen in laboratory studies. The infection responds to doxycycline treatment. In North America, the genus is represented by "*Candidatus* Neoehrlichia lotoris" found in raccoons, but no human cases have been identified, and the tick associate has not been determined.

Q Fever

First recognized among livestock handlers in Australia in 1935, Q fever is now known to occur on five other continents (Europe, Asia, Africa, North America, South America) and is probably worldwide in distribution. The etiologic agent, *Coxiella burnetii*, is a bacterium that develops in the phagolysosomes of the cytoplasm of susceptible cells. *Coxiella burnetii* can survive for months or years outside host cells under environmental conditions that are lethal to other bacteria. It can survive in dried tick feces, dried or frozen tissues, soil, and water.

After an incubation period of about 20 days, Q fever is characterized by sudden onset of fever, chills, sweats, diarrhea, sore throat, painful sensitivity to light, muscle pain, and headache. Fever may persist for 2 weeks and show a biphasic pattern. Fatigue, enlargement of the liver, and inflammation of the lungs, accompanied by a mild cough and chest pain, occur frequently. A rash is usually absent; when present, it appears on the trunk and shoulders. Q fever may become chronic, in which case it causes inflammation of the lining of the heart and its valves. The case-fatality rate is less than 1% in acute cases but may rise to 30% in chronic cases.

Transmission by ticks was first reported in 1938. Both argasid and ixodid ticks have been found infected naturally with *C. burnetii* or similar organisms. Subadult ticks infected while feeding on bacteremic hosts develop a generalized infection in their tissues. Following the transstadial molt, nymphs or adults transmit *C. burnetii* by bite, and females can pass the organism transovarially. Argasid ticks also can disseminate the organism via infectious coxal fluids. Notably, *C. burnetii* can survive in contaminated tick feces for as long as 6 years, which facilitates spread to humans and domestic animals. More recent work has questioned many of the historical associations of ticks with *C. burnetii* (Duron et al., 2015). *Coxiella*-like endosymbionts are widespread in many species of ticks and may have been misidentified as pathogenic *C. burnetii*. Recent studies have shown that *Coxiella*-like symbionts may be found in all life stages of several species of *Amblyomma* ticks, densely colonizing the salivary glands and ovaries with a function of improving reproductive fitness (Machado-Ferreira et al., 2016). Further work will be needed to better understand the true role of ticks in natural cycles of *C. burnetii.*

Coxiella burnetii can be maintained in enzootic cycles involving domestic animals (e.g., sheep, cattle, goats), wildlife, and their associated ticks. A cycle exists among Australian kangaroos (*Macropus major* and *M. minor*), the marsupial bandicoot (*Isoodon torosus*), and their associated host-specific ticks. Transmission to cattle and humans occurs when wild mammals also are parasitized by the nonspecific *Ixodes holocyclus*. In mammals, infection is usually asymptomatic, but abortions sometimes occur. Small mammals (e.g., *Apodemus, Microtus, Clethrionomys, Arvicola,* and *Pitymys* species) living in and around agricultural communities may link the domestic and sylvatic cycles. These animals develop high rickettsemias and shed the organism in their feces for weeks after becoming infected. Dogs, cats, birds, and reptiles also are susceptible to infection and may play a role in maintaining the infection in natural habitats. Although ticks are important in maintaining the pathogen horizontally and vertically in enzootic cycles, they rarely transmit *C. burnetii* to humans by bite. Instead, persons who handle infected animals or their products, or materials contaminated by tick feces, are at increased risk of acquiring *C. burnetii*. Tick excreta are an important source of infection because they are often highly contaminated and easily aerosolized. However, aerosols emanating from afterbirth membranes and associated fluids, blood, urine, feces, nasopharyngeal discharges, and milk containing high concentrations of the organism constitute the most common means for spreading the infection. As these materials dry, *C. burnetii* can be spread in aerosolized dust and debris present in animal stalls, barns, storerooms, and similar facilities. The most common site of Q fever epidemics is on farms or in farming communities, usually when domestic animals are being handled, such as during wool-shearing, lambing, calving, and slaughtering. Milk and milk products may be particularly important means of disseminating *C. burnetii* to humans; the organism may survive in contaminated milk and butter for up to 3 months.

Lyme Disease

Lyme disease, also known as Lyme borreliosis, erythema chronicum migrans, Bannwarth's syndrome, and tick-borne meningopolyneuritis, is a tick-borne disease caused by spirochetes in the group of related species known as the

Borrelia burgdorferi sensu lato (s.l.) complex (Fig. 27.26). This expanding group now includes at least 23 named genospecies and many recognized "genotypes" that remain unnamed at the time of writing; at least four *B. burgdorferi* s.l. species are important pathogens for humans, including *Borrelia burgdorferi* sensu stricto (s.s.), *B. afzelii, B. garinii*, and *B. speilmanii.* Several other genospecies reportedly infect humans occasionally or rarely (e.g., *B. bissettii, B. lusitaniae, B. americana, B. andersonii, B. bissettii,* and *B. mayonii)* (Clark et al., 2013; Golovchenko et al., 2016; Pritt et al., 2016; Rudenko et al., 2016).

The genome of the B31-type strain of *B. burgdorferi* s.s. was sequenced in 1997. North American and European populations of *B. burgdorferi* s.s. reportedly belong to genetically distinct populations, and this genospecies may have originated in Europe instead of North America as proposed earlier (Margos et al., 2008). During the previous two decades, whole genome sequences have been determined for isolates of several others of the *B. burgdorferi* s.l. complex, including *B. afzelii, B. bissettii, B. garinii, B. chilensis, B. mayonii, B. miyomotoi, B. spielmanii,* and *B. valaisiana* (Casjens et al., 2011a; Casjens et al., 2011b; Schutzer et al., 2011; Schutzer et al., 2012).

Although the etiologic agent was not discovered until 1981 (Burgdorfer et al., 1982), human cases have been documented in the medical literature dating back to the early 19th century. First recognized in the United States as a new form of inflammatory arthritis in Old Lyme, Connecticut, during the mid-l970s (Steere et al., 1977), Lyme disease and related disorders have since been reported from most states in the United States, southern Canada, and many countries of Europe and Asia. Human cases of Lyme disease also have been reported from Africa, Australia, Mexico, and South America (including Uruguay, Argentina, and Chile).

Lyme disease is the most commonly reported vector-borne disease throughout the temperate regions of the Northern Hemisphere, including North America and Europe, where the various syndromes account for hundreds of thousands of new cases annually. Using data collected between 2005 and 2010, the U.S. Centers for Disease Control and Prevention (CDC) estimated an incidence of 106.6 cases per 100,000 persons and that approximately 329,000 (95% confidence interval 296,000−376,000 and about 10 times the number of cases reported) people in the United States develop Lyme disease annually (Nelson et al., 2015). In Europe, the highest frequencies of the disease occur in Central Europe and Scandinavian countries including Austria, Germany, Slovenia, and Sweden where the annual incidence has been estimated to be as high as 120 (Slovenia) to 130 (Austria) cases per 100,000 residents (Steere et al., 2005). Much work remains to determine the public or veterinary health risks of *B. chilensis* in South America (Ivanova et al., 2014).

In the United States, Lyme disease is most prevalent in the northeastern states, especially in New York, Pennsylvania, New Jersey, and southern New England (Fig. 27.25); during 2008−2015 a total of 275,589 cases were reported to the CDC (Schwartz et al., 2017). Other major regional foci occur in the Upper Midwest, especially in Wisconsin and Minnesota, and in northern California. In the northeastern United States, people living in close proximity to forests, or in suburban communities having a mosaic patchwork of wooded areas and homes, have the highest risk of exposure to spirochete-infected ticks. White-tailed deer thrive in these habitats and, consequently, *I. scapularis* ticks abound. Moreover, infection rates in *I. scapularis* nymphs and adults are high. In one study in New York, 30% of nymphal and 50% of adult ticks were found to be infected with *B. burgdorferi.*

On the other hand, northern California cases are most likely to occur in semirural or rural settings where *I. pacificus* is abundant. However, infection prevalences in this tick (typically 1%−2% in adults, 2%−15% in nymphs) are generally much lower than they are in northeastern populations of *I. scapularis,* and the risk of infection to humans is correspondingly lower. Cases occur throughout the year, but most often are seen in spring and early summer when the nymphs reach peak densities. In contrast, *I. pacificus* adults are primarily active in fall and winter, when temperatures are cool and humidities are high.

In addition to greater awareness and increased tick-surveillance activities, several ecological and epidemiological factors have contributed to the current epidemic of Lyme disease in the northeastern United States and Europe. These include the occurrence of abundant, efficient tick vectors on both continents; the presence of numerous natural hosts for the immature and adult stages of the vectors; high rates of spirochetal infection in reservoir populations; anthropogenic changes (e.g., deforestation, reforestation, suburbanization) that favor an increased abundance of amplifying hosts and infected vector ticks; and close proximity of susceptible human populations to populations of tick vectors. Moreover, efficient transspecies transmission among the mammalian hosts of *B. burgdorferi,* a generalist microparasite, seems to have fueled the rapid epidemic spread of Lyme disease in the northeastern United States (Hanincová et al., 2006). In some European countries, the greater species diversity of *B. burgdorferi* s.l. spirochetes pathogenic for humans also may have contributed to the upsurge in reported cases as populations of *I. ricinus* expanded and increased in abundance.

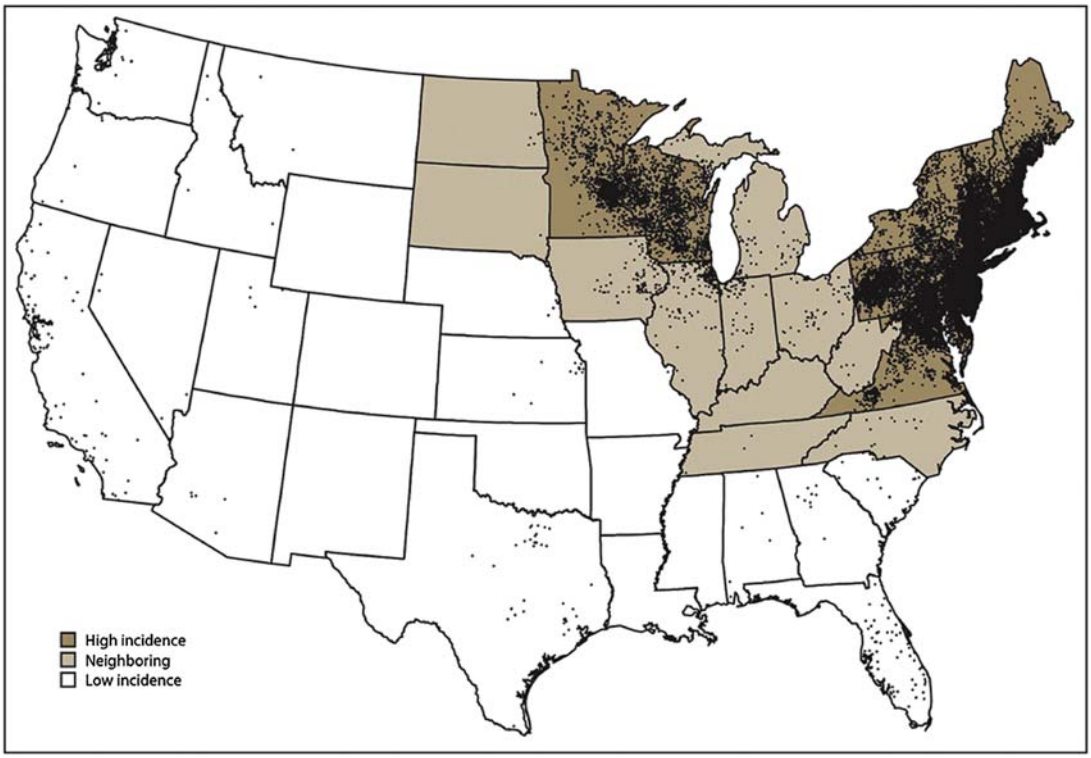


FIGURE 27.25 Lyme disease, mean annual number of confirmed cases by county of residence—United States, 2008−2015. *Courtesy of Centers for Disease Control and Prevention.*

When injected into humans by a feeding tick, borreliae multiply and disseminate in the skin. Gradually, they invade the bloodstream and may spread throughout the body, often localizing in the bursae of the large joints, in the central or peripheral nervous systems, and in the heart. Clinical signs and symptoms usually appear within 1−2 weeks (range, 3−32 days) following the bite of an infectious tick. Most cases occur during the late spring or summer, coincident with the seasonal activity of the nymphal stages of the primary vectors. *Ixodes persulcatus* is a notable exception; the adult female of this Eurasian tick seems to be the primary life stage that transmits spirochetes to humans (Piesman and Humair, 2012).

Early-stage Lyme disease is characterized by nonspecific ("flu-like") symptoms and by an erythematous skin rash, erythema migrans (EM), which is present in 60%−80% of patients. Erythema migrans is a slowly expanding, usually circular or elliptical, but sometimes triangular- or irregularly shaped lesion that often exhibits bright red outer margins and partial central clearing (Fig. 27.26B). Most patients have one EM lesion at the site of tick attachment, but 25%−50% may develop multiple satellite lesions. The rash should not be confused with erythematous skin lesions that develop within minutes to a few hours, and tend to expand rapidly in size, after the attachment of an *Ixodes* tick. Such lesions typically result from allergic hypersensitivity reactions following the injection of tick-salivary proteins (Steere et al., 2016). In the range of the lone star tick, a clinical entity known as southern tick-associated rash illness (STARI) is temporally linked to a bite by *Amblyomma americanum*. An erythema migrans-like skin lesion is the dominant sign, but the rash may be accompanied by mild systemic features such as myalgia, arthralgia, fatigue, fever, chills, and headache. The cause is not yet known, and serologic and molecular studies for borreliosis and rickettsiosis have not shown evidence for these infections. Recently, a new diagnostic approach using metabolic characterization has led to the ability to differentiate STARI from early Lyme disease with an accuracy of 85%−98% (Molens et al., 2017).

Untreated Lyme patients may manifest no further signs or symptoms of illness, or they may go on to develop late-stage Lyme disease within one to several months. Late manifestations, cardiac, neurologic, arthritic, or further dermatologic abnormalities may occur either alone (e.g., acrodermatitis chronica atrophicans, caused by *B. afzelii*) or in combination. Recently, several sudden fatal cases of Lyme carditis were reported (Muehlenbachs et al., 2016).

The following account of the remarkably diverse ecology of *B. burgdorferi* s.l. spirochetes is selective because of

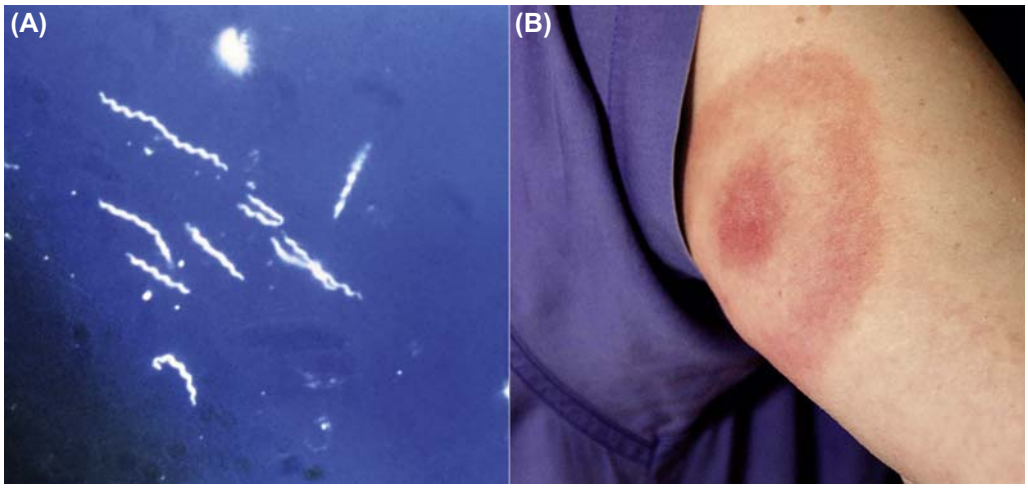


FIGURE 27.26 Lyme disease: (A) *Borrelia burgdorferi* spirochetes as seen under darkfield microscopy and (B) erythema migrans skin lesion on arm of patient, commonly seen in early stage of Lyme disease. *Courtesy of Centers for Disease Control and Prevention.*

space constraints. The interested reader is referred to pertinent chapters in Gray et al. (2002) and to Piesman and Humair (2011) for greater in-depth coverage of the voluminous literature regarding the ecology in different geographic regions.

At least 40 species of ixodid ticks and two species of argasid tick have been found infected naturally with *B. burgdurferi* s.l. spirochetes. In most endemic foci, however, only a single member of the *Ixodes ricinus* complex serves as the primary vector to people. Thus, spirochetes are transmitted to humans by *I. scapularis* and *I. pacificus,* in eastern and western North America, respectively; by *I. ricinus* in Europe and western Asia; and by *I. persulcatus* in eastern Europe and Asia. Other *Ixodes* ticks that seldom or never attach to humans, and do (e.g., *I. jellisoni)* or do not belong to the *I. ricinus* complex (e.g., *I. spinipalpis* in the western United States; *I. dentatus* in the eastern United States; and *I. ovatus* in Japan), may serve as efficient enzootic (maintenance) vectors of *B. burgdurferi* s.l. Ticks in other genera rarely serve as vectors to people. For example, *A. americanum* in New Jersey and the southeastern United States has been implicated, even though several experimental studies have shown that it is an incompetent vector of certain isolates of *B. burgdorferi* (Feir et al., 1994; Clark et al., 2013; Rudenko et al., 2016).

The timing of feeding by different stages of a vector also may influence prevalence in vectors and hosts, as well as risks for people. Infective nymphal *I. scapularis*, infected with *B. burgdorferi* s.l. from feeding as larvae during the summer of the preceding year, feed on naïve mice during spring and ensure that reservoir hosts are available to infect the cohort of larvae that emerges from eggs during the summer. This reversal of the feeding phenology (nymphs feed during spring before larvae feed during summer) amplifies the prevalence of infection in both the reservoir hosts and the vectors, therefore increasing the risk of transmission to people in the northeastern United States (Spielman et al., 1985).

In *Ixodes* spp. immatures, development of *B. burgdorferi* s.s. begins with ingestion of an infectious bloodmeal. *Borrelia burgdurferi* s.l. normally develop extracellularly by binary fission in the midgut diverticula of *lxodes* spp. ticks, although spirochetes have been found occasionally in oocytes of the ovaries and in secretory cells of the salivary glands. Following the transstadial molt and resumption of tick feeding on another host, spirochetes escape from the midgut, enter the hemocoel, and migrate to the salivary glands (Zung et al., 1989). In some ticks, borreliae spread to other organs as well. Thus, spirochetes are maintained within populations of vector ticks by transstadial passage and by replenishment as noninfected ticks feed on infectious hosts. Transovarial transmission of *B. burgdorferi* s.l. has been documented in some of its primary vectors, but this mechanism appears to be inefficient for perpetuating and distributing most genospecies (e.g., *B. burgdorferi* s.l. by *I. pacificus* or *I. scapularis).*

The potential importance of the seasonal timing of vectors on epidemiologic patterns is illustrated by the classic 2-year transmission cycle involving *I. scapularis* in the northeastern and upper midwestern regions of the United States. In the colder, northern, regions, larval *I. scapularis* feed on reservoir hosts later in the summer than do nymphs of the prior year's cohort. This results in nymphs infected with borreliae feeding on, and infecting, naïve reservoir hosts; uninfected larvae then feed on those infected hosts and cycle amplifies in the tick populations increasing the risks to people in the region (Spielman et al., 1985: Lane, 1994; Piesman and Gray, 1994). Where winters are somewhat mild, the larvae feed simultaneously or

earlier in the season than do the nymphs and little or no amplification occurs by this ecological pattern of immature feeding. However, when ecological patterns seem simple, we should all look closer at the details. More recent accounts have shown the importance of simultaneous feeding activity and future studies focused on the importance of the timing of feeding may be warranted.

In addition to ticks, spirochetes have been detected in mosquitoes, deer flies, and horse flies in the northeastern United States and Europe, where anecdotal accounts suggest that some individuals may acquire spirochetal infections following the bites of blood-sucking insects. Although the overall role of insects in the ecology and epidemiology of *B. burgdorferi* s.l. appears to be minimal, and the finding of pathogens consumed in a bloodmeal should not be used to implicate a vector, further investigation may be warranted.

Wherever the ecology of *B. burgdorferi* s.l. has been studied intensively, typically one or more species of rodents or insectivores, and less often birds or lizards, have been implicated as primary reservoir hosts. Different genospecies may be associated with different vertebrates or even classes of vertebrates. Thus, *B. afzelii* and *B. burgdorferi* are associated predominantly with small mammals, *B. garinii* and *B. valaisiana* primarily with birds, and *B. lusitaniae with* lizards; however, *B. burgdorferi* and *B. garinii* parasitize some birds and small mammals, respectively.

The term reservoir, as used herein, pertains to those vertebrate host species that (1) are infected commonly with a particular borrelial genospecies, (2) maintain infections for prolonged periods, if not for life, (3) remain infectious for ticks that feed on them, and (4) are fed upon commonly by vector-competent ticks. However, each local nidus includes a different community of potential reservoir hosts, and it is the community ecology of hosts and vectors that determines the importance of each local host species in the ecological maintenance of pathogens, including *B. burgdorferi* s.l. Body size and natural history have been suggested to characterize reservoir hosts of B. burgdorferi s.l.; specifically small body size, populations that occur in high density, and a fast pace of life characterize reservoir competence (Ostfeld et al., 2014; Barbour et al., 2015).

In North America, competent reservoirs of *B. burgdorferi* s.l. include white-footed mice, eastern chipmunks, short-tailed shrews, and masked shrews in the Northeast and Upper Midwest, and western gray squirrels, dusky-footed woodrats, some species of chipmumks, and California kangaroo rats in the farwestern United States. In Europe, common shrews, bank voles, wood mice, yellow-necked field mice, edible and garden dormice, gray squirrels, red squirrels, hedgehogs, and hares exhibit varying degrees of reservoir competence for *B. burgdorferi* s.l., and reservoirs of *B. garinii* include a variety of migratory birds (Piesman and Humair, 2011). In some geographic regions, a few species of birds (e.g., American robin and song sparrow in the United States; Eurasian blackbird) may play significant enzootiologic roles by providing populations of immature ticks with bloodmeals, by infecting vector ticks with *B. burgdorferi* s.l. and by transporting infected ticks considerable distances and thereby establishing new foci of infection. Conversely, some vertebrates that are excellent hosts of vector ticks, such as deer and certain lizards in North America, and roe deer in Europe, do not serve as reservoir hosts even though they may greatly increase the tick population. They help to reduce tick-infection prevalences and the risk of human exposure to spirochetes; this has been termed **zooprophylaxis**. Nonetheless, these and certain other vertebrate hosts are important tick-maintenance hosts that sustain local or regional tick abundance.

As an example, the western fence lizard is an important maintenance host of *I. pacificus* immatures in many biotopes in California. Its reservoir incompetence stems from the fact that it contains a heat-labile, spirochete-killing (borreliacidal) factor in its blood. This factor destroys spirochetes in the midgut diverticula of infected nymphs either while they feed, or soon after they have fed, on lizard blood, with the result that, after the transstadial molt, the adult ticks are devoid of spirochetes. Eliminating spirochetal infections from vector ticks like *I. pacificus* seems like it might reduce the force of transmission of a zoonotic agent to humans or other animals by reducing the prevalence in infected adult ticks, but the ecological impacts are complicated; even a zooprophylactic species, such as the western fence lizard, may increase the force of transmission by promoting larger tick populations (Swei et al., 2011).

The mechanism responsible for the borreliacidal activity in preimmune sera from the western fence lizard and the southern alligator lizard was demonstrated to reside in proteins comprising the alternative complement pathway (Lane and Quistad, 1998; Kuo et al., 2000). In Europe, complement-mediated borreliacidal effects observed for specific combinations of vertebrate-host serum and different genospecies of *B. burgdorferi* s.l. generally coincided with what was known about the reservoir potential of the mammalian and avian species evaluated (Kurtenbach et al., 1998, 2002).

To understand the role of lizards, birds, or mammals in the community ecology of *B. burgdorferi* s.l. and Lyme disease risk, each vertebrate species must be assessed separately under both field and laboratory conditions before biologically meaningful conclusions can be reached (e.g., (Mather et al., 1989; LoGiudice et al., 2003; Brisson et al., 2008), and then the numbers (or, if possible, the relative proportion) of ticks that feed on each host must be

determined in the field. In that regard, few lizards had been studied intensively enough with the exception of the western fence lizard in northern California. Findings stemming from the earlier studies collectively resulted in a belief that other lizards, in general, might be reservoir-incompetent hosts. However, although some lizards indeed are nonreservoir hosts, others are reservoir competent. In the southeastern United States, several lizards are hosts for *B. burgdorferi* s.l. and serve as a source of spirochete infection for ticks (Levin et al., 1996; Clark et al., 2005). Likewise, in several European countries (Germany, Italy, Slovakia) and in Tunisia, at least four species of lizards have been implicated as primary reservoir hosts of *B. lusitaniae* (Tijsse-Klasen et al., 2010; Ragagli et al., 2011).

The various *B. burgdorferi* s.l. genospecies are generalist pathogens that infect a range of hosts. Likewise, the ticks that transmit infections to humans, domestic animals and many wildlife species are generalist feeders. Although the tendency is to look for simple reservoirs for control purposes, the reservoirs and vectors of this group of pathogens is complex and managers need to know the local system of vectors and vertebrate hosts maintaining the pathogen in nature in order to manage the risks for people or domestic animals.

Tick-Borne Relapsing Fever and *Borrelia miyamotoi* Disease

Human cases of tick-borne relapsing fevers (TBRF), also known as tick-borne spirochetoses and endemic relapsing fevers, are caused by about 20 species of borreliae; and the list will likely continue to grow. Classically, all TBRFs were associated with one or a couple of different species of argasid tick in the genus *Ornithodoros*. More recently, a spirochete, *B. miyamotoi*, which is closely related to TBRF spirochetes from North America, and divergent from *B. burgdorferi* s.l., has been shown to be transmitted by the primary vectors of Lyme disease; resulting in the names "argasid-borne relapsing fevers" and hard tick-borne relapsing fever (or *B. miyamotoi* disease, BMD). *Borrelia miyamotoi* has been found to occur in from Russia, Europe, Japan, and the United States (Fish, 2013; Crowder et al., 2014; Telford et al., 2015; Khasnatinov et al., 2016; Iwabu-Itoh et al., 2017).

Human cases of TBRF occur on five of the seven continents, and cycles include the associations of ticks and borreliae shown in Table 27.4. Early descriptions confused TBRFs with louse-borne relapsing fever caused by *B. recurrentis*, and transmission by ticks was not recognized until the pioneering work of Dutton and Todd (1905) who detected spirochetes in *Ornithodoros moubata* from East Africa; and after Joseph Dutton died of the disease in 1904 at the age of only 30 years (Köhler, 2006).

Onset of TBRF in humans is characterized by fever, chills, and a throbbing headache, usually without a pronounced rash or an ulcer at the bite site. Following the incubation period of approximately 1 week, an episode of fever usually lasts 3−5 days during which time spirochetes are present in the peripheral blood. The febrile period ends in a period of crisis involving very high fever (up to 106.7°F). Subsequent relapses of 3−5 days follow periods of 5−7 days during which spirochetes are difficult to find in the bloodstream. This alternating cycle of febrile and afebrile periods in untreated patients may be repeated two or more times, accounting for the epithets of relapsing fevers (Dworkin et al., 2008). Depending on the number of cycles, the illness may be extended for several weeks or longer and sometimes ends in death. Other signs or symptoms that occur often include muscle ache, joint pain, abdominal pain, nausea, vomiting, diarrhea, and a petechial rash on the trunk; acute respiratory distress has occurred and TBRF has been associated with neural disease. Although the full range of the pathology caused is beyond our scope, the pathophysiology appears to be related to mild disseminated intravascular coagulation and vascular microemboli that form around individual borreliae (Dworkin et al., 2008). This range of signs indicates that severe clinical conditions might occur more often than previously reported.

The epidemiology of TBRF in the United States, and the possibilities for reemergence of tick- and louse-borne relapsing-fever infections globally, have been reviewed recently (Dworkin et al., 2008; Trape et al., 2013; Forrester et al., 2015). Argasid borne borreliae show a high level of vector specificity, mostly with species in the genus *Ornthodoros*, which is in contrast to the specificity of most *B. burgdorferi* s.l. genospecies and of *B. miyamotoi* with their primary vectors.

In certain TBRF-endemic areas of Tanzania, the annual incidence of *B. duttonii* infection reportedly is 384 per 1,000 in infants (less than 1 year of age) and 163 per 1,000 in children (less than 5 years of age); the perinatal mortality rate is a staggering 436 per 1,000 (McConnell, 2003). Likewise, in Senegal, the average incidence of *B. crocidurae* infection in all age groups was 11 per 100 person-years from 1990 to 2003 (Vial et al., 2006). In marked contrast, the incidence of TBRF in endemic areas of developed countries (e.g., Israel, United States) is orders of magnitude lower (Sidi et al., 2005).

The relapsing nature of the disease is explained by the antigenic variability of the borreliae (Dworkin et al., 2008; Forrester et al., 2015; Lopez et al., 2016). Some spirochetes are able to alter their surface-protein composition, probably through transposition of the genes encoding them.

TABLE 27.4 Associations of Argasid-Borne Relapsing Fever Spirochetes, Their Vectors, Main Reservoir Group, and the General Geographic Areas Where the Diseases Occur

Borrelia sp.	*Ornithodoros* sp. Vectors	Reservoirs	Geographic Region
B. baltazardii	*Unknown*	Unknown	Iran
B. brasiliensis	*O. brasiliensis*	Unknown	Brazil
B. causasica	*O. verrucosus*	Rodents	Caucasus Mountains to Iraq
B. crocidurae	*O. sonrai*	Rodents & insectivores	West and North Africa
B. dipodilli	*O. erraticus*[a]	Rodents	North Africa, East Africa, western Asia
B. duttoni	*O. moubata; O. porcinus*	Humans Humans	Central Africa, Eastern Africa, and southern Africa Central Africa, Eastern Africa, and southern Africa
B. graingeri	*O. graingeri*	Rodents	Kenya
B. hermsii	*O. hermsi*	Rodents	Western USA and southwestern Canada
B. hispanica	*O. macrocanus;* *O. occidentalis;* *O. kairouanensis*	Rodents Probably rodents Probably rodents	Iberian Peninsula, Greece, Cyprus and North Africa Iberian Peninsula and North Africa Tunisia
B. kalaharica	*Unknown*	Unknown	Southwestern Africa
B. latyschewii	*O. tartakowskyi*	Reptiles & rodents	Central Asia, Iran, and Iraq
B. mazzottii	*O. talaje*	Rodents	Southern USA, Mexico, and Central America
B. merionesi	*O. costalis;* *O. merionesi*	Rodents Rodents	North Africa North Africa
B. microti	*O. erraticus*	Rodents	Africa and Iran
B. parkeri	*O. parkeri*	Rodents	Western USA
B. persica	*O. tholozani*	Rodents	Middle East and Asia
B. turicatae	*O. turicata*	Rodents	USA and Mexico
B. tillae	*O. zumpti*	Rodents	Southern Africa
B. neotropicales	Unknown	Unknown	Central America
B. venezuelensis	*O. rudis*	Rodents	Central America and South America

[a]*The* O. erraticus *complex of ticks in North and West Africa has been partially resolved (Trape et al., 2013), but further resolution is merited to define geographic distributions and clarify the taxonomy.*

Consequently, new populations of spirochetes emerge in an infected host with each relapse and they multiply before the host can mount an effective antibody response against them. Crisis occurs as the spirochetes are controlled by the immune response, the infection is controlled (or death occurs) and a short period of respite occurs.

In their tick vectors, borreliae ingested with blood disseminate to the internal organs, including the salivary glands. Spirochetes are passed transstadially so that, once infected, ticks remain so for life; transovarial transmission has not been shown to be important in the epidemiology of relapsing fevers (Dworkin et al., 2008; Tabuchi et al., 2008). This, together with the long life-span of *Ornithodoros* ticks, and the resultant lag times in the transmission cycle, enhances the likelihood that relapsing fever spirochetes will persist in tick-infested habitats for prolonged periods. Transovarial transmission of relapsing-fever group spirochetes has been demonstrated in *O. coriaceus*, *O. erraticus*, *O. hermsi*, *O. moubata*, *O. tartakovskyi*, *O. tholmani*, *O. turicata*, *O. sonrai*, and *O. verrucosus*, but not *in O. parkeri, O. rudis,* or *O. talaje* (Burgdorfer and Schwan, 1991; Dworkin et al., 2008; Tabuchi et al., 2008). Borreliae also invade the coxal glands of some *Omithodoros* spp. and can be transmitted to hosts via coxal fluid excreted during or soon after the bloodmeal.

Rodents serve as the primary reservoir hosts of most borreliae transmitted by *Omithodoros* ticks. Four notable exceptions are *B. anserina*, *B. coriaceae, "B. lonestari",* and *B. duttoni.* In the United States "*B. lonestari*" was once thought to be the cause of southern tick-associated rash illness (STARI); the association of STARI and "*B. lonestari*" has been discounted. Columbian black-tailed deer have been implicated as reservoirs of *B. coriaceus* in California, and white-tailed deer as reservoirs of

ixodid-transmitted "*B. lonestari*" in the southern United States. *Borrelia anserina* is an important pathogen of chickens and other fowl but is not zoonotic.

The established dogma is that only humans serve as reservoirs of *B. duttoni,* the cause of East African tick-borne or endemic relapsing fever. Recent evidence, however, suggests that *B. duttoni* (or a related strain) is a zoonosis in central Tanzania (McCall et al., 2007). Domestic animals associated closely with households (i.e., tembe houses consisting of adobe walls, flat earthen roofs, and soil floors) in a TBRF endemic region were infected with borreliae that shared greatest homology with *B. duttonii.* Nearly half of 122 houses surveyed were infested with *O. moubata* s.l. ticks, and 11% of the chickens and 9% of the pigs tested were PCR positive. Also, a mark-release-recapture experiment revealed that about 3% of the recaptured ticks had moved from their release sites in pigpens into adjoining or nearby houses 7−25 days postrelease; whether this is generally true for *B. duttoni* or related to a specific strain of borreliae in Tanzania remains to be reported and more work is warranted.

Epidemiologically, TBRF is a highly focal disease; presumably as a result of the host specificity and natural history of the tick vectors. In the United States, most human cases are caused by *B. hermsii,* between 1990 and 2011, TBRF was reported mainly from 12 western states (Fig. 27.27) with 70% of the cases being reported from California, Colorado, and Washington (Forrester et al., 2015); however, the most cases for a county (n = 29) came from Kootenai County, Idaho. Because this is not a reportable disease in the US, the numbers reported to authorities likely under-report the actual number of cases. The median elevation for the 10 counties with the most cases was 3,840 ft (range 1,178−7,562 ft) illustrating that TBRF occurs most commonly at mid-elevations where the vector, *O. hermsi* lives in rodent burrows in coniferous forests and feeds most commonly on rodents such as chipmunks, tree squirrels, and ground squirrels. Interestingly, *B. hermsii* has been isolated from a northern spotted owl that died from the infection (Thomas et al., 2002) suggesting that avian species might also be involved as part of the reservoir (Dworkin et al., 2008; Yabsley et al., 2012). In addition, to *B. hermsii*, *O. turicata* transmits *B. turicatae* in the southwestern US, Florida, Mexico, and Central America. This vector lives in limestone caves, animal burrows, and in peri-domestic structures where there are animals to feed upon (Dworkin et al., 2008). People generally become infected at night when they stay in structures or caves where the vectors occur.

Another vector of relapsing-fever spirochetes in western North America is *O. parkeri,* which typically occurs at lower elevations than *O. hermsi.* Although new isolates of *B. parkeri* were only recently obtained, cases have been identified from appropriate exposure to semi-arid, sagebrush, habitats, prairie dog burrows, and other rodent burrows, since 1934. This tick rarely infects humans because *O. parkeri* ticks inhabit the burrows of rodents

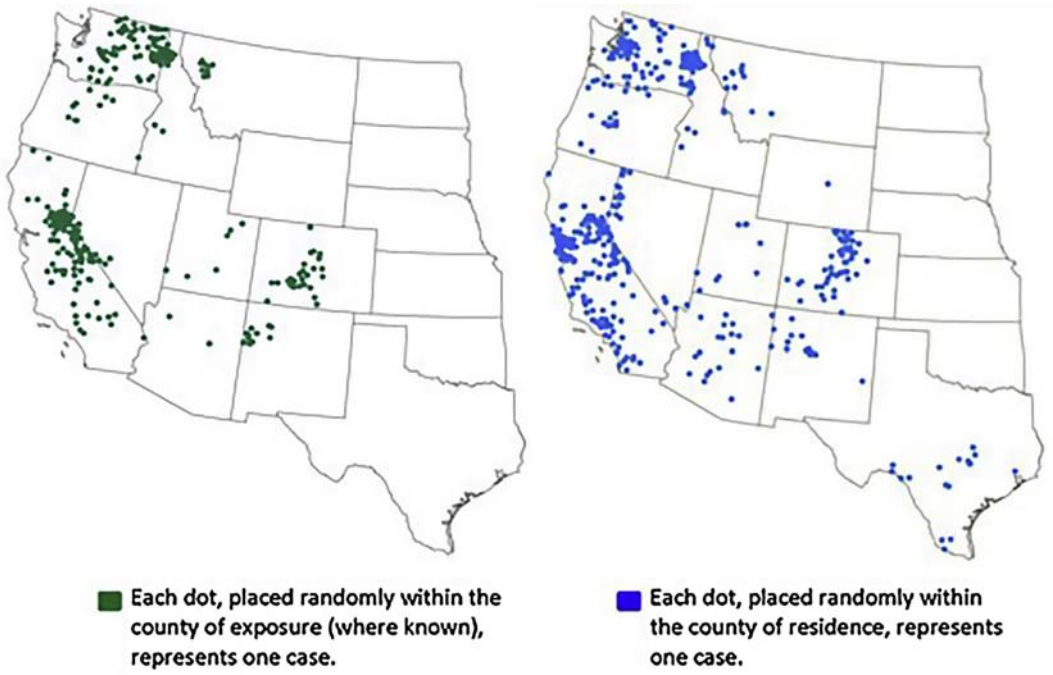


FIGURE 27.27 Human cases of tick-borne relapsing fever in the United States between 1990 and 2011. *Courtesy of Centers for Disease Control and Prevention.*

(e.g., California ground squirrels) and therefore have little opportunity to feed upon people.

Borrelia miyamotoi has been known since the 1990s (Fukunaga et al., 1995), but was only reported as a human pathogen in the US in 2013 (Branda and Rosenberg, 2013; Krause et al., 2015). The geographic range, like that of Lyme disease, includes the Holarctic region overlapping the distributions of the primary vectors *I. ricinus, I. persulcatus, I. pacificus*, and *I. scapularis*. It differs from other TBRFs because it is transmitted by a hard tick and because the signs are somewhat less pronounced (Telford et al., 2015). However, the organism clearly groups phylogenetically with the TBRF borreliae from North America (Barbour, 2014). The pathogen has been identified in a number of wildlife species and birds. High prevalences have been noted in wild turkeys collected in Tennessee.

One of the difficulties of studying TBRF has been related to the classification of the borreliae, for which taxonomy continues to be revised and expanded. As an example of taxonomic changes in the vector, the *O. erraticus*-complex used to be discussed as the "large variety" and the "small variety." Trape and coworkers (Trape et al., 2013) recently described nine species that were once all discussed as *O. erraticus* in western Africa, northern Africa, and the Iberian Peninsula, and additional work is needed to resolve species relationships in Central Asia, the Middle East, and Eastern Europe. As noted by Estrada-Peña et al. (2010), there are about 190 species of argasid ticks, and the generic and subgeneric taxonomy is somewhat controversial for about 130 of those species. The potential for understanding the natural history, and therefore the potential for management, depends upon the assumption that we know what species we are studying or reporting. Only then can researchers evaluate the nuances of transmission cycles and accurately describe the risks.

Tularemia

Tularemia was first identified as a distinct clinical entity in Japan in 1837, where it was attributed to infected hares. It was first recognized in the western United States in 1911 by McCoy, who described it as "a plague-like disease of rodents." The organism was detected in tissues of the California ground squirrel. In the United States, it has long been associated with hunters, who acquire the infection while skinning wild rabbits (hence the colloquial name, "**rabbit fever**"), and with persons who handle infected livestock, especially sheep. Tularemia is increasingly becoming more of a concern throughout Europe with over 15,000 cases recorded between 1997 and 2013 (Maurin and Gyuranecz, 2016). Workers in the former Soviet Union recognized this disease among trappers handling European water voles (*Arvicola amphibius*) and established that it was caused by the same bacterium. For excellent overviews of tularemia, see Jellison (1974), Bell (1988), Eisen (2007), Foley and Neito (2010), Telford and Goethert (2011), Carvalho et al. (2014), Mani et al. (2016) and Maurin and Gyuranecz (2016).

The causative agent, *Francisella tularensis*, is a pleomorphic, gram-negative, aerobic bacterium. It exists in different forms termed biovars, which are strains having special biochemical or physiological properties. Two biovars of *F. tularensis* occur in North America; a highly virulent form associated with rabbits, cats, sheep, and ticks (type A, biovar *F. t. tularensis*), and an apparently waterborne, less virulent form associated with beavers, muskrats, and voles (type B, biovar *F. t. holarctica*). Biovar *F. t. tularensis* (Type A1 (virulent subtype in the central United States) and A2 (less virulent in the western United States) is the most common and known from North America and recently Europe. This type is fatal in 5%−7% of untreated patients. Biovar *F. t. holarctica* (=*palaearctica*) occurs throughout the Northern Hemisphere and is rarely fatal to humans. A third biovar, *F. t. mediaasiatica,* is found in Central Asia, whereas a fourth biovar, *F. t. novicida*, exists in parts of the United States and possibly in Australia.

Francisella tularensis is transmitted by many blood-feeding arthropods other than ticks, including deer flies, mosquitoes, and fleas (Petersen et al., 2009). It is also transmitted by handling infected animals, inhalation of contaminated dust, drinking infected water, and eating insufficiently cooked, infected meat. Patients infected with *F. tularensis* experience fever, headache, and nausea, usually accompanied by development of an ulcerated lesion at the site of inoculation. Other clinical manifestations include enlargement of regional lymph nodes, pneumonia, and occasionally a rash. Although seven clinical types have been recognized, the ulceroglandular form accounts for about 80% of cases (Jellison, 1974). The pneumonic form is particularly prone to produce severe illness; if left untreated, it may persist for 2−3 months and become chronic thereafter.

Many species of ixodid ticks have been found infected naturally with *F. tularensis*. In North America alone, this agent has been detected in, or isolated from, at least 13 species of ixodids in four genera (one *Amblyomma* spp., five *Dermacentor* spp., two *Haemaphysalis* spp., and five *Ixodes* spp.). Ticks are considered by some workers to be reservoirs of *F. tularensis*, or at least part of a multi-host reservoir system, together with their primary vertebrate hosts. Transstadial passage of the agent occurs in susceptible ticks, but earlier claims that transovarial

transmission also occurs have not been confirmed. Ongoing studies on Martha's Vineyard, Massachusetts (USA) have demonstrated a wide diversity of *F. tularensis* genotypes in *D. variabilis*, suggesting long-standing enzootic transmission. All of the infected ticks harbored biovar *F. t. tularensis,* yet the infection rate was quite low (<1%) (Goethert et al., 2004). Recent studies have demonstrated that *A. americanum* and *D. variabilis* can both serve as bridge vectors for *F. tularensis* in the central United States and are most likely responsible for the tick-related cases observed in the region during the summer months (Mani et al., 2016).

Although tularemia is mainly an infection of wild lagomorphs and rodents, natural infections have been reported in numerous species of mammals (both domestic and wild) and birds. In the United States, a relatively high number of cases come from handling infected cats (bites, scratches, body fluids or tissues). In one study of 106 tularemia cases in Nebraska between 1998 and 2012, 48% of the cases were cat-associated (Larson et al., 2014). Fish, frogs, and toads have been found infected occasionally. Skunks and raccoons, both hosts of *D. variabilis*, have recently been identified as important sentinels for enzootic transmission in the eastern United States (Berrada et al., 2006).

Epizootics are spread mainly by water or ticks. Waterborne epizootics occur in western Siberia, where lakes and other fresh water habitats are contaminated by infected dead and dying muskrats and voles. Fur trappers and others who handled carcasses of infected water voles, muskrats, or water contaminated by them, have contracted the disease in large numbers. *Ixodes apronophorus* transmits *F. tularensis* among European water voles, furthering the spread of the disease along shorelines. Interestingly, recent studies have demonstrated that mosquito larvae can acquire *F. t. holarctica* from water and homogenates of 5 day old adult mosquitoes can infect mice (Bäckman et al., 2015).

In parts of North America, *F. tularensis* is acquired by hunters as they skin freshly killed rabbits. Lagomorphs were identified as the source of infection in over 80% of the cases of tularemia acquired in California between 1927 and 1951; of these, jackrabbits were implicated in 71% of the cases in which a distinction was made between jackrabbits and cottontail rabbits. On the other hand, ticks are much more significant than lagomorphs as a source of human infection in the south-central United States, especially in Arkansas and Missouri, where the lone star tick (*Amblyomma americanum*) is the primary vector (Mani et al., 2016). Since 1990, 41% of the tularemia cases in the United States have been reported from this region, with the cases involving *A. americanum* nymphs and adults and *Dermacentor variabilis* adults (Eisen, 2007).

Tick Paralysis

Tick paralysis is a host reaction to compounds secreted in the saliva of feeding ticks. This malady has been reported from North America, Europe, Asia, South Africa, and eastern Australia. It was first reported in Australia in 1824 by William Howell who described a tick "which buries itself in the flesh and would in the end destroy either man or beast if not removed in time" (cited in Harwood and James, 1979).

The affliction is characterized by a progressive, flaccid, ascending paralysis. In humans, it usually begins in the legs with muscle weakness and loss of motor coordination and sensation. Paralysis gradually extends to the trunk, with loss of coordination in the abdominal muscles, back muscles, and eventually the intercostal muscles of the chest. Paralysis of the latter may lead to death from respiratory failure. During advanced stages, the patient may be unable to sit up or move the arms and legs; chewing, swallowing, and speaking may become difficult. The condition progresses rapidly, and death may ensue within 24–48 h after onset of symptoms. In North America, the case-fatality rate is about 10% in the Pacific Northwest; most of those who die are children (Gregson, 1973). In Washington state alone, 33 cases were reported between 1946 and 1996, mostly in children less than 8 years old (Dworkin et al., 1999). In a 1-week period in May 2006, four cases of tick paralysis were reported from Colorado (CDC, 2006). In most North American cases, symptoms abate within hours following detection and removal of the attached tick or ticks, and recovery may be complete within 48 h. If paralysis has progressed too far, complete recovery may take up to 6 weeks. In contrast, paralysis induced by the Australian tick, *Ixodes holocyclus*, may worsen after tick removal, and full recovery may take up to several weeks (Grattan-Smith et al., 1997).

The nature of the toxin(s) causing tick paralysis has not been determined for most species of ticks that cause this condition. Intensive research on the salivary components of *I. holocylcus* has revealed the existence of a protein, named **holocyclotoxin**, which produces paralytic symptoms. A different salivary-gland protein has been implicated as the toxin in *D. andersoni* females.

Typically, only female ticks can induce tick paralysis. In southern Africa, however, nymphs of the **Karoo paralysis tick** (*Ixodes rubicundus*) can cause paralysis in laboratory rabbits, and larvae of the soft tick *Argas walkerae* produce a toxic protein that can paralyze chickens. Female ticks must be attached to the host for several days (usually 4–6) before they begin secreting the toxins. For excellent reviews of the mechanisms of pathology among the tick paralyses, see Gothe et al. (1999) and Mans et al. (2004).

Seventy-three species of ticks in 10 genera reportedly cause tick paralysis in humans, other mammals, and birds (Durden and Mans, 2016). Worldwide, the ticks of greatest concern for humans are *I. holocyclus, I. rubicundus*, *D. andersoni,* and *D. variabilis*. *Dermacentor andersoni* populations from various regions in Canada differ in their paralyzing ability. Apparently this is under genetic control, as selection can increase this ability in the laboratory (Lysyk and Majak, 2003).

Although reported from many regions in North America, tick paralysis in humans and domesticated animals has been documented most often from the Pacific Northwest in the United States and British Columbia in Canada. Cases in humans and other animals occur in spring and early summer, coincident with the activity period of adult *D. andersoni*. In the eastern United States, cases in dogs and humans are caused by *D. variabilis*, whereas in California this tick has been associated only with paralysis in dogs.

In contrast to paralysis produced by *Dermacentor* species or *I. holocyclus*, in which feeding by one female is sufficient to cause this condition, severity of the disease in South Africa is directly related to the number of attached *I. rubicundus*.

In England and France, human paralysis has been attributed infrequently to *I. hexagonus*. In California, several mild cases have been ascribed to *I. pacificus* prior to the 1950s, but these earlier observations were not well documented and no new cases in humans have been reported since then.

Tick-Bite Allergies

The bites of many species of ticks can cause host reactions other than paralysis. These range from minor, localized, inflammatory reactions that subside soon after tick removal to severe, systemic reactions involving skin rash, fever, nausea, vomiting, diarrhea, shock, and death. Severe toxic or allergic reactions may follow the bites of the soft ticks *Argas brumpti, A. reflexus, Ornithodoros coriaceus, O. moubata,* and *O. turicata.* In Europe, severe reactions and even loss of consciousness have occurred following attacks by the pigeon tick, *A. reflexus*, which infests buildings where pigeons roost. This tick is particularly prone to bite people if the birds have been driven away. In the far-western United States, reports that bites of the **pajaroello tick** (*O. coriaceus*) are more feared than those of rattlesnakes seem exaggerated, but severe allergic reactions have been documented (characterized by edema, pain, erythema, tissue necrosis, ulceration, and prolonged healing). Bites of the bat tick, *Carios kelleyi*, in Iowa induced large erythematous lesions in some individuals (Gill et al., 2004).

Attached *Ixodes pacificus* sometimes causes severe allergic reactions and, in rare cases, anaphylactic shock in persons previously sensitized to its bite (Van Wye et al., 1991). Likewise, individuals bitten by *I. holocyclus* may experience anaphylactic reactions involving tick-specific IgE.

Alpha-Gal Allergy

A novel food allergy to α-1,3-galactose, a mammalian oligosaccharide, was recognized in the early 2000s and has been termed alpha-gal allergy (also known as alpha-1,3-galactose allergy or red meat allergy) (van Nunen, 2015). Patients treated with the oncologic drug cetuximab had reported hypersensitivity reactions found to be associated with elevated levels of IgE to alpha-gal molecules. Occurrence of increased alpha-gal IgE was greater then expected in non-oncologic patients in the southern United States. Later, it was noted that the distribution of patients with the reaction was similar to that of the lone star tick, *Amblyomma americanum*. Weeks to months after a bite by *A. americanum*, a hypersensitivity reaction might occur following ingestion of red meat from nonprimate mammals (primarily beef, lamb, or pork). As case reports and more structured studies were published, the reaction to ingestion of red meat following a known tick bite appeared to be both temporally associated and dose-dependent. IgE to the alpha-gal epitope develops after bites from the lone star tick. The food allergy manifests generally 3−6 h after ingestion of red meat in both adults and children. The clinical signs usually appear as gastrointestinal symptoms and hives, but can range from chronic urticaria to life-threatening anaphylaxis.

While most patients have been identified in the United States, the allergy has now been reported from multiple countries and associated with additional tick species in those regions (e.g., *Ixodes ricinus* in Europe, *Ixodes holocyclus* in Australia, *Haemaphysalis longicornis* in Asia, and *Amblyomma sculptum* in South America) (Araujo et al., 2016). The epitope has been identified in whole tick homogenates, midgut homogenates, and tick saliva. Patients with B-negative blood groups may be more susceptible (Hamsten et al., 2013). This tick-associated allergy is an emerging phenomenon and deserves further study as to what factors influence development of this atypical allergy and how the reactions might be prevented.

VETERINARY IMPORTANCE

Ticks are of veterinary concern mostly because of the many microbial disease agents they can transmit to livestock, companion animals, and wildlife. Ticks also are important because of the debilitating and sometimes fatal host reactions produced in domesticated livestock and companion

animals as a result of the feeding activities of certain species (e.g., tick toxicosis, tick paralysis). Moreover, livestock, as well as wildlife, may suffer from exsanguination, leading to anemia and death. In Oklahoma and Texas (USA), for example, significant mortality in white-tailed deer fawns has been associated with heavy infestations of *Amblyomma americanum*. Livestock breeds in tick-infested areas have been subjected to natural selection and are able to acquire immunity to infestation upon exposure to local tick species. Exotic livestock breeds from tick-free regions, have no such immunity when first imported and do not acquire a high degree of immunity on exposure.

Livestock and poultry that are heavily infested with ticks may experience economically significant reductions in body weight, milk or egg production, and general unthriftiness, and may on occasion even die of anemia when infested by large numbers of ticks. Some species routinely or incidentally invade the auditory canal of bovines or other mammals, a condition known as otoacariasis, which may be accompanied by serious secondary bacterial infections. Tick bites, especially by species with a long hypostome (e.g., *Amblyomma*) may generate abscesses by secondary bacterial infection and cause the loss of one or more quarters of the udder. Other consequences can be lameness, due to ticks attached in the interdigital space and wounds that attract myiasis-producing flies, causing significant reduction in the value of the hide, fleece, or carcass.

The following section provides an overview of the major tick-borne diseases of veterinary importance.

Piroplasmoses

Piroplasmoses are protozoan diseases caused by *Babesia* (Family Babesiidae) (Fig. 27.28) and *Theileria* (Family Theileriidae, which also includes the genus *Cytauxzoon*). Parasites of both genera live and multiply in erythrocytes of the vertebrate host. The term itself refers to the developmental stage of the protozoan in the erythrocytes, i.e.,"piroplasm," literally meaning a pear-shaped structure. This is the stage infective to ticks. In ticks the parasite undergoes a sexual cycle with macro- and microgametes developing in the midgut and ending with infective sporozoites in the salivary glands.

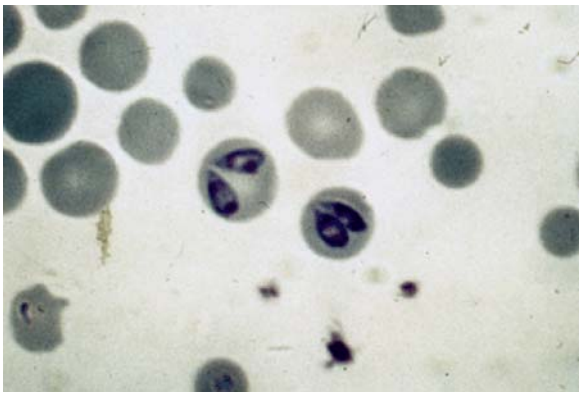


FIGURE 27.28 *Babesia* species in the blood of an infected animal. *Courtesy of Gerrit Uilenberg.*

Some piroplasm species were formerly classified as *Babesia*. However, after it was discovered that they undergo schizogony during development in vertebrate hosts and that they are not transmitted transovarially, they were moved to other genera (Uilenberg, 2006). Examples include *Babesia equi* and *B. microti*. *Babesia equi* is now classified as *Theileria equi* (Mehlhorn et al., 1986), whereas, based on phylogenic studies using molecular comparisons, *B. microti* has been placed between the families Babesiidae and Theileriidae (Kjemtrup et al., 2006).

Babesia and *Theileria* species exhibit basic differences in their developmental biology and ability to be transmitted by infected ticks to their offspring. Whereas *Babesia* (sensu stricto) is transmitted both transstadially (i.e., from one developmental stage of the tick to the next) and transovarially (from mother to offspring via her eggs), *Theileria* is transmitted only transstadially. As a result, *Babesia* persists in infected ticks throughout their development as larvae, nymphs, and adults; unfed larvae of the next generation are already infected when they hatch from the eggs. In contrast, *Theileria* does not persist into the next generation. Unfed tick larvae are never infective. Nymphs and adults become infective only if they feed on an infected vertebrate host or if the tick was infected in its previous developmental stage.

There are also genus-specific differences between these piroplasms in their vertebrate hosts. *Babesia* develops and multiplies only in erythrocytes (Fig. 27.28), whereas *Theileria* develops and multiplies in both erythrocytes and lymphoid cells. The latter first undergoes multiplication (schizogony) in lymphocytes, producing macroschizonts (i.e., schizonts containing large nuclei, also known as Koch's blue bodies), which appear a few days after the onset of symptoms. Later, as infected lymphocytes transform to lymphoblasts, microschizonts appear in the lymphoblasts. Merozoites released from the lymphoblasts then invade host erythrocytes. Piroplasms of both genera multiply in the eyrthrocytes by budding, with *Babesia* usually producing two daughter cells and *Theileria* usually four.

Babesioses

Infections of cattle by *Babesia* species is often severe, especially when involving *B. bovis* or *B. bigemina* (in tropical and subtropical regions) and *B. divergens* (in Europe and parts of North Africa). The severity depends on the susceptibility of the animal (natural resistance) and its

immune status (acquired resistance). Animals develop high temperatures, cease feeding, and become anaemic. One of the more characteristic and common symptoms is hemoglobinuria, causing the urine to become red or brownish, hence the name "redwater" for this disease. Icterus (jaundice) is often seen in severe cases. As babesiosis progresses, the animals become lethargic and eventually lapse into a coma and may die. Central nervous symptoms are often seen in infections by *B. bovis*. Considerable variation in severity has been noted in different geographical regions. This is attributed to differences in virulence among the parasites, as well as variation in the susceptibility of different cattle breeds. Nursing animals are protected by passive immunity acquired from antibodies in the colostrum of immune cows, resulting in young animals being more resistant than older ones. When all young animals in a population are infected, there is typically little or no clinical disease, particularly in local breeds. This situation is called **endemic stability**. Endemic stability may also occur in theilerial and ehrlichial infections.

The most important vectors of **bovine babesiosis** are *Rhipicephalus* (previously *Boophilus*) species especially *R.* (*B.*) *microplus* and *R.* (*B.*) *annulatus.* Although eradicated from the United States in the early decades of the 20th century, *R. annulatus* and *R. microplus* still occur in Mexico, and occasionally in extreme southern Texas near the Mexican border, and stringent controls are maintained to prevent their reintroduction. Moreover, white-tailed deer are hosts for *R. annulatus* and *R. microplus* and serve as a wild reservoir for these vector ticks and possibly the *Babesia* species. *Babesia divergens* is transmitted by the tick *Ixodes ricinus* in Europe and North Africa. In eastern, central, and southern Africa, *R.* (*B.*) *decoloratus* is an important vector (but only of *B. bigemina*), with *R.* (*B.*) *geigyi* replacing it in West Africa. Domestic buffalo, or water buffalo, may also contract babesiosis, with *B. orientalis* having been described recently from buffalo in China.

Dogs are often victims of **canine babesiosis**, caused primarily by large (*B. canis, B. rossi,* and *B. vogeli*), and small (*B. gibsoni,* and *B. conradae)* forms. The brown dog tick (*Rhipicephalus sanguineus*) (Fig. 27.12) is a widespread vector, whereas, *Dermacentor reticulatus* is an important vector in Europe, and ticks of the *Haemaphysalis leachi* group are known to transmit agents of canine babesiosis in Africa.

Babesiosis also affects other animals, such as small ruminants and horses (*Babesia caballi*) (Schetters and Brown, 2006; Scoles and Ueti, 2015). In the latter case, this disease is of particular concern regarding the international movement and commerce of horses. Wild and domestic cats in southern Africa have been identified as infected with *B. felis*, *B. leo*, and *B. lengau* (Bosman et al., 2007, 2013). The tick vectors of these pathogens are unknown. Wild mammals are commonly infected, usually with host-specific *Babesia* species.

Immunization against bovine babesiosis is carried out in some countries by injecting attenuated strains of the parasites, produced in donor cattle, with all the inherent risks associated with live vaccines. While tick control is emphasized, there also are commercially available inactivated vaccines against canine babesiosis, produced in blood cultures. Their efficacy, however, has not yet been well evaluated, while the existence of several species and/or strains further complicates their practical use.

Theilerioses

Theileria species infect a wide range of domestic and wild animals, particularly in the Old World. The more important agents of veterinary interest are *T. annulata* and *T. parva* in cattle, *T. equi* in horses, *T. lestoquardi* in sheep, and *Cytauxzoon felis* in domestic cats.

East Coast Fever (ECF) is a disease of cattle and domestic buffalo caused by *Theileria parva.* The disease, which has been known from East Africa since the 19th century, is widespread in eastern, central, and southern Africa. Movement of cattle has played a major role in the periodic outbreaks of ECF during the 20th century. Epizootics with high mortality tend to occur when very susceptible exotic breeds (e.g., taurine breeds, but even Asian zebu breeds) are introduced into areas endemic for *T. parva*. In endemic areas there may be a situation approaching endemic stability in local breeds.

An estimated 25 million cattle are at risk for acquiring ECF. Infected animals develop enlarged lymph glands and, after a few days, develop a high fever, become listless, and stop feeding. This may be followed by diarrhea and mucous discharges from the eyes and nose, and frequently by pulmonary signs, due to edema of the lungs. Mortality may exceed 90% in adult animals, but is usually much less in calves.

Theileria parva can also causes severe illness in some cattle, called **Corridor disease**. This results from transmission of the parasite from wild buffalo, the primary host of *T. parva*, to domestic cattle by ticks. It is believed that classical ECF has evolved from adaptation of the parasite to tick transmission between cattle. Water buffalo are also highly susceptible to ECF, but there are very few of them in endemic regions of Africa.

The primary vector of *T. parva* is *Rhipicephalus appendiculatus*, a tick whose geographical distribution coincides largely with that of ECF throughout much of eastern, central, and southern Africa. Corridor disease also occurs outside the known range of *R. appendiculatus* but within that of another competent vector, *Rhipicephalus zambeziensis*.

Another, somewhat milder disease of cattle is **tropical theileriosis**, caused by *Theileria annulata*. It is transmitted by several *Hyalomma* species (e.g., *H. anatolicum* in Eurasia) and affects both domestic cattle (*Bos* spp.) and the Asian domestic buffalo. Although clinical signs are similar, this disease differs from babesioses in the absence of hemoglobinuria and the less severe anemia that it causes in infected animals.

Tropical theilerioses is arguably of even greater importance than ECF because of its much wider distribution throughout North Africa, northern parts of sub-Saharan Africa, southern Europe, the Middle East, and elsewhere in central Asia, India, and China. In this regard the name of the disease is a misnomer because it also occurs in some temperate regions. Similarly, its other name, **Mediterranean theileriosis**, is misleading, as it occurs outside the Mediterranean basin. Other bovine-related theileriosis affecting large ruminants include *T. mutans* (Benign theileriosis), *T. taurotragi* (Benign African theileriosis), *T. velifera*, *T. orientalis* (oriental theileriosis), *T. buffeli*, *T. sinesis* and *T. sergenti*.

Immunization against tropical theileriosis and East Coast fever of cattle is used in some countries. In the case of tropical theilerioses, live attenuated, schizont-based vaccines, produced in lymphoblastoid cell cultures against *T. annulata*, have provided satisfactory results. However, similar vaccines for ECF have not proved effective. Instead, immunizations entail injection of live, fully virulent sporozoites obtained from ticks, followed by specific treatment to prevent clinical disease. Although homologous immunity is excellent, antigenic diversity complicates the results in the field.

Many other *Theileria* spp. cause infection, and often disease, in other livestock and wildlife in different regions of the world. Like *Babesia caballi*, *Theileria equi* is of significant concern to horse owners and a major obstacle to the international movement of horses (Scoles and Ueti, 2015; Wise et al., 2013). In small ruminants, particularly sheep, *Theileria lestoquardi* (**malignant theileriosis**) can be a serious problem, causing fatalities where it occurs in the Old World. Additionally, recent discoveries of new species of pathogenic *Theileria* in small ruminants (*T. luwenshuni* and *T. uilenbergi*) which cause cervine theileriosis, have been made in China.

Domestic cats are prone to suffer from a theilerial disease called **feline cytauxzoonosis**, caused by *Cytauxzoon felis*. It is closely related to *Theileria* and in the United States is often fatal in untreated or naive animals. While originally thought to be transmitted by *Dermacentor variabilis,* the primary vector is now considered to be *A. americanum*. The bobcat (*Lynx rufus*) and domestic cats may be the primary hosts of *C. felis.* The disease course is rapid, with onset of fever, lethargy, and anorexia after an incubation period of 5−20 days. Death often occurs within a week of onset. Leukocytosis, hemolytic anemia, icterus, and elevated hepatic enzymes are often seen on laboratory studies. Other species of *Cytauxzoon* occur in wild cats (Felidae) in various parts of the world (Meinkoth and Kocan, 2005).

Louping Ill

Although known to sheep herders in Scotland for centuries, louping ill (LI) was not recognized as a separate clinical entity until 1913. Its viral etiology was not established until 1931. Louping ill virus causes an economically important disease of sheep and red grouse (a game bird) in England and Scotland. It also occasionally infects cattle, horses, pigs, and humans, often with severe or fatal consequences. The causative agent is a flavivirus that is antigenically similar to other members of the **Tick-borne encephalitis complex**, and the only member of the complex present in the British Isles. Similar diseases occur sporadically in a few other European and Scandinavian countries. The latter are caused by viruses distinguishable from LI virus by nucleotide sequencing. They have been named according to the country in which they were first recognized, such as Spanish sheep or goat encephalomyelitis and Turkish/Greek goat encephalomyelitis (Gritsun et al., 2003).

Infected sheep lose their appetites and become feverish. On about the fifth day after onset, the fever rises and the animals become uncoordinated and develop tremors. Seriously ill animals walk with an awkward, erratic, "louping" gait, hence the name of the disease. Many sheep die shortly after locomotor signs appear, but others develop a chronic condition that may persist for several weeks. Mortality in different breeds of sheep varies considerably, reaching 100% in some susceptible flocks. Recovered animals often show signs of permanent neurologic damage. Experimentally infected red grouse experience mortality rates of up to 78%.

Historically, the disease has been most prevalent in areas of unimproved pastures and moorlands where *Ixodes ricinus* is abundant. LI is passed transstadially in ticks but not transovarially. Field and laboratory studies suggest that the vector competence of *I. ricinus* for the virus is not high. In enzootic areas of northern Britain, for example, only 0.1%−0.4% of ticks are infected with the LI virus.

Although *I. ricinus* parasitizes many vertebrate hosts, tick populations inhabiting sheep rangeland are supported principally by this animal. Small mammals are not abundant on many upland sites grazed by sheep and, when present, tend to support low tick loads. This and other limited evidence suggest that small mammals are not infected with LI virus. Moreover, sheep exposed to LI virus develop high viremias and are infective to ticks for several days. Other vertebrates, both domestic and wild,

that may contribute to the maintenance of the virus are cattle, goats, mountain hares, and several species of ground-inhabiting birds, especially red grouse, willow grouse, and ptarmigan. These species serve as hosts for *I. ricinus* and occasionally develop viremias high enough to infect feeding ticks. Two other experimentally proven routes of transmission may amplify LI virus in natural foci: nonviremic transmission of the virus among co-feeding infected and uninfected ticks on mountain hares, and the ingestion of infected ticks by red grouse during their first season (Gilbert, 2016). It has been estimated from field observations that 73%–98% of viral infections in young-of-year red grouse might occur as a result of eating infected ticks.

African Swine Fever

This disease was first recognized in Kenya in 1921, as a catastrophic illness that killed 99% of infected pigs. Since then, sporadic epidemics of African swine fever have been reported from many African countries south of the Sahara, in Europe, and in the western hemisphere in Cuba, Haiti, the Dominican Republic, and Brazil.

The disease is caused by a large icosahedral DNA virus (family Iridoviridae) that attacks cells of the reticuloendothelial system, especially monocytes. The host range of the virus is limited to domestic pigs, European wild boars, warthogs, and bush pigs, all species of the family Suidae. In its acute form, animals develop fever about 3 days postinfection, the fever persists for three or 4 days, the temperature drops, and death ensues. In its subacute form, an irregular fever lasts for three or 4 weeks, whereupon the animals either recover or die. In its chronic form, animals may survive for long periods before succumbing to a secondary illness, most commonly pneumonia. Chronically infected animals usually experience stunted growth and emaciation, and serve as long-term reservoirs of the virus.

The primary vectors in Africa are *Ornithodoros* spp. of the *moubata* group, particularly *O. porcinus* (Jori et al., 2013). These ticks occur in eastern, central, and southern Africa. In these regions there is a sylvatic cycle between ticks and wild Suidae, particularly the warthog, in which the cases of infection show no symptoms. Once the infection has passed to domestic pigs, it spreads as a contagious disease and has been transported as such to various parts of the world. In north and west Africa, ticks in the *Ornithodoros erraticus* group are the primary vectors. These ticks spread to the Iberian Peninsula (Spain and Portugal) where ASFV became established in local *Ornithodoros erraticus* populations, which made eradication a difficult and lengthy affair. In West Africa, the tick *Ornithodoros sonrai*, also in the *O. erraticus* group, is suspected of playing a role in the persistence of the virus. In the New World, *O. coriaceus*, *O. turicata,* and other *Ornithodoros* species have been demonstrated to be competent experimental vectors, raising concern about the potential establishment of African swine fever virus in North America. It is a porcine disease of major global importance, because of the high mortality it causes and the absence of a vaccine, despite numerous attempts to develop one.

Diseases Caused by Members of the Family Anaplasmataceae

The taxonomy of the Family Anaplasmataceae has been stable since 2001. The genera *Anaplasma*, *Ehrlichia* (including the former *Cowdria*), *Neorickettsia*, and *Wolbachia* have been united in the family Anaplasmataceae, to which the genus *Aegyptianella* has been added. The genus *Neoehrlichia* has also been proposed, but not formally described.

Ehrlichioses and Anaplasmoses

The diseases caused by members of the genera *Ehrlichia* and *Anaplasma* have long been known in veterinary medicine. These pathogens infect the leukocytes or platelets of livestock, companion animals, and wildlife. The ehrlichiae grow as distinct microcolonies, or **morulae**, within the cytoplasm of host cells (Fig. 27.24). The disease manifestations caused by these agents can range from asymptomatic to fatal.

Canine ehrlichiosis, due to *E. canis*, was first recognized in 1935. The cosmopolitan distribution of this pathogen corresponds with that of its primary vector, *Rhipicephalus sanguineus*. *Ehrlichia canis* occurs in mononuclear cells, but it is often difficult to find in blood smears. In the United States, serosurveys of civilian and military dogs have revealed that the disease is present in most states. Dogs infected with *E. canis* develop fever, conjunctivitis, and swelling of various tissues. The disease causes a reduction in the numbers of all blood cells (red, white, and platelets), and thus is also referred to as **canine tropical pancytopenia**. Infected animals stop eating, lose weight, and frequently appear depressed. Acute infection is often followed by a debilitating chronic phase, accompanied by anemia and sometimes hemorrhagic nasal bleeding. The German Shepherd breed is particularly susceptible to acute severe illness. These animals suffer from low white-blood-cell counts and damage to the lymph glands, bone marrow, and spleen. Animals with severe infection usually die without antibiotic treatment. In other breeds, particularly mongrels, the disease is often milder and ranges from asymptomatic to chronically symptomatic.

Transstadial transmission of *E. canis* occurs in *R. sanguineus*, and transstadially infected nymphal or

adult ticks can transmit *E. canis* to susceptible dogs. Transovarial transmission occurs rarely, if at all. Dogs apparently serve as the primary reservoir *of E. canis* because inapparent infections can persist for over 5 years, and the agent is continually present in chronically infected dogs.

Infection by *E. ewingii* causes a disease in dogs known as **canine granulocytic ehrlichiosis**. The pathogen grows within neutrophils of infected animals. The infection is usually mild and may manifest as polyarthritis. Dogs can maintain infections with this pathogen for over 2 years during which they can serve as reservoir hosts for the maintenance of the pathogen in local communities (Starkey et al., 2015). Although its actual distribution may be wider, *E. ewingii* is primarily found in the south-central and southern United States (Beall et al., 2012). White-tailed deer may serve as a natural wildlife reservoir. The agent is passed from *A. americanum* nymphs to adults, and transstadially infected adults can transmit the infection to susceptible dogs (Anziani et al., 1990). Naturally infected lone star ticks have been identified in North Carolina and elsewhere (Wolf et al., 2000). *Ehrlichia ewingii* is morphologically indistinguishable from *A. phagocytophilum* in blood smears, and molecular assays provide confirmation of infection. In the south-central and southeastern United States, dogs can also become infected with *E. chaffeensis*, the agent of human monocytotropic ehrlichiosis, when fed upon by an infected *A. americanum* tick. On its own, *E. chaffeensis* produces only a mild disease in dogs. However, coinfections with either *E. ewingii* or *E. canis* can produce more severe outcomes.

Recently, a new *Ehrlichia*, *E. muris eauclairensis*, was detected in both people and dogs in the upper midwestern United States. The blacklegged tick, *I. scapularis*, has been identified as the primary vector in experimental-infected and field-collected ticks. Little is known regarding the ecology or the effect of this pathogen on dogs except for one case in which the dog experienced fever, decreased appetite, lethargy, and recurrent vomiting (Hegarty et al., 2012).

Heartwater

Heartwater is an ehrlichial disease of large ungulates (livestock and game) caused by *Ehrlichia* (formerly *Cowdria*) *ruminantium* (Fig. 27.29). The disease occurs primarily in sub-Saharan Africa and neighboring islands (e.g., Madagascar, Reunion Island, Mauritius, the Comoros, and São Tomé). It has been inadvertently transported to the western hemisphere where it now occurs on several Caribbean islands. It is one of four most important tick-borne cattle diseases in tropical regions (babesioses, theilerioses, anaplasmosis, and heartwater).

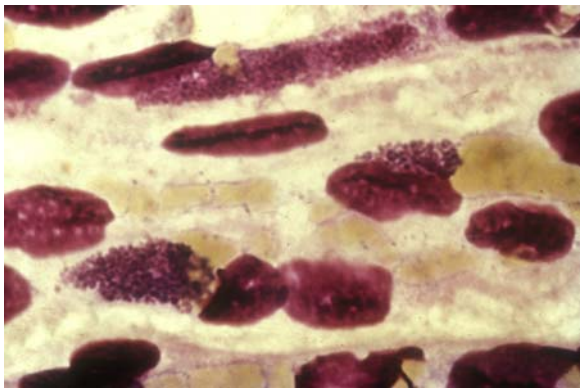


FIGURE 27.29 *Ehrlichia ruminantium* is seen as three masses of organisms in brain tissue of a goat that had died of heartwater. *Courtesy of Gerrit Uilenberg.*

Heartwater affects all domestic ruminants, especially cattle, sheep, and goats. Domestic buffaloes are also highly susceptible, but they are almost absent in endemic regions. As in several other tick-borne diseases, local breeds are much less susceptible than exotic ones, and endemic stability may occur, particularly in cattle. However, even local breeds of small ruminants may suffer considerable losses.

Infected animals develop fever, and after a few days, develop central nervous system signs. They may become disoriented, and show signs of motor disorder, especially abnormal walking, trembling, and muscle twitching. In addition, cattle may develop profuse diarrhea. As the illness progresses, they develop convulsions and die soon afterwards. Dead and dying animals commonly show a massive accumulation of fluids in the membrane surrounding the heart (pericardium) and edema in the lungs and other organs. Surviving animals become immune. Ruminants which appear healthy can actually be carrying *E. ruminantium* at low levels which can be infective to ticks for up to a year (Allsopp, 2010). Additionally, ticks in heartwater endemic areas have very low infection rates (1%−7%) which increases the difficulty of elimination of the pathogen once it is established in a particular region. Calves are protected by a short period of age-dependent tolerance, and possibly to some extent also by maternal antibodies transferred in milk. As a result, infected calves often develop only mild illness, or none at all. The disease is quite severe in exotic breeds of sheep and goats, with Angora goats being a most susceptible breed. Introduction of exotic livestock disrupts endemic stability and often leads to epidemics of the disease. Similarly, rapid resurgences of vector tick populations following drought also can lead to devastating epidemics. Moreover, endemic stability can be disrupted by excessive use of pesticides, which destroys the natural herd immunity that results from constant, low-level challenge by small numbers of infected ticks.

In ruminants, multiplication of *E. ruminantium* is observed in endothelial cells, which is easiest seen in the capillaries of brain-cortex smears (Fig. 27.29). However, its presence in circulating neutrophils in the blood has also been demonstrated.

The agent of heartwater is transmitted by at least 10 African species of *Amblyomma* ticks, most of which have indiscriminant feeding habits. In view of its enormous geographic range and adaptability to varying climatic conditions, the tropical bont tick, *A. variegatum* is the primary vector in most enzootic areas of Africa and the Caribbean. The bont tick, *Amblyomma hebraeum* (Fig. 27.18), which is found in subtropical southern Africa, is also an important vector. Other *Amblyomma* species are important locally as vectors to livestock and wild ungulates. *Ehrlichia ruminantium* is maintained transstadially within populations of ticks, in which there is multiplication in the gut and later in the salivary glands. The pathogen is transmitted by the bite of the tick. Ticks remain infected for long periods, possibly for life. Transovarial transmission has been reported but appears to be exceptional.

There is considerable strain diversity, which complicates the development of a reliable and safe vaccine. Immunization is currently carried out, particularly in South Africa, by inoculating blood from a sheep reacting to a well-characterized stock (i.e., Ball 3). The injection has to be carried out intravenously, and the animals have to be closely monitored in order to treat them in good time. The procedure is risky from several points of view and is labor-intensive and far from 100% effective.

In the 18th or 19th century, the tick *A. variegatum* and the causal agent of heartwater were introduced into the Caribbean region with cattle from West Africa. The tick is, or has been, present on several islands of the Lesser Antilles, whereas heartwater is known to occur on Guadeloupe, Marie-Galante, and Antigua. It is likely that the tick is spread from island to island by another African immigrant, the cattle egret. Because of the constant threat of invasion of more islands and particularly the American mainland, an eradication program has been set up, but lack of continuity in financing and international coordination has led to intermittent abandonment of this program (Pegram, 2006). Some islands have been freed from the tick, but the constant migration of cattle egrets can easily result in reinvasion. Invasion of the North American or South American mainlands would be disastrous for the livestock industry, not only because of heartwater, but even more so because *A. variegatum* is associated with severe dermatophilosis. Three American species of *Amblyomma* ticks are experimental vectors of heartwater, one of them, *A. maculatum*, being an efficient vector. Additionally, white-tailed deer are susceptible hosts and could serve as potential reservoirs should heartwater become introduced to North America.

For further information about heartwater, see the reviews by Camus et al. (1996), Mahan (2006), and Allsopp (2010, 2015).

Granulocytic Anaplasmosis

Anaplasma phagocytophilum is an important pathogen of livestock and domestic animals (including dogs and cats), and also a cause of human granulocytic ehrlichiosis, an important zoonosis. In Europe, *Ixodes ricinus* transmits this rickettsia, the causative agent of **tick-borne fever** (also called **pasture disease**) to sheep, cattle, and goats. Reduced milk production, and abortions can occur in infected animals. The organism infects granulocytes (neutrophils and eosinophils) and induces a marked immunosuppression that may predispose animals to secondary infections (such as pyaemia by *Staphylococcus aureus* in lambs) and reduce their antibody response to vaccination against other diseases. *Anaplasma phagocytophilum* also causes infection and febrile illness in horses in the United States and has also been found in Europe. In the USA it is transmitted by *Ixodes pacificus* and *I. scapularis*. It may also cause a mild illness in dogs, in which it is morphologically indistinguishable from *Ehrlichia ewingii*. *Anaplasma phagocytophilum* is passed transstadially, but not transovarially, in the tick vector. Further review of this topic can be found in Dugat et al. (2015).

Other Anaplasmoses

Anaplasma platys infects canine platelets, causing their numbers to fluctuate over time. While primarily considered a canine pathogen, variants have been identified as causing disease in cattle and humans. This infection is mild and is often diagnosed as **canine cyclic thrombocytopenia**. The tick vector(s) of this species have not been determined, although *Rhipicephalus sanguineus* is often found to be infected. This species has been identified in the southern United States, Greece, Taiwan, and Japan and other locations.

In many tropical and subtropical regions, cattle and domestic buffalo can be infected with *A. bovis*, which is usually benign, whereas more pathogenic congeneric parasites occur in Central and West Africa, and Brazil. Known vectors belong to the genera *Hyalomma*, *Rhipicephalus* and *Amblyomma*. *Anaplasma bovis* develops in mononuclear cells, as does the closely related *Ehrlichia ovina* in small ruminants. Many other *Anaplasma* spp. and *Ehrlichia* spp. have been described, and more are being discovered every year. One example is *"Anaplasma capra"* identified from goats in China. It has been found to infect humans, although the agent has not been fully characterized after isolation into pure culture. The pathogenic potential of these newly recognized taxa may not be fully known, and further study is needed.

Erythrocytic Anaplasmosis

This section is limited to "classical" anaplasmosis, caused by rickettsial agents residing in red blood cells. Anaplasmosis was first described in South Africa in 1910 by Max Theiler, who also identified and named the primary agent 1 year later. This parasite, *Anaplasma marginale*, and two related species, sometimes considered to be subspecies or variants of it, (*A. centrale* and *A. ovis*), infect red blood cells of cattle and sheep throughout much of the world. Other described species either are not recognized as separate taxa or have no standing in taxonomic nomenclature. Anaplasmosis is now considered one of the most important diseases of livestock. The causative agent, *Anaplasma marginale*, is a pleomorphic, coccoid rickettsia that occurs and multiplies in membrane-bound inclusions called colonies in the cytoplasm of infected erythrocytes.

Disease onset is abrupt following a lengthy incubation period of 2−6 weeks. Common clinical manifestations include fever of several days duration, labored breathing, loss of muscle tone in the rumen, constipation, and hemolytic anemia, often followed by jaundice (Fig. 27.30). Animals usually recover when infected with strains of mild virulence, whereas 30%−50% of those infected with highly virulent strains may die. Moreover, severity of disease and case-fatality rates increases with age. Cattle that recover from acute anaplasmosis maintain a persistent, low-level parasitemia that protects them from reinfection; however, they constitute a reservoir of infection in the herd.

Approximately 20 species of ixodid ticks serve as vectors of *Anaplasma* species. The main vectors in subtropical and tropical countries are *Rhipicephalus* (*Boophilus*) ticks: *R.* (*B.*) *microplus*, *R.* (*B.*) *annulatus*, *R.* (*B.*) *decoloratus*, and probably *R.* (*B.*) *geigyi*. *Hyalomma* spp. and other *Rhipicephalus* spp. also play an important role. In North America, the primary vectors are *Dermacentor* species: *D. andersoni*, *D. albipictus, D. occidentalis*, and *D. variabilis*. In Europe, *D. reticulatus* and possibly *D. marginatus* appear to play a role. Blood-sucking flies in the family Tabanidae (horse flies and deer flies), and other blood-sucking insects, have been implicated as mechanical vectors. Mechanical transmission can also occur during vaccination and dehorning campaigns, when the same needles, syringes, or other instruments are used on several animals.

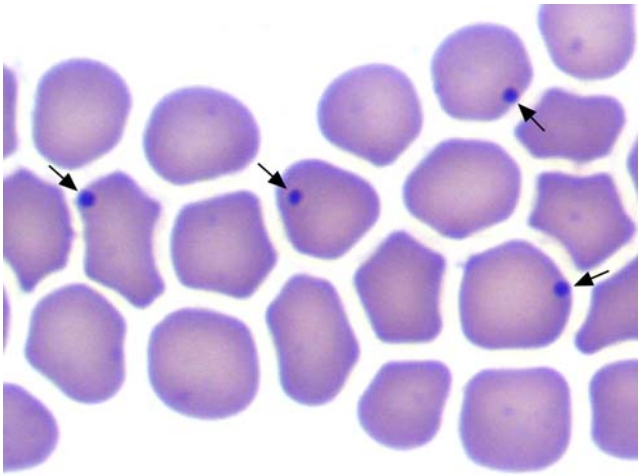


FIGURE 27.30 Blood smear showing *Anaplasma marginale* (arrows) in bovine red blood cells. *(Photograph by Susan M. Noh, U. S. Department of Agriculture).*

Anaplasma marginale undergoes a complex developmental cycle in ticks involving five morphological forms (Kocan et al., 2010). Details of the life cycle have been elucidated in *D. andersoni* and are presumably representative of the parasite's development in its other tick vectors. It has recently been shown that endosymbiont bacteria within the ticks may influence tick susceptibility for *A. marginale* infection (Gall et al., 2016). The genetic and morphologic characteristics of *Anaplasma* spp. and their development within ticks are similar to those of *Ehrlichia ruminantium* and other *Ehrlichia* spp. in their tick vectors. *Anaplasma marginale* is passed transstadially, but not transovarially, within ticks. *Dermacentor andersoni* females infected as nymphs begin transmitting the infection by the sixth day of feeding on a susceptible host, whereas male ticks that acquire infection as adults can transmit the pathogen within 24 h. Male ticks are of considerable importance as vectors because they feed repeatedly. They also readily transfer between hosts that are in close contact, and are therefore capable of transmitting *A. marginale* to multiple hosts. The transfer of ticks between individuals also explains why one-host ticks such as *Rhipicephalus* (*Boophilus*) spp. are vectors of *A. marginale*.

Cases occur frequently on farms located adjacent to tick-infested woodlands. In the eastern United States, the presence of white-tailed deer is considered a risk factor because this cervid is an important host of tick vectors. However, white-tailed deer are not competent reservoirs of *A. marginale* and therefore do not serve as a source of infection for noninfected ticks. In the western United States, mule deer are not only primary hosts but also competent **reservoirs** of *A. marginale*. Although national statistics are not available, prevalence rates of up to 20% are reported from states throughout the United States where tick vectors are present.

Immunization against bovine anaplasmosis is complicated by the existence of antigenic diversity, particularly when inactivated vaccines are used. In several countries the related rickettsia *A. centrale* is injected as a live vaccine, obtained from donor cattle, and gives a satisfactory degree of protection against anaplasmosis caused by *A. marginale*.

Borrelioses

Borrelioses are diseases of birds and mammals caused by spirochetes in the genus *Borrelia* (Fig. 27.31). Important tick-borne borrelioses include avian spirochetosis and Lyme disease. **Avian spirochetosis** is a highly fatal disease of turkeys, pheasants, geese, doves, chickens, and canaries in Europe, Africa, Siberia, Australia, Indonesia, India, and North, Central, and South America. It causes severe losses to the poultry industry in certain countries. Infected birds develop high fever, diarrhea, and become cyanotic. Birds that survive develop a long-lasting immunity. *Argas persicus,* and related ticks (subgenus *Persicargas*) transmit the etiologic agent, *B. anserina*, via infectious tick feces or by bite. *Borrelia anserina* is related to borreliae in the relapsing fever group. Transstadial passage and transovarial transmission occur, and ticks can remain infective for 6 months or longer.

Dogs, cats, cattle, horses, and possibly sheep can be infected with certain etiologic agents of **Lyme disease** (*Borrelia burgdorferi* s.l.). This disease, or related disorders, has been reported from numerous countries on five continents: Africa, Asia, Europe, North America, and South America. Earlier claims that *B. burgdorferi* is present in Australia have not been confirmed by recent field, sentinel, and laboratory studies. However, recent studies in various South American countries indicate the presence of Lyme-like illnesses (Baggio−Yoshinari syndrome in Brazil), some of which have been attributed to strains of *Borrelia* spp., including members of the *B. burgdorferi* sensu lato complex. Populations of domestic dogs living in areas highly endemic for *B. burgdorferi* can have seroprevalence rates as high as 90%, even though relatively few seropositive animals manifest overt clinical signs. Indeed, 25%−50% of apparently healthy dogs in some areas may have significant antibody titers to *B. burgdorferi*. In hyperendemic foci of the northeastern, northcentral and farwestern United States, the most commonly observed clinical manifestations among dogs are lameness, inappetence, fever, and fatigue. Dysfunction of the central nervous system, heart block (secondary to myocarditis), and a renal syndrome have been associated with *B. burgdorferi* infection in some dogs.

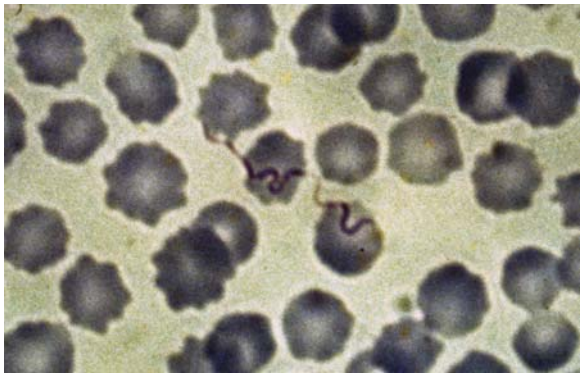


FIGURE 27.31 A *Borrelia* spirochete in the peripheral blood of an infected cow, Wright-Giemsa stain. *Courtesy of Gerrit Uilenberg.*

Cats reportedly are exposed to *Ixodes scapularis* infected with *B. burgdorferi* and can develop elevated antibody titers to this spirochete; however, the clinical significance of these findings is unknown. In northern California (USA), outdoor cats living in rural or semirural settings occasionally are bitten by *I. pacificus* females but apparently at a frequency much lower than that of dogs.

Lyme disease has been reported in cattle and horses in the northeastern and upper midwestern United States. Antibodies against *B. burgdorferi* have been detected in serum or synovial fluid in cattle exhibiting lameness or arthritis, but a causal relationship between exposure to such spirochetes and clinical disease in bovines has not been established. In some areas of the United States and Europe, serological studies have recorded prevalence rates of up to 48% in horses living in areas with known exposure to *B. burgdorferi*-infected ticks. Although arthritis, edema, and dermatitis have been observed in some animals, clinical illness attributable to *B. burgdorferi* infection apparently is uncommon in horses.

Bovine borreliosis is a benign disease of cattle, sheep, and horses that occurs in Africa and Australia (Burgdorfer and Schwan, 1991). Infected animals experience one or two attacks of fever, loss of appetite, weight loss, anemia, and weakness. The causative agent, *Borrelia theileri*, is transmitted by *Rhipicephalus* spp. and possibly other genera of ixodid ticks. This borrelia, along with the lone star agent ("*B. lonestari*") and *B. miyamotoi*, form a clade that is distinct from, but most closely related to, the relapsing fever group spirochetes. Recently, several relapsing fever bacteria, including *B. turicatae*, *B. hermsii*, and *B. persica*, have been reported as causing disease in dogs (Cutler et al., 2017).

Epizootic bovine abortion (EBA), also known as "foothill abortion," is a major disease of rangeland cattle in the far-western United States, particularly California. Since its first recognition as a distinct clinical entity during the 1950s, various microorganisms have been evaluated or proposed as potential etiologic agents. Recently, a novel delta-proteobacterium (*Pajaroellobacter abortibovis*) identified in thymus tissue from EBA-affected fetuses and detected in wild-caught *O. coriaceus* ticks by PCR was implicated as the etiologic agent (King et al., 2005; Brooks et al., 2016).

Tularemia

Tularemia, caused by the bacterium *Francisella tularensis* subsp. *tularensis*, is primarily a disease of rodents and

lagomorphs (rabbits and hares) in the Northern Hemisphere. Although best known for its public health importance, *F. tularensis* also is a veterinary pathogen that can cause devastating epizootics in domestic sheep. Reliable reports of epizootics in North America date back to 1923 when serious losses occurred in eastern Montana and southern Idaho (Jellison, 1974). These outbreaks shared several features: animals were put on rangeland enzootic for *F. tularensis* in early spring, they grazed in sagebrush areas where *D. andersoni* ticks were abundant, and they became heavily infested with ticks. *Dermacentor andersoni* is considered the principal vector *F. tularensis* in the western United States. As many as 50% of a sheep herd may become sick, and 10% may die within a few days. Diseased animals that survive such outbreaks may lose weight and condition during their illnesses. Although sheep usually are quite resistant to infection with *F. tularensis*, reduced vitality of animals after a long winter, shortage of feed, exposure to early spring storms, and heavy infestations of ticks predispose flocks to epizootics. Over 14,000 cases of tularemia in sheep were recorded in the United States between 1923 and 1945. In contrast, only about 40 cases were reported in sheep in Canada by 1945. Although outbreaks continue to occur, case numbers have dropped considerably due timely detection and effective control measures.

Epizootics of tularemia among sheep constitute a risk factor for humans. Jellison and Kohls (1955) presented records of 189 human cases of tularemia associated with the sheep industry in the United States. Of these, 66 cases occurred among sheep shearers. Other individuals found to be at particular risk were sheep owners, sheep herders, veterinarians, and spouses of sheep handlers.

Domestic cats appear to be more susceptible to *F. tularensis* subsp. *tularensis* than dogs. Infection occurs when a cat ingests an infected animal, the most common being rabbits. These infected cats, then, are able to transmit tularemia to their owners through bite, scratch, and contact of bodily fluids with the human's skin. In the central United States, feline tularemia cases peak between March and June and between September and November. These seasonal peaks may reflect what is actually happening in the definitive rabbit hosts and their relationship with tick vectors. The May/June peak coincides with the primary questing activity of *A. americanum*, which are important vectors. The September/November seasonal peak coincides with the activity cycles of two other potential tick vectors, *Haemaphysalis leporispalustris* and *I. scapularis*, both of which are known to feed on rabbits and reported with natural infections of *F. tularensis* (Mani et al., 2016). More research is needed to better understand the link between cats, rabbits, humans, and potential tick vectors.

Q Fever

Coxiella burnetii, the agent that causes Q fever, is widespread in populations of domestic livestock and wildlife. Infection in mammals other than humans, however, is typically benign. For that reason, this disease is discussed primarily in the section on public health importance. Nonetheless, *C. burnetii* is also a veterinary pathogen, which can induce abortion in pregnant cows and ewes. Goats are known reservoirs of *C. burnetii*, but recent research suggests their role in the natural maintenance of the disease is limited. It is interesting that the genotypes found in human endocarditis are associated with genotypes circulating in goats. Because infected animals excrete large numbers of *C. burnetii* in their waste and in birthing tissues and fluids, they pose a significant health risk for animal handlers. Infected ticks produce infectious feces that can contaminate the wool of sheep and become aerosolized when handled. However, for the general public, the danger of acquiring infection arises from inhalation of bacteria-laden dust as it becomes airborne. As noted earlier, *Coxiella*-like endosymbiotic bacteria are often found in ticks, but are not thought to infect animals.

Dermatophilosis

Dermatophilosis, also called **cutaneous streptothricosis**, is a skin disease of domestic and wild mammals, including occasional humans, and is caused by the bacterium *Dermatophilus congolensis*. It is especially economically important to cattle and sheep production. In sheep, the disease may be known as **lumpy wool**. The pathogen is transmitted from animal to animal by mobile zoospores. The infection is widespread, probably cosmopolitan, and normally benign. Transmission and the development of severe skin lesions (Fig. 27.32) following infection are favored by humidity and heat, and by the presence of certain *Amblyomma* spp., notably, the tropical bont tick

FIGURE 27.32 Senepol cattle on the Caribbean island of Nevis, affected by severe dermatophilosis. *Courtesy of Gerrit Uilenberg.*

A. variegatum. The role of this tick had long been suspected in Africa, and even earlier in the Caribbean region, where this African tick had been introduced.

The role of *A. variegatum* in this disease was experimentally confirmed by Walker and Lloyd (1993). Although *D. congolensis* is not introduced when the tick feeds, the saliva of attached *A. variegatum* adults influences the pathogenesis of dermatophilosis, causing severe dermatophilosis in ruminants when the tick is present. Dermatophilosis therefore is not a tick-borne disease but rather a tick-associated disease.

Host resistance to dermatophilosis varies significantly in different areas, with much higher resistance in local breeds than in exotic stock. The constant high humidity in the Antilles is particularly favorable for the transmission and development of dermatophilosis and also to the multiplication and survival of *A. variegatum*. The recent expansion of *A. variegatum* in Zimbabwe into the Highveld, the prime cattle production zone in the country, has challenged earlier notions of how arid conditions affect the spread of this disease. As a result of the significant reduction of tick control throughout the country, there are reports of herd infections rates up to 60%. While still associated with the rainy season, there are challenges for how to implement tick control in areas experiencing low resource challenges, particularly among exotic breeds.

Dermatophilosis is considered the most important cattle disease in some of the Caribbean islands, where it presents a greater concern than heartwater. In the 1980s, when cattle on many of the islands were newly exposed to *A. variegatum*, they were particularly susceptible to severe dermatophilosis (Fig. 27.32). On the island of Nevis, for example, losses of 75% of cattle to this disease were reported and farmers were forced to abandon their cattle-breeding operations (Hadrill et al., 1990). This is in contrast to the islands of Guadeloupe and Marie-Galante, where the tick was introduced from endemic areas of West Africa two or three centuries ago and "Creole" sheep became more resistant to infection. No vaccine is currently available, and the only prevention is intensive tick control. This poses a significant concern should *A. variegatum* be introduced to the American mainland.

Tick Paralysis

The first reports of tick paralysis in livestock originated in Australia in 1890 and in British Columbia, Canada, in 1912. This condition is most common in livestock and pets and causes injury or death to thousands of animals each year. Tick paralysis has been reported from many countries in North America, Europe, Asia, and Africa. There are at least 73 species of ticks that can cause paralysis; however, most studies have only focused on a relatively few (Mans et al., 2004; Durden and Mans, 2016).

In South Africa, the **Karoo paralysis tick** (*Ixodes rubicundus*) is thought to be responsible for annual losses of sheep and goats and a low percentage of game animals in some areas. Other animals affected include cattle and species of wild antelope. Induction of paralysis by *I. rubicundus* is directly related to the total number of ticks feeding on a host. Stock farmers regard tick paralysis as one of the most important problems affecting their operations. The disease occurs in hill rangeland or mountainous terrain covered with a "Karoo" type of vegetation, which is grassy areas interspersed with shrubs or trees. Although *I. rubicundus* parasitizes many wild mammals, only antelopes are known to develop paralysis.

Another tick that paralyzes sheep and goats in Africa, particularly in South Africa, although not as severely or as often as *I. rubicundus*, is *Rhipicephalus evertsi evertsi*. This tick has been recognized as a cause of tick paralysis since 1900. The induction, duration, and severity of the paralysis are related to the number of female ticks that have engorged to body weights of 15−21 mg. *Hyalomma* ticks have been implicated as causing paresis or paralysis of camels in the Sudan and Somalia.

In North America, three species of *Dermacentor* ticks cause paralysis in companion animals and livestock. In the eastern and western United States, *D. variabilis* is a common cause of tick paralysis in domestic dogs. In the Sierra Nevada foothills of northern California, for example, an average of six cases was seen in two veterinary practices during a 1-year investigation (Lane et al., 1984). Dogs were infested with a mean of 32 ticks; 98% were *D. variabilis* adults. Another tick from the same region, *D. occidentalis*, is responsible for occasional cases of tick paralysis in cattle, ponies, and deer, but not dogs.

The most important species of paralysis-inducing tick in North America is *D. andersoni*, which was responsible for paralyzing sheep and cattle in the Pacific Northwest (British Columbia, Washington, Idaho, and Montana. Individual outbreaks have involved up to 320 animals with cases occurring most frequently from April to June when adult *D. andersoni* activity is greatest.

In Australia, *Ixodes holocyclus* induces paralysis in dogs, cats, horses, and humans. This tick inhabits a narrow zone along the eastern coast of Queensland and Victoria. Drugs administered along with hyperimmune serum to dogs with advanced paralysis improve chances for full recovery. Among several drugs tested, phenoxybenzamine hydrochloride, an alpha-adrenergic-blocking agent, has been found to be most effective.

In Europe and Asia, tick paralysis is widely scattered. In Macedonia and Bulgaria, paralysis in sheep, goats, chickens and cattle has been attributed to *Haemaphysalis punctata*, and in Crete, the former Yugoslavia, and the former Soviet Union, livestock are sometimes paralyzed by

bites of *I. ricinus.* Recently, *R. sanguinius* has been linked with tick paralysis in dogs in Italy.

Birds are also susceptible to tick paralyses. Larvae of *Argas walkerae* induce paralysis in chickens in South Africa. A toxic fraction isolated from replete larvae of this argasid tick consists of two proteins having "membranophilic" properties and molecular masses of 32 and 60 kDa; extracts containing these proteins induced paralysis in 1-day-old chicks. In the southeastern United States, a number of species of wild birds, especially passeriforms, are paralyzed by attached *Ixodes brunneus* females. Single adult ticks can render a bird incapacitated and can lead to death from exposure and predation.

General reviews of tick paralysis and tick toxicosis (see later) have been given by Gothe (1999), Mans et al. (2004), and Durden and Mans (2016).

Tick Toxicoses

Toxic reactions have been associated with the bites of certain species of ticks, notably, argasids. In Africa, cattle bitten by *Ornithodoros savignyi* may die of toxicosis in just 1 day. Sheep attacked by *O. lahorensis* in eastern Europe and in the southern region of the former Soviet Union may tremble, gnash their teeth, exude frothy saliva, experience paralysis, and sometimes perish. In Brazil, *Ornithodoros brasiliensis* has been broadly implicated in human and canine cases of tick toxicosis. In Europe, *Argas persicus* can cause leg weakness in ducks and geese; this condition resembles a true toxicosis and not a paralysis. A toxic illness that affects mainly calves in large areas of central, eastern, and southern Africa is known as **sweating sickness**. Wild hosts include eland, antelope, and zebra. The illness, which begins 4 or more days after *Hyalomma truncatum* tick attachment, is characterized by fever, loss of appetite, lacrimation, salivation, and an eczema-like skin disease but no paralysis. Approximately 75% of afflicted animals die. The active principal component is a salivary gland protein; this is present in females of only certain strains of this tick species. A similar disease has been reported in India and Sri Lanka.

Large numbers of *Rhipicephalus appendiculatus* (the brown ear tick) in southern Africa also have been suspected of causing tick toxicosis in cattle. This may be partly complicated by the transmission of the mildly pathogenic agent *Theileria taurotragi*.

PREVENTION AND CONTROL

Historically, control of ticks and tick-borne diseases almost always relied on pesticides to kill the ticks and o drugs to kill the infectious agents. The cattle tick *R.* (*B.*) *annulatus* was eradicated in the United States by dipping cattle in pesticide solutions, thereby eliminating the deadly Texas cattle fever. Quarantine, pasture rotation, and elimination of deer also were used in the effort to eradicate this vector. It has been estimated that reintroduction of cattle ticks, *Rhipicephalus* (*B.*) spp., could cost the United States cattle industry more than US$1 billion annually to achieve eradication again. Costs to the worldwide cattle industry were estimated in 1984 by the Food and Agriculture Organization of the United Nations at more than US$7 billion. Damage to other livestock and valuable wildlife by ticks and tick-borne diseases is much more difficult to estimate. Losses due to human illnesses have never been calculated.

Treatment with acaricides still provides the most widely used means to control or prevent tick attacks. However, intensive use of acaricides has resulted in many populations of ticks resistant to the limited chemical tools currently available for use (Rodriquez-Vivas et al., 2018). Promising alternatives, such as **vaccines** or **pheromone-acaricidal treatments**, are being investigated. Two commercial recombinant vaccines have been available against the southern cattle tick, *R.* (*B.*) *microplus*, based on a so-called concealed antigen, which occurs in the tick gut but is not exposed to the host immune system during normal feeding. Research continues on multiple other antigens and other important tick species. These and other novel alternatives are discussed later. Integrated management of ticks using a combination of techniques and tools will provide the most sustainable means of reducing tick numbers.

Personal Protection

Personal protective measures are the most effective means for preventing tick bites in persons who enter tick-infested habitats. However, these practices are used less often than desired even in endemic areas (Butler et al., 2016). People at risk for tick bite should wear boots, socks, long trousers, and light-colored clothing. Trousers should be tucked into the boots, socks should be drawn over trousers, and the socks should be taped to form a tight seal. The clothing should be treated with a repellent or **acaricide**. It is now possible to obtain clothing permanently impregnated with permethrin that remains efficacious for the life of the garment, despite repeated washings (Faulde and Uedelhoven, 2006; Vaughn et al., 2014). A recent study showed that wearing protective clothing was 40% effective in preventing Lyme disease (Vasquez et al., 2008). Exposed skin also should be treated with repellents suitable for use on humans (Pages et al., 2014). Permethrin should not be applied to skin.

Each person should conduct daily self-examinations ("tick checks") for ticks during and after exposure to tick-infested areas. Early removal of attached ticks is

important in minimizing the risk of contracting tick-borne diseases. Ticks should be removed by grasping the capitulum as close to the skin as possible with a pair of fine forceps and gently pulling the tick with a slow, steady force until its mouthparts release their hold. Turning or twisting the tick should be avoided to prevent the hypostome breaking off in the wound.

The most widely used personal protectant is the repellent DEET, usually available as a lotion or a spray. Applications should be repeated as per label instructions to maintain maximum protection, but should be applied cautiously on children to avoid adverse reactions that occasionally follow overuse. Newer formulations of DEET, picaridin, and some plant-based repellents will provide varying periods of protection and may have characteristics that are more desirable to the user (e.g., non-oily feel, rapid evaporation of the carrier, etc.).

Acaricides

Acaricides are chemicals used to kill ticks and mites. The term ixodicides is sometimes applied to acaricides used against ticks. Acaricides include arsenical preparations, chlorinated hydrocarbons (e.g., DDT and lindane), organophosphorus compounds (e.g., coumaphos), carbamates (e.g., carbaryl), formamidines (e.g., amitraz), pyrethroids (e.g., permethrin, flumethrin), formamidines (e.g., amitraz), macrocyclic lactones (e.g., ivermectin), phenylpyrazoles (e.g., fipronil), insect growth regulators (e.g., fluazuron), and isoxazolines (e.g., afoxalaner, fluralaner, sarolaner). The synthetic pyrethroids are among the safest and most effective pesticides and are now widely used for tick control. Fipronil is a moderately toxic broad-spectrum phenylpyrazole insecticide, widely used against ticks and other ectoparasites of pets. The introduction of the isoxazolines has provided a convenient oral dosing for pets with long-lasting efficacy.

One way to kill ticks on host animals is to dip livestock and pets in a pesticide bath. When used for cattle, this is termed a cattle dip. Dipping alone is not always effective. Often, ticks hidden in sheltered locations (e.g., between the toes, in the ears, or under the tail) are missed and survive to lay eggs and reestablish the pest population. This is valid for motorized spray-races (facilities where cattle are directed into a chute where pressurized spray is directed from various angles). For intensive tick control, dipping is therefore supplemented by applying an acaricidal cream ("tick-grease") or spray to such sites. Acaricides can be delivered as sprays, using manual or motorized high-pressure sprayers to provide a mist that can reach every part of the animal's body. They can also be delivered as **pour-ons** or **spot-ons**. These are topical formulations in which the acaricide is mixed with surfactants to spread the liquid over the animal's hair coat. Finally, they may be applied as **dusts**, in which acaricides are mixed with talc and deposited directly onto the animal's fur. The familiar "flea powders" for pets, which are effective against ticks as well as fleas, and the dust bags used for treating cattle are examples of acaricidal dusts.

To achieve long-lasting efficacy, acaricides can be incorporated into plastic or other suitable matrices that provide a slow release of the toxicant over a period of weeks or months. **Plastic collars**, such as the familiar flea and tick collars, are widely used for control of ticks on cats and dogs. Newer collar products containing flumethrin for ticks and imidicloprid for fleas have been used to reduce ticks in rickettsial endemic areas (Drexler et al., 2014). Similarly, acaricide-impregnated plastic ear tags are widely used for control of ear-infesting ticks (e.g., Gulf Coast tick and spinose ear tick) on cattle and other large domestic animals. However, they are much less effective for control of ticks that attach around the groin, udder, and other parts of the hindquarters of these animals. **Systemic acaricides** offer another means of providing long-lasting and effective tick control. In this case, the toxicant is introduced into the host's blood to kill ticks as they feed on the treated animals. Unfortunately, most acaricides are too toxic to administer to animals systemically. An exception is ivermectin, which can provide excellent control of certain ticks on cattle for 2−3 months.

Each application method has its advantages and disadvantages. Dips and spray-races are suitable for treating large numbers of animals. The efficacy of manual spraying depends on the person applying it and it can only be applied to a limited number of animals, whereas the application by pour-ons can be expensive. Dip sites where persistent arsenical and chlorinated hydrocarbon compounds may have been used for many years are often heavily contaminated and can represent a significant environmental hazard.

The development of **acaricide resistance** by ticks is a continuing concern. Ticks have been found to be resistant to arsenic, cyclodiene pesticides, other chlorinated hydrocarbons, organophosphorus insecticides, pyrethroids, and formamidines. Resistance may occur in one or more species in an area, while other species in the same locality remain acaricide-susceptible. Some strains of cattle ticks in Australia and elsewhere have been found to be resistant to most or all of the acaricides currently in use, including pyrethroids and amitraz. Resistance of cattle ticks to pyrethroids and organophosphorus compounds has also been found in Mexico and poses concerns regarding the possible reestablishment of these ticks into the United States. Continued research is necessary to discover and develop new pesticide products to overcome resistance to compounds already in use. Efforts to identify genetic markers of resistance and develop monitoring assays will provide tools to identify resistant populations so that

knowledge can be used to enhance other tick control strategies (Lees and Bowman, 2007; Miller et al., 2017).

Pheromone-Assisted Control

The difficulties and high cost of tick control on animals have stimulated interest in alternatives to the conventional methods described above. Such alternatives help to reduce the use of acaricides. Research with tick pheromones suggests that combinations of pheromones and acaricides can be significantly more effective for controlling ticks than the acaricide alone, because ticks are unlikely to develop resistance to their own pheromones (Carr and Roe, 2016). A pheromone-acaricide combination applied to a single spot-on cattle can be effective in killing the Gulf Coast tick. Another promising device is the "tick decoy" in which the sex pheromone 2,6-dichlorophenol and an acaricide are impregnated into plastic beads on the surface of which "mounting" sex pheromone is applied. Male ticks are attracted to decoys on the animal's hair coat and killed. This also disrupts mating activity, so that any surviving females cannot lay viable eggs. For the livestock-parasitizing bont ticks *Amblyomma hebraeum* and *A. variegatum,* a tail-tag decoy was developed that uses a mixture of tick-specific phenols to attract ticks to specific sites on cattle and kill them when they attach nearby. Field trials with tail-tags have demonstrated promising efficacy for up to 3 months (Norval et al., 1996; Kelly et al., 2014).

Passive Treatment

Another way to apply acaricides to animals is by means of self-treating devices. Animals seeking food or nesting materials visit these devices and acquire an acaricide, spreading it over their fur and skin to kill ticks. An example is biodegradable cardboard tubes containing a permethrin-impregnated cotton balls. Mice collect the cotton for nesting material, thereby spreading the pesticide among nest mates. Such tubes have been effective in reducing populations of *Ixodes scapularis* and the occurrence of Lyme disease in some localities, especially in residential communities; however, they have not been effective in other situations. Bait boxes containing the acaricide fipronil were found to be effective in killing *I. scapularis* nymphs and larvae on small mammals, reducing these stages by as much as 68% and 84%, respectively, and the infection rate of white-footed mice with *B. burgdorferi* by as much as 53%. Subsequently, the abundance of *I. scapularis* adults in the targeted area was reduced by 77%. Tick infection with *Anaplasma phagocytophilum* was also significantly reduced (Dolan et al., 2004). A commercialized version of the bait box has found to eliminate up to 97% of *I. scapularis* on rodents (Schultze et al., 2017).

Another example is the **self-treating applicator** for controlling blacklegged ticks (*I. scapularis*) on white-tailed deer (Sonenshine et al., 1996). Animals become coated with oil containing an acaricide as they remove food from the applicator. A similar technique was used in Zimbabwe for treating wild ungulates (Duncan and Monks, 1992). Perhaps the most effective example of this strategy is the "four-poster"self-applicating device for treating white-tailed deer against ticks. Deer attracted to corn or other food bait in the device acquire acaricide from cloth-covered rollers. Field studies showed up to 80% and 99.5% efficacy in controlling *I. scapularis* and *A. americanum* nymphs, respectively (Carroll et al., 2002).

Hormone-Assisted Control

Hormones and insect growth regulators (IGRs) such as methoprene also have been used to disrupt tick development in laboratory experiments. Analogues or mimics of ecdysteroids are effective in killing ticks by delaying their development, disrupting oviposition, or killing the larvae when they hatch from eggs deposited by treated females. However, these compounds do not appear to be uniformly effective against all types of ticks.

BIOLOGICAL CONTROL

The use of biological control has been successful with ticks. Fungal and nematode pathogens have been effective within particular environmental conditions. Predators have also been investigated, but their role is generally considered augmentive and is not likely to significantly reduce tick numbers.

In a recent study, integrated use of broadcast application of the fungus *Metarhizium anisopliae*, fipronil-based bait stations to treat rodents, and reduction of deer was able to significantly reduce questing tick nymphs in the study area (Williams et al., 2018). Such combination of approaches will lead to better and more sustainable control.

Vaccines

Anti-tick vaccines have been used successfully. In Australia, a commercial recombinant antigen vaccine has been developed for the control of the southern cattle tick *R.* (*B.*) *microplus*, based on a so-called concealed antigen (Bm86) in cells of the tick gut. A similar recombinant vaccine, based on the same antigen, has been developed in Cuba. Recent reports suggest that the recombinant Bm86 can reduce tick fecundity by as much as 90%. Although it is possible that antigen-resistant strains of cattle ticks may

appear, large-scale vaccination of cattle herds with these recombinant vaccines offers a promising alternative or supplement to acaricides. The vaccines are active not only against *R.* (*B.*) *microplus*, but also against related species.

Research on many other antigens and other tick species is in progress (de la Fuentes and Contreras, 2015). Of special interest is the development of novel combinations using RNAi (see Chapter 28) to silence subolesin and a tick-protective antigen, Rs86 (similar to Bm86); the synergistic effect of silencing both genes causes a much greater reduction of tick feeding and oviposition than targeting either one alone (de la Fuente et al., 2006). Another promising vaccine targets tick-cement protein, disrupting attachment success, as well as midgut injury and the tick's ability to transmit pathogens (Labuda et al., 2006). More recently, peptide vaccines produced to the tick ribosomal protein P0 have shown 90% efficacy against *Rhipicephalus sanguineus* when fed upon immunized rabbits (Rodriquez-Mallon et al., 2012). This same vaccine showed 96% efficacy against *R.* (*B.*) *microplus* (Rodriquez-Mallon et al., 2015).

Management

Management practices provide another means of reducing tick numbers. Zero-grazing (i.e., keeping animals confined in stables) minimizes exposure to ticks. Acaricides can be applied directly to vegetation. However, because ticks commonly occur in microhabitats covered by vegetation, leaf litter, soil, and other natural materials, or in the nests, burrows, and other cavities used by their hosts, they often do not come in direct contact with these toxicants. Therefore, to be effective, the acaricides must reach the ticks as vapors or by contact when the ticks move about while seeking hosts. Public opposition to treatment of natural habitats with pesticides has made it unpopular to use this form of tick control except for the most compelling reasons. In recent years, acaricidal treatment of natural areas has been limited largely to military bases or selected recreational areas. Alternatives include habitat modifications such as burning or clearing vegetation or host removal (e.g., removal of deer by hunting and deer-exclusion fences). Burning or clearing vegetation removes the dense cover under which ticks shelter, thereby reducing ground-level humidity as well as exposing them to intense ultraviolet radiation and heat. Such changes can make the habitat unsuitable for tick survival. In field trials in southwestern Georgia, regular prescribed burning was found to significantly reduce tick populations and may also reduce exposure to tick-borne pathogens (Gleim et al., 2017).

Integrated Tick Management

Integrated management of ticks provides the most sustainable and effective means of reducing ticks in the residential environment (Stafford, 2007). Integrated control of ticks also can include the timing of acaricide treatments (e.g., when most engorged females of a particular species drop from their host); livestock management practices such as rotational grazing; cattle-breed resistance, selected use of acaricides, and pathogens and predators of ticks.

Monitoring

Tick surveys often are conducted to determine whether tick control is warranted and, if so, when it should be implemented. The most common method for sampling ticks is the use of a **flag** or **drag cloth** pulled or dragged through the vegetation. Ticks collected on the cloth are counted as the number of a given species per unit of distance dragged (e.g., 100 m) or the number collected per hour of dragging. Although absolute measures of tick population densities cannot be obtained in this manner, the relative abundance of ticks in different areas sampled can be determined. Tick species collected can provide an indication of potential risks of tick-borne diseases in a given area. There are biases to each method of tick monitoring, so the results must be carefully interpreted.

An alternative to dragging is the use of **carbon dioxide traps**. Carbon dioxide gas from a block of dry ice or from a compressed-gas cylinder is the tick attractant. Ticks adhering to or crawling around the trap are counted after a few to several hours of operation. When more reliable estimates of tick abundance are required, a mark-and-recapture method can be used. Using this method, the numbers of marked ticks recaptured from a previous sample are compared with the number of unmarked ticks to obtain an estimate of the entire tick population in the area studied.

For tick-infested cattle, horses, mules, and other livestock, a time-tested method is the **scratching technique**, whereby livestock inspectors pass their hands over different regions of the animal's body to detect attached ticks. A similar technique is used in combination with visual inspection to examine wild animals. For example, investigators can be assigned to deer check stations during hunting season to count all ticks on hunter-killed animals. Another technique for sampling ticks is to trap small and medium-sized wild animals and hold them over trays filled with water or alcohol to catch fed ticks as they detach.

Eradication

In a few cases, tick eradication may be practicable. An example is the Cattle Tick Fever Eradication Program that was initiated in the southern United States in 1907. This program led to the eradication of *R.* (*B.*) *annulatus, R.* (*B.*) *microplus,* and Texas cattle fever from the United States by 1960. However, reinvasion from Mexico continues to be a constant threat, because of illegal movement of livestock across the United States−Mexico border and uncontrollable wild hosts. Tick utilization of native wildlife and exotic hoofstock is a growing concern (Perez de Leon et al., 2012). Attempts to eradicate *R* (*B.*) *microplus* in other areas (U.S. Virgin Islands, Argentina, Uruguay, Australia, and Papua New Guinea) have been unsuccessful, despite reductions of more than 99% of the tick populations in some localities. Acaricide resistance, as well as reinvasion by ticks and their rapid repopulation of areas in the eradication zone, are major contributing factors.

An attempt to eradicate *Amblyomma variegatum*, a vector of the agent of heartwater and associated with bovine dermatophilosis, from islands in the Caribbean has been successful on some islands (Eddy, 2002; Pegram et al., 2004). However, it has not achieved its overall purpose, which was to eradicate the tick from the Western Hemisphere. Although the lack of international collaboration, political will, and funding have been contributing factors, there are also technical reasons that have prevented successful eradication of this tick on some of the islands where it is well established. The tick continues to be a major threat to the American mainland and the Greater Antilles.

Occasionally eradication has been achieved in the case of exotic species that were recognized soon after their introduction to a new area. An example includes the eradication of the African species *R. evertsi* soon after it was introduced into a wild-animal compound in Florida. Eradication is easiest if exotic species are identified as soon as possible after their introduction. This was addressed following the discovery of *Amblyomma marmoreum* and *A. sparsum*, both vectors of heartwater, on nine different premises in Florida (Burridge et al., 2000).

REFERENCES AND FURTHER READING

Allsopp, B. A. (2010). Natural history of *Ehrlichia ruminantium*. *Veterinary Parasitology, 167*, 123−135.

Allsopp, B. A. (2015). Heartwater−*Ehrlichia ruminantium* infection. *Revue Scientifique et Technique/Office International des Épizooties, 34*, 557−568.

Alverez-Hernandez, G., Roldan, J. F. G., Milan, N. S. H., Lash, R. R., Behravesh, C. B., & Paddock, C. D. (2017). Rocky Mountain spotted fever in Mexico: Past, present, and future. *Lancet Infectious Diseases, 17*, e189−e196.

Anderson, J. F., Sonenshine, D. E., & Valenzuela, J. (2008). Exploring the mialome of ticks: An annotated catalogue of midgut transcripts from the hard tick *Dermacentor variabilis* (Acari: Ixodidae). *BMC Genomics, 9*, 552. https://doi.org/10.1186/1471-2164-9-552.

Anderson, J. M., Moore, I. N., Nagata, B. M., Ribeiro, J. M. C., Valenzuela, J. G., & Sonenshine, D. E. (2017). Ticks, *Ixodes scapularis*, feed repeatedly on white-footed mice despite strong inflammatory 1 response: An expanding paradigm for understanding tick-host-interactions. *Frontiers in Immunology, 8*, 1784.

Anziani, O. S., Ewing, S. A., & Barker, R. W. (1990). Experimental transmission of a granulocytic form of the tribe Ehrlichieae by *Dermacentor variabilis* and *Amblyomma americanum* to dogs. *American Journal of Veterinary Research, 51*, 929−931.

Araujo, R. N., Franco, P. F., Rodrigues, H., Santos, L. C. B., McKay, C. S., Sanhueza, C. A., et al. (2016). *Amblyomma sculptum* tick saliva: α-gal identification, antibody response and possible association with red meat allergy in Brazil. *International Journal of Parasitology, 46*, 213−220.

Attoui, H., Jaafar, F. M., de Micco, P., & de Lamballerie, X. (2005). Coltiviruses and seadorna viruses in North America, Europe, and Asia. *Emerging Infectious Diseases, 11*, 1673−1679.

Bäckman, S., Näslund, J., Forsman, M., & Thelaus, J. (2015). Transmission of tularemia from a water source by transstadial maintenance in a mosquito vector. *Scientific Reports, 5*, 7793.

Balashov, Y. S. (1972). Bloodsucking ticks (Ixodoidea) − vectors of diseases of man and animals. *Miscellaneous Publications of the Entomological Society of America, 8*, 161−376.

Barbour, A. G. (2014). Phylogeny of a relapsing fever *Borrelia* species transmitted by the hard tick *Ixodes scapularis*. *Infection, Genetics and Evolution, 27*, 551−558.

Barbour, A. G., Bunikis, J., Fish, D., & Hanincova, K. (2015). Association between body size and reservoir competence of mammals bearing *Borrelia burgdorferi* at an endemic site in the northeastern United States. *Parasites & Vectors, 8*, 299.

Barker, S. C., & Murrell, A. (2002). Phylogeny, evolution and historical zoogeography of ticks: A review of recent progress. *Experimental & Applied Acarology, 28*, 55−68.

Beall, M. J., Alleman, A. R., Breitschwerdt, E. B., Cohn, L. A., Couto, C. G., Dryden, M. W., et al. (2012). Seroprevalence of *Ehrlichia canis, Ehrlichia chaffeensis*, and *Ehrlichia ewingii* in dogs in North America. *Parasites & Vectors, 5*, 29.

Beati, L., & Keirans, J. E. (2001). Analysis of the systematic relationships among ticks of the genera *Rhipicephalus* and *Boophilus* (Acari: Ixodidae) based on mitochondrial 12S ribosomal DNA gene sequences and morphological characters. *Journal of Parasitology, 87*, 32−48.

Bell, J. F. (1988). Tularemia. In J. H. Steele (Ed.), *CRC handbook series in zoonoses. Section A. Bacterial, rickettsial, and mycotic disease* (Vol. 2, pp. 161−193). Boca Raton, FL: CRC Press.

Bente, D. A., Forrester, N. L., Watts, D. M., McAuley, A. J., Whitehouse, C. A., & Bray, M. (2013). Crimean-Congo hemorrhagic fever: History, epidemiology, pathogenesis, clinical syndrome, and genetic diversity. *Antiviral Research, 100*, 159−189.

Berrada, Z. L., Goethert, H. K., & Telford, S. R., III (2006). Raccoons and skunks as sentinels for enzootic tularemia. *Emerging Infectious Diseases, 12*, 1019−1021.

Beugnet, F., & Moreau, Y. (2015). Babesiosis. *Revue Scientifique et Technique/Office International des Épizooties, 34*, 627−639.

Bissinger, B. W., Donohue, K. V., Khalil, S. M., Grozinger, C. M., Sonenshine, D. E., Zhu, J., et al. (2011). Synganglion transcriptome and developmental global gene expression in adult females of the American dog tick, *Dermacentor variabilis* (Acari: Ixodidae). *Insect Molecular Biology, 20*, 465−491. https://doi.org/10.1111/j.1365-2583.2011.01086.x.

Borovickova, B., & Hypša, V. (2005). Ontogeny of tick hemocytes: A comparative analysis of *Ixodes ricinus* and *Ornithodoros moubata*. *Experimental & Applied Acarology, 35*, 317−333.

Bosco-Lauth, A. M., Panella, N. A., Root, J. J., Gidlewski, T., Lash, R. R., Harmon, J. R., et al. (2015). Serological investigation of Heartland virus (Bunyaviridae: *Phlebovirus*) exposure in wild and domestic animals adjacent to human case sites in Missouri, 2012−2013. *American Journal of Tropical Medicine and Hygiene, 92*, 1163−1167.

Bosman, A. M., Oosthuizen, M. C., Venter, E. H., Steyl, J. C., Gous, T. A., & Penzhorn, B. L. (2013). *Babesia lengau* associated with cerebral and haemolytic babesiosis in two domestic cats. *Parasites and Vectors, 6*, 128.

Bosman, A. M., Venter, E. H., & Penzhorn, B. L. (2007). Occurrence of *Babesia felis* and *Babesia leo* in various wild felid species and domestic cats in Southern Africa, based on reverse line blot analysis. *Veterinary Parasitology, 144*, 33−38.

Boyer, K. M., Munford, R. S., Maupin, G. O., Pattison, C. P., Fox, M. D., Barnes, A. M., et al. (1977). Tick-borne relapsing fever: An interstate outbreak originating at Grand Canyon National Park. *American Journal of Epidemiology, 105*, 469−479.

Branda, J. A., & Rosenberg, E. S. (2013). *Borrelia miyamotoi*: A lesson in disease discovery. *Annals of Internal Medicine, 159*, 61−62.

Brisson, D., Dykhuizen, D. E., & Ostfeld, R. S. (2008). Conspicuous impacts of inconspicuous hosts on the Lyme disease epidemic. *Proceedings of the Royal Society of London B: Biological Sciences, 275*, 227−235.

Brooks, R. S., Blanchard, M. T., Clothier, K. A., Fish, S., Anderson, M. L., & Stott, J. L. (2016). Characterization of *Pajaroellobacter abortibovis*, the etiologic agent of epizootic bovine abortion. *Veterinary Microbiology, 192*, 73−80.

Buller, R. S., Arens, M., Hmiel, S. P., Paddock, C. D., Summer, J. W., Rikihisa, Y., et al. (1999). *Ehrlichia ewingii*, a newly recognized agent of human ehrlichiosis. *New England Journal of Medicine, 341*, 148−155.

Burgdorfer, W., Barbour, A. G., Hayes, S. F., Benach, J. L., Grunwaldt, E., & Davis, J. P. (1982). Lyme disease − a tick-borne spirochetosis? *Science, 216*, 1317−1319.

Burgdorfer, W., & Schwan, T. G. (1991). Borrelia. In A. Balows, W. J. Hausler, Jr., K. L. Herrman, H. D. Isenberg, & H. J. Shadomy (Eds.), *Manual of clinical microbiology* (5th ed., pp. 560−566). Washington, D.C: American Society of Microbiology.

Burger, D. B., Crause, J. C., Spickett, A. M., & Neitz, A. W. H. (1991). A comparative study of proteins present in sweating-sickness-inducing and non-inducing strains of *Hyalomma truncatum* ticks. *Experimental & Applied Acarology, 13*, 59−63.

Burridge, M. J., Simmons, L. A., & Allan, S. A. (2000). Introduction of potential heartwater vectors and other exotic ticks in Florida on imported reptiles. *Journal of Parasitology, 86*, 700−704.

Butler, A. D., Sedghi, T., Petrini, J. R., & Ahmadi, R. (2015). Tick-borne disease preventive practices and perceptions in an endemic area. *Ticks and Tick-borne Diseases, 7*, 331−337.

Camus, E., & Barreé, N. (1995). Vector situation of tick-borne diseases in the Caribbean Islands. *Veterinary Parasitology, 57*, 167−176.

Camus, E., Barré, N., Martinez, D., & Uilenberg, G. (1996). *Heartwater (cowdriosis): A review* (2nd ed.). Paris: Office International des Epizooties, 177 pp.

Carr, A. L., Mitchell, R. D., III, Dhammi, A., Bissinger, B. W., Sonenshine, D. E., & Roe, R. M. (2017). Tick Haller's organ, a new paradigm for arthropod olfaction: How ticks differ from insects. *Journal of Molecular Science, 18*(7). https://doi.org/10.3390/ijms1807156. pii: E1563.

Carr, A. L., & Roe, M. (2016). Acarine attractants: Chemoreception, bioassay, chemistry and control. *Pesticide Biochemistry and Physiology, 131*, 60−79.

Carroll, J. F., Allen, P. C., Hill, D. E., Pound, J. M., Miller, J. A., & George, J. E. (2002). Control of *Ixodes scapularis* and *Amblyomma americanum* through use of the '4-poster' treatment device on deer in Maryland. *Experimental & Applied Acarology, 28*, 289−296.

Carvalho, C. L., Lopes de Carvalho, I., Zé-Zé, L., Núncio, M. S., & Duarte, E. L. (2014). Tularaemia: A challenging zoonosis. *Comparative Immunology Microbiology and Infectious Diseases, 37*, 85−96.

Casjens, S. R., Fraser-Liggett, C. M., Mongodin, E. F., Qiu, W. G., Dunn, J. J., Luft, B. J., et al. (2011a). Whole genome sequence of an unusual *Borrelia burgdorferi* sensu lato isolate. *Journal of Bacteriology, 193*, 1489−1490.

Casjens, S. R., Mongodin, E. F., Qiu, W. G., Dunn, J. J., Luft, B. J., Fraser-Liggett, C. M., et al. (2011b). Whole-genome sequences of two *Borrelia afzelii* and two *Borrelia garinii* Lyme disease agent isolates. *Journal of Bacteriology, 193*, 6995−6996.

Centers for Disease Control and Prevention. (2001). Outbreak of Powassan encephalitis − Maine and Vermont, 1999-2001. *Morbidity and Mortality Weekly Report, 50*, 761−764.

Centers for Disease Control and Prevention. (2006). Cluster of tick paralysis cases—Colorado, 2006. *Morbidity and Mortality Weekly Report, 55*, 933−995.

Ceraul, S. M., Sonenshine, D. E., & Hynes, W. L. (2002). Investigations into the resistance of the tick, *Dermacentor variabilis* (Say) (Acari: Ixodidae) following challenge with the bacterium, *Escherichia coli* (Enterobacteriales: Enterobacteriaceae). *Journal of Medical Entomology, 39*, 376−383.

Ceraul, S. M., Sonenshine, D. E., Ratzlaff, R. E., & Hynes, W. L. (2003). An arthropod defensin expressed by the hemocytes of the American dog tick, *Dermacentor variabilis* (Acari: Ixodidae). *Insect Biochemistry and Molecular Biology, 33*, 1099−1103.

Charrel, R. N., Attoui, H., Butenko, A. M., Clegg, J. C., Deubel, V., Frolova, T. V., et al. (2004). Tick-borne virus diseases of human interest in Europe. *Clinical Microbiology and Infection, 10*, 1040−1055.

Childs, J. E., & Paddock, C. D. (2003). The ascendancy of *Amblyomma americanum* as a vector of pathogens affecting humans in the United States. *Annual Review of Entomology, 48*, 307−337.

Chmelař, J., Kotál, J., Karim, S., Kopacek, P., Francischetti, I. M., Pedra, J. H., et al. (2016). Sialomes and mialomes: A systems-biology view of tick tissues and tick-host interactions. *Trends in Parasitology, 32*, 242−254.

Clark, K., Hendricks, A., & Burge, D. (2005). Molecular identification and analysis of *Borrelia burgdorferi* sensu lato in lizards in the southeastern United States. *Applied and Environmental Microbiology, 71*, 2616−2625.

Clark, K. L., Leydet, B., & Hartman, S. (2013). Lyme borreliosis in human patients in Florida and Georgia, USA. *International Journal of Medical Sciences, 10*, 915–931.

Clifford, C. M., Anastos, A., & Elbl, A. (1961). The larval ixodid ticks of the eastern United States. *Miscellaneous Publications of the Entomological Society of America, 2*, 213–217.

Conrad, P. A., Kjemtrup, A. M., Carreno, R. A., Thomford, J., Wainwright, K., Eberhard, M., et al. (2006). Description of *Babesia duncani* n. sp. (Apicomplexa: Babesiidae) from humans and its differentiation from other piroplasms. *International Journal of Parasitology, 36*, 779–789.

Coons, B., Rosell-Davis, R., & Tarnowski, B. I. (1986). Bloodmeal digestion in ticks. In J. R. Sauer, & J. A. Hair (Eds.), *Morphology, physiology and behavioral biology of ticks* (pp. 248–279). Chichester, U.K: Ellis Harwood.

Crowder, C. D., Carolan, H. E., Rounds, M. A., Honig, V., Mothes, B., Haag, H., et al. (2014). Prevalence of *Borrelia miyamotoi* in *Ixodes* ticks in Europe and the United States. *Emerging Infectious Diseases, 20*, 1678–1682.

Cutler, S. J., Ruzic-Sabljic, E., & Potkonjak, A. (2017). Emerging borreliae - expanding beyond Lyme borreliosis. *Molecular and Cellular Probes, 31*, 22–27.

Dahlgren, F. S., Heitman, K. N., Drexler, N. A., Massung, R. F., & Behravesh, C. B. (2015). Human granulocytic anaplasmosis in the United States from 2008 to 2012: A summary of national surveillance data. *American Journal of Tropical Medicine and Hygiene, 93*, 66–72.

de la Fuente, J., Almazán, C., Naranjo, V., Blouin, E. F., & Kocan, K. M. (2006). Synergistic effect of silencing the expression of tick protective antigens 4D8 and Rs86 in *Rhipicephalus sanguineus* by RNA interference. *Parasitology Research, 99*, 108–113.

de la Fuentes, J., & Contreras, M. (2015). Tick vaccines: Current status and future directions. *Expert Review of Vaccines, 14*, 1367–1376.

Demma, L. J., Traeger, M., Nicholson, W. L., Paddock, C. D., Blau, D., Eremeeva, M., et al. (2005b). Rocky Mountain spotted fever from an unexpected tick vector in Arizona. *New England Journal of Medicine, 353*, 587–594.

Drexler, N., Miller, M., Gerding, J., Todd, S., Adams, L., Dahlgren, F. S., et al. (2014). Community-based control of the brown dog tick in a region with high rates of Rocky Mountain spotted fever, 2012–2013. *PLoS One, 9*, e112368.

Drexler, N. A., Yaglom, H., Casal, M., Fierro, M., Kriner, P., Murphy, B., et al. (2017). Fatal Rocky Mountain spotted fever along the United States-Mexico border, 2013–2016. *Emerging Infectious Diseases, 23*, 1621–1626.

Dugat, T., Lagrée, A.-C., Maillard, R., Boulouis, H.-J., & Haddad, N. (2015). Opening the black box of *Anaplasma phagocytophilum* diversity: Current situation and future perspectives. *Frontiers in Cellular and Infection Microbiology, 5*, 61.

Duncan, I. M., & Monks, N. (1992). Tick control on eland (*Taurotragus oryx*) and buffalo (*Syncerus caffer*). *The Journal of the South African Veterinary Association, 63*, 7–10.

Durden, L. A., & Keirans, J. E. (1996). Nymphs of the genus *Ixodes* (Acari: Ixodidae) of the United States: Taxonomy, identification key, distribution, hosts, and medical/veterinary importance. Lanham, MD: *Thomas Say Publications in Entomology, Monographs*: *Entomological Society of America*, 95 pp.

Durden, L. A., & Mans, B. (2016). Tick paralysis: Some host and tick perspectives. In J. Janovy, Jr., & G. W. Esch (Eds.), *A century of parasitology: Discoveries, ideas and lessons learned by scientists who published in the Journal of Parasitology, 1914–2014* (pp. 167–176). Hoboken, New Jersey: Wiley.

Duron, O., Sidi-Boumedine, K., Rousset, E., Moutailler, S., & Jourdain, E. (2015). The importance of ticks in Q fever transmission: What has (and has not) been demonstrated? *Trends in Parasitology, 31*, 536–552.

Dworkin, M. S., Shoemaker, P. C., & Anderson, D. E. (1999). Tick paralysis: 33 human cases in Washington state, 1946–1996. *Clinical Infectious Diseases, 29*, 1435–1439.

Dworkin, M. S., Shoemaker, P. C., Fritz, C. L., Dowell, M. E., & Anderson, D. E., Jr. (2002). The epidemiology of tick-borne relapsing fever in the United States. *The American Journal of Tropical Medicine and Hygiene, 66*, 753–758.

Dworkin, M. S., Schwan, T. G., Anderson, D. E., Jr., & Borchardt, S. M. (2008). Tick-borne relapsing fever. *Infectious Disease Clinics of North America, 22*, 449–468.

Ebel, G. (2010). Update on Powassan virus: Emergence of a North American tick-borne flavivirus. *Annual Review of Entomology, 55*, 95–110.

Egekwu, N., Sonenshine, D. E., Bissinger, B. W., & Roe, R. M. (2014). Transcriptome of the female synganglion of the black-legged tick *Ixodes scapularis* (Acari: Ixodidae) with comparison between Illumina and 454 systems. *PLoS One, 9*(7), e102667.

Egekwu, N. I., Sonenshine, D. E., Garman, H., Barshis, D. J., Cox, N., Bissinger, B. W., et al. (2016). Comparison of synganglion neuropeptides, neuropeptide receptors and neurotransmitter receptors and their gene expression in response to feeding in *Ixodes scapularis* (Ixodidae) versus *Ornithodoros turicata* (Argasidae). *Insect Molecular Biology, 25*, 72–92.

Eggenberger, L., Lamoreaux, W. R., & Coons, L. B. (1990). Hemocytic encapsulation of implants in the tick *Dermacentor variabilis*. *Experimental & Applied Acarology, 9*, 279–287.

Eisen, L. (2007). A call for renewed research on tick-borne *Francisella tularensis* in the Arkansas-Missouri primary natural focus of tularemia in humans. *Journal of Medical Entomology, 44*, 389–397.

Eisen, R. J., Kugeler, K. J., Eisen, L., Beard, C. B., & Paddock, C. D. (2017). Tick-borne zoonoses in the United States: Persistent and emerging threats to human health. *Institute for Laboratory Animal Research Journal, 23*, 1–17.

Estada-Peña, A., Mangold, A. J., Nava, S., Venzal, J. M., Labruna, M., & Guglielmone, A. A. (2010). A review of the systematics of the tick family Argasidae (Ixodida). *Acarologia, 50*, 317–333.

Faulde, M., & Uedelhoven, W. (2006). A new clothing impregnation method for personal protection against ticks and biting insects. *The International Journal of Medical Microbiology, 296* (Suppl. 40), 225–229.

Feir, D., Santanello, C. R., Li, B. W., Xie, C. S., Masters, E., Marconi, R., et al. (1994). Evidence supporting the presence of *Borrelia burgdorferi* in Missouri. *American Journal of Tropical Medicine and Hygiene, 51*, 475–482.

Filippova, N. A. (1966). Argasid ticks (Argasidae). *Fauna SSSR, Paukoobraznye, 4*(3), 255 pp. (in Russian).

Filippova, N. A. (1967). Ixodid ticks of the subfamily Ixodinae. *Fauna SSSR, Paukoobraznye, 4*(4), 396 pp. (in Russian).

Fish, D. (2013). *Borrelia miyamotoi*: More lessons on disease discovery. *Annals of Internal Medicine, 159*, 648.

Foley, J. E., & Nieto, N. C. (2010). Tularemia. *Veterinary Microbiology, 140*, 332−338.

Forrester, J. D., Kjemtrup, A. M., Fritz, C. L., Marsden-Haug, N., Nichols, J. B., Tengelsen, L. A., et al. (2015). Tickborne relapsing fever - United States, 1990-2011. *Morbidity and Mortality Weekly Report, 64*, 58−60.

Francischetti, I. M. B., Nunes, A. S., Mans, B., & Ribeiro, J. M. C. (2009). The role of saliva in tick feeding. *Frontiers in Bioscience, 14*, 2051−2088.

Fukunaga, M., Takahashi, Y., Tsuruta, Y., Matsushita, O., Ralph, D., McClelland, M., et al. (1995). Genetic and phenotypic analysis of *Borrelia miyamotoi* sp. nov., isolated from the ixodid tick *Ixodes persulcatus*, the vector for Lyme disease in Japan. *International Journal of Systematic Bacteriology, 45*, 804−810.

Gall, C. A., Reif, K. E., Scoiles, G. A., Mason, K. L., Mousel, M., Noh, S. M., et al. (2016). The bacterial microbiome of *Dermacentor andersoni* ticks influences pathogen susceptibility. *International Society for Microbial Ecology Journal, 10*, 1846−1855.

Genchi, C. (2007). Human babesiosis, an emerging zoonosis. *Parassitologia, 49*, 29−31.

Gern, L., & Humair, P.-F. (2002). Ecology of *Borrelia burgdorferi* sensu lato in Europe. In J. Gray, O. Kahl, R. S. Lane, & G. Stanek (Eds.), *Lyme borreliosis: Biology, epidemiology and control* (pp. 149−174). New York: CABI Publishing.

Gilbert, L. (2016). Louping ill virus in the UK: A review of the hosts, transmission and ecological consequences of control. *Experimental & Applied Acarology, 68*, 363−374.

Gill, J. S., Rowley, W. A., Bush, P. J., Viner, J. P., & Gilchrist, M. J. R. (2004). Detection of human blood in the bat tick, *Carios (Ornithodoros) kelleyi* (Acari: Argasidae). *Journal of Medical Entomology, 41*, 1179−1181.

Gleim, E. R., Conner, L. M., Bergaus, R. D., Levin, M. L., Zemtsova, G. E., & Yabsley, M. J. (2014). The phenology of ticks and the effects of long-term prescribed burning on tick population dynamics in southwestern Georgia and northwestern Florida. *PLoS One, 9*, e112174.

Goodman, J. L., Dennis, D. T., & Sonenshine, D. E. (Eds.). (2005). *Tick-borne diseases of humans*. Washington, D.C.: ASM Press, 401 pp.

Goethert, H. K., Shani, I., & Telford, S. R., III (2004). Genotypic diversity of *Francisella tularensis* infecting *Dermacentor variabilis* ticks on Martha's Vineyard, Massachusetts. *Journal of Clinical Microbiology, 42*, 4968−4973.

Golovchenko, M., Vancová, M., Clark, K., Oliver, J. H., Grubhoffer, L., & Rudenko, N. (2016). A divergent spirochete strain isolated from a resident of the southeastern United States was identified by multilocus sequence typing as *Borrelia bissettii*. *Parasites & Vectors, 9*, 68.

Gothe, R. (1999). *Tick toxicoses*. Munich: Hieronymus, 377 pp. (in German).

Gough, J. M., & Kemp, D. H. (1995). Acid phosphatase in midgut digestive cells in partially fed females of the cattle tick *Boophilus microplus*. *Journal of Parasitology, 81*, 341−349.

Grard, G., Moureau, G., Charrel, R. N., Lemasson, J.−J., Gonzalez, J.-P., Gallian, P., et al. (2007). Genetic characterization of tick-borne flaviviruses: New insights into evolution, pathogenetic determinants and taxonomy. *Virology, 361*, 80−92.

Grattan-Smith, P. J., Morris, J. G., Johnston, H. M., Yiannikas, C., Malik, R., Russell, R., et al. (1997). Clinical and neurophysiological features of tick paralysis. *Brain, 120*, 1975−1987.

Gray, J. S. (1991). The development and seasonal activity of the tick *Ixodes ricinus*: A vector of Lyme borreliosis. *Review of Medical and Veterinary Entomology, 79*, 323−333.

Gray, J., Kahl, O., Lane, R. S., & Stanek, G. (2002). *Lyme borreliosis: Biology, epidemiology and control*. New York, NY: CABI Publishing.

Gray, J., Kahl, O., Lane, R. S., Levin, M. L., & Tsao, J. I. (2016). Diapause in ticks of the medically important *Ixodes ricinus* species complex. *Ticks and Tick-borne Diseases, 7*, 992−1003.

Gregson, J. D. (1973). Tick paralysis: An appraisal of natural and experimental data. *Monograph No. 9*. Ottawa: Canada Dept. Agric, 109 pp.

Gritsun, T. S., Lashkevich, V. A., & Gould, E. A. (2003). Tick-borne encephalitis. *Antiviral Research, 57*, 129−146.

Grubhoffer, L., Kovar, V., & Rudenko, N. (2004). Tick lectins: Structural and functional properties. *Parasitology (Suppl), 129*, S113−S125.

Guglielmone, A. A., Robbins, R. G., Apanaskevich, D. A., Petney, T. N., Estrada-Pena, A., & Horak, I. G. (2014). *The hard ticks of the World (Acari: Ixodida: Ixodidae)*. Dordrecht: Springer, 738 pp.

Guglielmone, A. A., Robbins, R. G., Apanaskevich, D. A., Petney, T. N., Estrada-Pena, A., Horak, I. G., et al. (2010). The Argasidae, Ixodidae and Nuttalliellidae (Acari: Ixodida) of the world: A list of valid species names. *Zootaxa, 2528*, 1−28.

Hadrill, D. J., Boid, R., Jones, T. W., & Bell-Sakyi, L. (1990). Bovine babesiosis on Nevis − implications for tick control. *Veterinary Record, 126*, 403−404.

Hamsten, C., Tran, T. A. T., Starkhammar, M., Brauner, A., Commins, S. P., Mills-Platt, T. A. E., et al. (2013). Red meat allergy in Sweden: Association with tick sensitization and B-negative blood groups. *Journal of Allergy and Clinical Immunology, 132*, 1431−1434 (plus electronic only 1434e1-e6).

Hanincová, K., Kurtenbach, K., Diuk-Wasser, M., Brei, B., & Fish, D. (2006). Epidemic spread of Lyme borreliosis, northeastern United States. *Emerging Infectious Diseases, 12*, 604−611.

Harwood, R. F., & James, M. T. (1979). *Entomology in human and animal health* (7th ed.). New York: MacMillan Publishing Co., 548 pp.

Hegarty, B. C., Maggi, R. G., Koskinen, P., Beall, M. J., Eberts, M., Chandrashekar, R., et al. (2012). *Ehrlichia muris* infection in a dog from Minnesota. *Journal of Veterinary Internal Medicine, 26*, 1217−1220.

Heinz, F. X., Holzmann, H., Essl, A., & Kundi, M. (2007). Field effectiveness of vaccination against tick-borne encephalitis. *Vaccine, 25*, 7559−7567.

Hermance, M. E., & Thangamani, S. (2017). Powassan virus: An emerging arbovirus of public health concern in North America. *Vector Borne and Zoonotic Diseases, 17*, 453−462. https://doi.org/10.1089/vbz.2017.2110.

Herwaldt, B. L., Cacciò, S., Gherlinzoni, F., Aspöck, H., Slemenda, S. B., Piccaluga, P., et al. (2003). Molecular characterization of a non-*Babesia divergens* organism causing zoonotic babesiosis in Europe. *Emerging Infectious Diseases, 9*, 942−948.

Herwaldt, B. L., de Bruyn, G., Pieniazek, N. J., Homer, M., Lofy, K. H., Slemenda, S. B., et al. (2004). *Babesia divergens*-like infection, Washington State. *Emerging Infectious Diseases, 10*, 622−629.

Holbrook, M. R. (2012). Kyasanur forest disease. *Antiviral Research, 96*, 353−362.

Hoogstraal, H. (1985). Argasid and nuttallielid ticks as parasites and vectors. *Advances in Parasitology, 24*, 135–238.

Horak, I. G., Camicas, J. L., & Keirans, J. E. (2002). The Argasidae, Ixodidae and Nuttallielidae (Acari: Ixodida): A world list of valid tick names. *Experimental & Applied Acarology, 28*, 27–54.

Horn, M., Nussbaumerova, M., Sanda, M., Kovarava, Z., Srba, J., Franta, Z., et al. (2009). Hemoglobin digestion in blood-feeding ticks: Mapping a multipeptidase pathway by functional proteomics. *Chemistry & Biology, 16*, 1053–1063.

Hynes, W. L., Ceraul, S. M., Todd, S. M., Sequin, K. C., & Sonenshine, D. E. (2005). A defensin-like gene expressed in the black-legged tick, *Ixodes scapularis*. *Medical and Veterinary Entomology, 19*, 339–344.

ICTV. (2017). *International Commission on Taxonomy of Viruses*.

Inoue, N., Hanada, K., Tsuji, N., Igarashi, I., Nagasawa, H., Mikami, T., et al. (2001). Characterization of phagocytic hemocytes in *Ornithodoros moubata* (Acari: Ixodidae). *Journal of Medical Entomology, 38*, 514–519.

Ivanova, L. B., Tomova, A., González-Acuña, D., Murúa, R., Moreno, C. X., Hernández, C., et al. (2014). *Borrelia chilensis*, a new member of the *Borrelia burgdorferi* sensu lato complex that extends the range of this genospecies in the Southern Hemisphere. *Environmental Microbiology, 16*, 1069–1080.

Iwabu-Itoh, Y., Bazartseren, B., Naranbaatar, O., Yondonjamts, E., Furuno, K., Lee, K., et al. (2017). Tick surveillance for *Borrelia miyamotoi* and phylogenetic analysis of isolates in Mongolia and Japan. *Ticks and Tick-borne Diseases, 8*, 850–857.

James, A. M., Liveris, D., Wormser, G. P., Schwartz, I., Montecalvo, M. A., & Johnson, B. J. B. (2001). *Borrelia lonestari* infection after a bite by an *Amblyomma americanum* tick. *Journal of Infectious Diseases, 183*, 1810–1814.

Jasinskas, A., Zhong, J., & Barbour, A. G. (2007). Highly prevalent *Coxiella* sp. bacterium in the tick vector *Amblyomma americanum*. *Applied and Environmental Microbiology, 73*, 334–336.

Jellison, W. L. (1974). *Tularemia in North America 1930–1974*. Missoula, MT: University of Montana Foundation, 276 pp.

Jellison, W. L., & Kohls, G. M. (1955). Tularemia in sheep and sheep industry workers in western United States. *Public Health Monograph, 28*, 1–17.

Johns, R., Sonenshine, D. E., & Hynes, W. L. (1998). Control of bacterial infections in the hard tick *Dermacentor variabilis* (Acari: Ixodidae): Evidence for the existence of antimicrobial proteins in tick hemolymph. *Journal of Medical Entomology, 35*, 458–464.

Johnson, D. K. H., Schiffman, E. K., Davis, J. P., Neitzel, D. F., Sloan, L. M., Nicholson, W. L., et al. (2015). Human infection with *Ehrlichia muris*-like pathogen, United States, 2007–2013. *Emerging Infectious Diseases, 21*, 1794–1799.

Jones, L. D., Hodgson, E., Williams, T., Higgs, S., & Nuttall, P. A. (1992). Saliva-activated transmission (SAT) of Thogoto virus: Relationship with vector potential of different haematophagous arthropods. *Medical and Veterinary Entomology, 6*, 261–265.

Jongejan, F., & Uilenberg, G. (2004). The global importance of ticks. *Parasitology, 129*, S3–S14.

Jori, F., Vial, L., Penrith, M. L., Pérez-Sánchez, R., Etter, E., Albina, E., et al. (2013). Review of the sylvatic cycle of African swine fever in sub-Saharan Africa and the Indian ocean. *Virus Research, 173*, 212–227.

Kazimírová, M., & Štibrániová, I. (2013). Tick salivary compounds: Their role in modulation of host defences and pathogen transmission. *Frontiers in Cellular and Infection Microbiology, 3*, 43.

Keirans, J. E. (1992). Systematics of the Ixodida (Argasidae, Ixodidae, Nuttalliellidae); an overview and some problems. In B. Fivaz, T. Petney, & I. G. Horak (Eds.), *Tick vector biology: Medical and veterinary aspects* (pp. 1–21). Berlin: Springer-Verlag.

Keirans, J. E., & Clifford, C. M. (1978). The genus *Ixodes* in the United States: A scanning electron microscope study and key to the adults. *Journal of Medical Entomology*, (Suppl. 2), 149 pp.

Keirans, J. E., Clifford, C. M., Hoogstraal, H., & Easton, E. R. (1976). Discovery of *Nuttalliella namaqua* Bedford (Acarina: Ixodoidea: Nuttalliellidae) in Tanzania and re-description of the female based on scanning electron microscopy. *Annals of the Entomological Society of America, 69*, 926–932.

Kelly, P. J., Lucas, H. M., Randolph, C. M., Ackerson, K., Blackburn, J. K., & Dark, M. J. (2014). Efficacy of slow-release tags impregnated with aggregation-attachment pheromone and deltamethrin for control of *Amblyomma variegatum* on St. Kitts, West Indies. *Parasites & Vectors, 7*, 182.

Khasnatinov, M. A., Danchinova, G. A., Takano, A., Kawabata, H., Ohashi, N., & Masuzawa, T. (2016). Prevalence of *Borrelia miyamotoi* in *Ixodes persulcatus* in Irkutsk City and its neighboring territories, Russia. *Ticks and Tick-borne Diseases, 7*, 394–397.

King, D. P., Chen, C. I., Blanchard, M. T., Aldridge, B. M., Anderson, M., Walker, R., et al. (2005). Molecular identification of a novel deltaproteobacterium as the etiologic agent of epizootic bovine abortion (foothill abortion). *Journal of Clinical Microbiology, 43*, 604–609.

Kjemtrup, A. M., Wainwright, K., Miller, M., Penzhorn, B. L., & Carreno, R. A. (2006). *Babesia conradae*, sp. nov., a small canine *Babesia* identified in California. *Veterinary Parasitology, 138*, 103–111.

Klompen, J. H. S., Black, W. C., Keirans, J. E., & Norris, D. E. (2000). Systematics and biogeography of hard ticks, a total evidence approach. *Cladistics, 16*, 79–102.

Klompen, J. H. S., Black, W. C., IV, Keirans, J. E., & Oliver, J. H., Jr. (1996). Evolution of ticks. *Annual Review of Entomology, 41*, 141–161.

Klompen, J. H. S., & Oliver, J. H., Jr. (1993). Systematic relationships in the soft ticks (Acari: Ixodida: Argasidae). *Systematic Entomology, 18*, 313–331.

Kocan, K. M., de la Fuente, J., Blouin, E. F., Coetzee, J. F., & Ewing, S. A. (2010). The natural history of *Anaplasma marginale*. *Veterinary Parasitology, 167*, 95–107.

Köhler, W. (2006). Killed in action: Microbiologists and clinicians as victims of their occupation Part 4: Tick-borne relapsing fever, Malta fever, glanders, SARS. *International Journal of Medical Microbiology, 296*, 1–4.

Korenberg, E. I., & Kovalevskii, Y. V. (1999). Main features of tick-borne encephalitis eco-epidemiology in Russia. *Zentralblatt Bakteriol, 289*, 525–539.

Kosoy, O. I., Lambert, A. J., Hawkinson, D. J., Pastula, D. M., Goldsmith, C. S., Hunt, D. C., et al. (2015). Novel Thogotovirus associated with febrile illness and death, United States, 2014. *Emerging Infectious Diseases, 21*, 760–764.

Krause, P. J., Fish, D., Narasimhan, S., & Barbour, A. G. (2015). *Borrelia myiamotoi* infection in nature and in humans. *Clinical Microbiology and Infectious Diseases, 21*, 631–639.

Krause, P. J., Grant-Kels, J. M., Tahan, S. R., Dardick, K. R., Alarcon-Chaidez, F., Bouchard, K., et al. (2009). Dermatologic changes induced by repeated. *Ixodes scapularis* bites and implications for prevention of tick-borne infection. *Vector Borne and Zoonotic Diseases, 9*, 603–610.

Kunze, U., & International Scientific Working Group on Tick-Borne Encephalitis. (2007). Tick-borne encephalitis: From epidemiology to vaccination recommendations in 2007. New issues − best practices. *Wiener Medizinische Wochenschrift, 157*, 228−232.

Kuo, M. M., Lane, R. S., & Giclas, P. C. (2000). A comparative study of mammalian and reptilian alternative pathway of complement-mediated killing of the Lyme disease spirochete (*Borrelia burgdorferi*). *Journal of Parasitology, 86*, 1223−1228.

Kurtenbach, K., De Michelis, S., Etti, S., Schafer, S. M., Sewell, H. S., Brade, V., et al. (2002). Host association of *Borrelia burgdorferi* sensu lato−the key role of host complement. *Trends in Microbiology, 10*, 74−79.

Kurtenbach, K., Sewell, H.-S., Ogden, N. H., Randolph, S. E., & Nuttall, P. A. (1998). Serum complement sensitivity as a key factor in Lyme disease ecology. *Infection and Immunity, 66*, 1248−1251.

Labuda, M., Kozuch, O., Zuffova, E., Elecková, E., Hails, R. S., & Nuttall, P. A. (1997). Tick-borne encephalitis virus transmission between ticks cofeeding on specific immune natural rodent hosts. *Virology, 235*, 138−143.

Labuda, M., Trimnell, A. R., Lickova, M., Kazimirova, M., Davies, G. M., Lissina, O., et al. (2006). An antivector vaccine protects against a lethal vector-borne pathogen. *PloS Pathogens, 2*, e27.

Lai, R., Lomas, L. O., Jonczy, J., Turner, P. C., & Rees, H. H. (2004). Two novel non-cationic defensin-like antimicrobial peptides from hemolymph of the female tick, *Amblyomma hebraeum*. *Biochemical Journal, 379*, 681−685.

Lane, R. S. (1994). Competence of ticks as vectors of microbial agents with an emphasis on *Borrelia burgdorferi*. In D. E. Sonenshine, & T. N. Mather (Eds.), *Ecological dynamics of tick-borne zoonoses* (pp. 45−67). New York: Oxford University Press.

Lane, R. S., Burgdorfer, W., Hayes, S. F., & Barbour, A. G. (1985). Isolation of a spirochete from the soft tick, *Ornithodoros coriaceus*: A possible agent of epizootic bovine abortion. *Science, 230*, 85−87.

Lane, R. S., Emmons, R. W., Devlin, V., Dondero, D. V., & Nelson, B. C. (1982). Survey for evidence of Colorado tick fever virus outside of the known endemic area in California. *American Journal of Tropical Medicine and Hygiene, 31*, 837−843.

Lane, R. S., & Manweiler, S. A. (1988). *Borrelia coriaceae* in its tick vector, *Ornithodoros coriaceus* (Acari: Argasidae), with emphasis on transstadial and transovarial infection. *Journal of Medical Entomology, 25*, 172−177.

Lane, R. S., Mun, J., Eisen, L., & Eisen, R. J. (2006). Refractoriness of the western fence lizard (*Sceloporus occidentalis*) to the Lyme disease group spirochete *Borrelia bissettii*. *Journal of Parasitology, 92*, 691−696.

Lane, R. S., Mun, J., Eisen, R. J., & Eisen, L. (2005). Western gray squirrel (Rodentia: Sciuridae): A primary reservoir host of *Borrelia burgdorferi* in Californian oak woodlands? *Journal of Medical Entomology, 42*, 388−396.

Lane, R. S., Peek, J., & Donaghey, P. J. (1984). Tick (Acari: Ixodidae) paralysis in dogs from northern California: Acarological and clinical findings. *Journal of Medical Entomology, 21*, 321−326.

Lane, R. S., & Quistad, G. B. (1998). Borreliacidal factor in the blood of the western fence lizard (*Sceloporus occidentalis*). *Journal of Parasitology, 84*, 29−34.

Lara, F. A., Lins, U., Paiva-Silva, G., Almeida, I. C., Braga, C. M., Miguens, F. C., et al. (2003). A new intracellular pathway of haem detoxification in the midgut of the cattle tick *Boophilus microplus*: Aggregation inside a specialized organelle, the hemosome. *Journal of Experimental Biology, 206*, 1707−1715.

Larson, M. A., Fey, P. D., Hinrichs, S. H., & Iwen, P. C. (2014). *Francisella tularensis* bacteria associated with feline tularemia in the United States. *Emerging Infectious Diseases, 20*, 2068−2071.

Latif, A. A., Putterill, J. F., de Klerk, D. G., Pienaar, R., & Mans, B. J. (2012). *Nuttalliella namaqua* (Ixodoidea: Nuttalliellidae): First description of the male, immature stages and re-description of the female. *PLoS One, 7*, e41651.

Lees, K., & Bowman, A. S. (2007). Tick neurobiology: Recent advances and the post-genomic era. *Invertebrate Neuroscience, 7*, 183−198.

Levin, M., Levine, J. F., Yang, S., Howard, P., & Apperson, C. S. (1996). Reservoir competence of the southeastern five-lined skink (*Eumeces inexpectatus*) and the green anole (*Anolis carolinensis*) for *Borrelia burgdorferi*. *American Journal of Tropical Medicine and Hygiene, 54*, 92−97.

Li, H., Zheng, Y.-C., Ma, L., Jia, N., Jiang, B.-G., Jiang, R.-R., et al. (2015). Human infection with a novel tick-borne *Anaplasma* species in China: A surveillance study. *Lancet Infect. Dis., 15*, 663−670.

Liu, Q., He, B., Huang, S.-Y., Wei, F., & Zhu, X.-Q. (2014). Severe fever with thrombocytopenia syndrome, an emerging tick-borne zoonosis. *Lancet Infect. Dis, 14*, 763−772.

LoGiudice, K., Ostfeld, R. S., Schmidt, K. A., & Keesing, F. (2003). The ecology of infectious disease: Effects of host diversity and community composition on Lyme disease risk. *Proceedings of the National Academy of Sciences of the United States of America, 100*, 567−571.

Lomas, L. O., Turner, P. C., & Rees, H. H. (1997). A novel neuropeptide-endocrine interaction controlling ecdysteroid production in ixodid ticks. *Proceedings of the Biological Sciences, 264*, 589−596.

Lopez, J. E., Krishnavahjala, A., Garcia, M. N., & Bermudez, S. (2016). Tick-borne relapsing fever spirochetes in the Americas. *Veterinary Science, 3*, 1−18.

Lysyk, T. J., & Majak, W. (2003). Increasing the paralyzing ability of a laboratory colony of *Dermacentor andersoni* Stiles. *Journal of Medical Entomology, 40*, 185−194.

Machado-Ferreira, E., Vizzoni, V. F., Balsemão-Pires, E., Moerbeck, L., Gazeta, G. S., Piesman, J., et al. (2016). *Coxiella* symbionts are widespread into hard ticks. *Parasitology Research, 115*, 4691−4699.

Mahan, S. M. (2006). Diagnosis and control of heartwater, *Ehrlichia ruminantium*, infection: An update. *CAB Reviews: Perspectives in Agriculture, Veterinary Science, Nutrition and Natural Resources, 1*(no. 055), 12 pp.

Mani, R. J., Morton, R. J., & Clinkenbeard, K. D. (2016). Ecology of tularemia in central US endemic region. *Current Tropical Medicine Reports, 3*, 75−79.

Mans, B. J., de Klerk, D., Pienaar, R., & Latif, A. A. (2011). *Nuttalliella namaqua*: A living fossil and closest relative to the ancestral tick lineage: Implications for the evolution of blood-feeding in ticks. *PLoS One, 6*, e23675.

Mans, B. J., Goethe, R., & Neitz, A. W. H. (2004). Biochemical perspectives on paralysis and other forms of toxicoses caused by ticks. *Parasitology, 129*, S95−S111.

Mans, B. J., de Klerk, D., Pienaar, R., de Castro, M. H., & Latif, A. A. (2012). The mitochondrial genomes of *Nuttalliella namaqua* (Ixodoidea: Nuttalliellidae) and *Argas africolumbae* (Ixodoidae: Argasidae): Estimation of divergence dates for the major tick lineages and reconstruction of ancestral blood-feeding characters. *PLoS One, 7*(11), e49461.

Mansfield, K. L., Jizhou, L., Phipps, L. P., & Johnson, N. (2017). Emerging tick-borne viruses in the twenty-first century. *Frontiers in Cellular and Infection Microbiology, 7*, 298.

Marfin, A. A., & Campbell, G. L. (2005). Colorado tick fever and related *Coltivirus* infections. In J. L. Goodman, D. T. Dennis, & D. E. Sonenshine (Eds.), *Tick-borne diseases of humans* (pp. 143–149). Washington, DC: ASM Press.

Margos, G., Gatewood, A. G., Aanensen, D. M., Hanincova, K., Terekhova, D., Vollmer, S. A., et al. (2008). MLST of housekeeping genes captures geographic population structure and suggests a European origin of *Borrelia burgdorferi*. *Proceedings of the National Academy of Sciences of the United States of America, 105*, 8730–8735.

Mather, T. N., Wilson, M. L., Moore, S. I., Ribeiro, J. M., & Spielman, A. (1989). Comparing the relative potential of rodents as reservoirs of the Lyme disease spirochete (*Borrelia burgdorferi*). *American Journal of Epidemiology, 130*, 143–150.

Matsuo, T., Cerruto Noya, C. A., Taylor, D., & Fujisaki, K. (2007). Immunohistochemical examination of PDGF-AB, TGF-beta and their receptors in the hemocytes of a tick, *Ornithodoros moubata* (Acari: Argasidae). *Journal of Veterinary Medical Science, 69*, 317–320.

Maurin, M., & Gyuranecz, M. (2016). Tularaemia: Clinical aspects in Europe. *Lancet Infectious Diseases, 16*, 113–124.

McCall, P. J., Hume, J. C. C., Motshegwa, K., Pignatelli, P., Talbert, A., & Kisinza, W. (2007). Does tick-borne relapsing fever have an animal reservoir in east Africa? *Vector Borne and Zoonotic Diseases, 7*, 659–666.

McConnell, J. (2003). Tick-borne relapsing fever under-reported. *Lancet Infectious Diseases, 3*, 604.

McCoy, K. D., Léger, E., & Dietrich, M. (2013). Host specialization in ticks and transmission of tick-borne diseases: A review. *Frontiers in Cellular and Infection Microbiology, 3*, 57.

Mehlhorn, H., Raether, W., Schein, E., Weber, M., & Uphoff, M. (1986). Licht- und elektronenmikroskopische Untersuchungen zumEntwicklungszyklus and Einfluss von Pentamidin auf die Morphologie der intraerythrocytären Stadienvon *Babesia microti*. *Deutsche tierärztliche Wochenschrift, 93*, 400–405.

Meinkoth, J. H., & Kocan, A. A. (2005). Feline cytauxzoonosis. *Veterinary Clinics of North America Small Animal Practice, 35*, 89–101.

Mendiola, J., Alonso, M., Marquetti, M. C., & Finlay, C. (1996). *Boophilus microplus*: Multiple proteolytic activities in the midgut. *Experimental Parasitology, 82*, 27–33.

Mitchell, R. D., 3rd, Zhu, J., Carr, A. L., Dhammi, A., Cave, G., Sonenshine, D. E., et al. (2017). Infrared light detection by the Haller's organ of adult American dog ticks, *Dermacentor variabilis* (Ixodida: Ixodidae). *Ticks and Tick-borne Diseases, 8*, 764–771.

Mitchell, R. E., 3rd, Ross, E., Osgood, C., Sonenshine, D. E., Donohue, K. V., Khalil, S. M., et al. (2007). Molecular characterization, tissue-specific expression and RNAi knockdown of the first vitellogenin receptor from a tick. *Insect Biochemistry and Molecular Biology, 37*, 375–388.

Molins, C. R., Ashton, L. V., Wormser, G. P., Andre, B. G., Hess, A. M., Delorey, M. J., et al. (2017). Metabolic differentiation of early Lyme disease from southern tick-associated rash illness (STARI). *Science Translational Medicine, 9*, eea12717.

Muehlenbachs, A., Bollweg, B. C., Schulz, T. J., Forrester, J. D., Carnes, M. D., Molins, C., et al. (2016). Cardiac tropism of *Borrelia burgdorferi*: An autopsy study of sudden cardiac death associated with Lyme carditis. *American Journal of Pathology, 186*, 1195–1205.

Murrel, A., & Barker, S. C. (2003). Synonomy of *Boophilus* Curtice, 1891 with *Rhipicephalus* Koch, 1844 (Acari: Ixodidae). *Systematic Parasitology, 56*, 169–172.

Murrel, A., Campbell, N. J., & Barker, S. C. (2000). Phylogenetic analyses of the rhipicephaline ticks indicate that the genus *Rhipicephalus* is paraphyletic. *Molecular Phylogenetics and Evolution, 16*, 1–7.

Nadolny, R. M., Wright, C. L., Hynes, W. L., Sonenshine, D. E., & Gaff, H. D. (2011). *Ixodes affinis* (Acari: Ixodidae) in southeastern Virginia and implications for the spread of *Borrelia burgdorferi*, the agent of Lyme disease. *Journal of Vector Ecology, 36*, 464–467.

Nakajima, Y., van der Goes van Naters-Yusui, A., Taylor, D., & Yamakawa, M. (2001). Two isoforms of a member of the arthropod defensin family from the soft tick, *Ornithodoros moubata* (Acari: Argasidae). *Insect Biochemistry and Molecular Biology, 31*, 747–751.

Nava, S., Estrada-Pena, A., Petney, T., Beati, L., Labruna, M. L., Szabo, M. P. J., et al. (2014). The taxonomic status of *Rhipicephalus sanguineus* (Latreille, 1806). *Veterinary Parasitology, 208*, 2–8.

Nelson, C. A., Saha, S., Kugeler, K. J., Delorey, M. J., Shankar, M. B., Hinckley, A. F., et al. (2015). Incidence of clinician-diagnosed Lyme disease, United States, 2005–2010. *Emerging Infectious Diseases, 21*, 1625–1631.

Nichols Heitman, K., Dahlgren, F. S., Drexler, N. A., Massung, R. F., & Behravesh, C. B. (2016). Increasing incidence of ehrlichiosis in the United States: A summary of national surveillance of *Ehrlichia chaffeensis* and *Ehrlichia ewingii* infections in the United States, 2008-2012. *American Journal of Tropical Medicine and Hygiene, 94*, 52–60.

Nicholson, W. L. (2017). Family Anaplasmataceae (anaplasmosis, ehrlichiosis, neorickettsiosis, and neoehrlichiosis). In S. S. Long, C. G. Prober, & M. Fischer (Eds.), *Principles and practice of pediatric infectious diseases* (5th ed., pp. 918–923). Philadelphia, PA: Elsevier.

Nieto, N. C., & Foley, J. E. (2008). Evaluation of squirrels (Rodentia: Sciuridae) as ecologically significant hosts for *Anaplasma phagocytophilum* in California. *Journal of Medical Entomology, 45*, 763–769.

Norval, R. A. I., Sonenshine, D. E., Allan, S. A., & Burridge, M. J. (1996). Efficacy of pheromone-acaricide impregnated tail-tag decoys for control of bont ticks, *Amblyomma hebraeum* on cattle in Zimbabwe. *Experimental & Applied Acarology, 20*, 31–46.

Nuss, G., Nuss, A. B., Meyer, J. M., Sonenshine, D. E., Roe, R. M., & Waterhouse, R. M. (2016). Genomic insights into the *Ixodes scapularis* tick vector of Lyme disease. *Nature Communications, 7*, 10507.

Nuttall, P. A., & Jones, L. D. (1991). Non-viraemic tick-borne virus transmission: Mechanism and significance. In F. Dusbabek, & V. Bukva (Eds.), *Modern acarology: Vol. 2. Proceedings, 8th Int. Congr. Acarol* (pp. 3–6). The Hague, The Netherlands: Academia Prague and SPB Academic Publishing.

Nuttall, P. A., & Labuda, M. (1994). Tick-borne encephalitis subgroup complex. In D. E. Sonenshine, & T. N. Mather (Eds.), *Ecological dynamics of tick-borne zoonoses* (pp. 351−391). New York: Oxford University Press.

Oliver, J. H., Jr., Owsley, M. R., Hutcheson, H. J., James, A. M., Chen, C., Irby, W. S., et al. (1993). Conspecificity of the ticks *Ixodes scapularis* and *I. dammini* (Acari: Ixodidae). *Journal of Medical Entomology, 30*, 54−63.

Ostfeld, R. S., Levi, T., Jolles, A. E., Martin, L. B., Hosseini, P. R., & Keesing, F. (2014). Life history and demographic drivers of reservoir competence for three tick-borne zoonotic pathogens. *PLoS One, 9*, e107387.

Paddock, C. D. (2005). *Rickettsia parkeri* as a paradigm for multiple causes of tick-borne spotted fever in the western hemisphere. *Annals of the New York Academy of Sciences, 1061*, 315−326.

Paddock, C. D., & Childs, J. E. (2003). *Ehrlichia chaffeensis*: A prototypical emerging pathogen. *Clinical Microbiology Reviews, 16*, 37−64.

Padgett, K. A., Bonilla, D., Eremeeva, M. E., Glaser, C., Lane, R. S., Porse, C. C., et al. (2016). The eco-epidemiology of Pacific Coast tick fever in California. *PLoS Neglected Tropical Diseases, 10*, e0005020.

Pages, F., Dautel, H., Duvallet, G., Kahl, O., de Gentile, L., & Boulanger, N. (2014). Tick repellents for human use: Prevention of tick bites and tick-borne diseases. *Vector-borne Zoonotic Dis, 14*, 85−93.

Parker, R. R., Philip, C. B., & Jellison, W. L. (1933). Potentialities of tick transmission in relation to geographical occurrence in the United States. *American Journal of Tropical Medicine and Hygiene, 13*, 341−379.

Parola, P., Paddock, C. D., & Raoult, D. (2005). Tick-borne rickettsioses around the world: Emerging diseases challenging old concepts. *Clinical Microbiology Reviews, 18*, 719−756.

Pau, U., Li, X., Wang, T., Montgomery, R. R., Ramamoorthy, N., DeSilva, A. M., et al. (2004). TROSPA, an *Ixodes scapularis* receptor for *Borrelia burgdorferi*. *Cell, 119*, 457−468.

Pegram, R. (2006). End of the Caribbean *Amblyomma* programme. *International Consortium on Ticks and Tick-borne Diseases*, (30), 4−6.

Pegram, R., Indar, L., Eddi, C., & George, J. (2004). The Caribbean *Amblyomma* program: Some ecological factors affecting its success. *Annals of the New York Academy of Sciences, 1026*, 302−311.

Perez de Leon, A. A., Teel, P. D., Auclair, A. N., Messenger, M. T., Guerrero, F. D., Schuster, G., et al. (2012). Integrated strategy for sustainable cattle fever tick eradication in USA is required to mitigate the impact of global change. *Frontiers in Physiology, 3*(195) (17 pp.).

Petersen, J. M., Mead, P. S., & Schreifer, M. E. (2009). *Francisella tularensis*: An arthropod-borne pathogen. *Veterinary Research, 40*, 07.

Petri, E., Gniel, D., & Zent, O. (2010). Tick-borne encephalitis (TBE) trends in epidemiology and current and future management. *Travel Medicine and Infectious Disease, 8*, 233−245.

Piesman, J. (2002). Ecology of *Borrelia burgdorferi* sensu lato in North America. In J. Gray, O. Kahl, R. S. Lane, & G. Stanek (Eds.), *Lyme borreliosis: Biology, epidemiology and control* (pp. 223−249). New York: CABI Publishing.

Piesman, J., & Gern, L. (2004). Lyme borreliosis in Europe and north America. *Parasitology, 129*, S191−S220.

Piesman, J., & Gray, J. (1994). Lyme disease/Lyme borreliosis. In D. E. Sonenshine, & T. N. Mather (Eds.), *Ecological dynamics of tick-borne zoonoses*. New York, NY: Oxford University Press.

Piesman, J., & Humair, P.-F. (2011). The spirochetes and vector ticks of Lyme borreliosis in nature. In S. Sood (Ed.), *Lyme borreliosis in Europe and North America* (pp. 37−51). Hoboken, NJ: John Wiley and Sons.

Pritt, B. S., Respicio-Kingry, L. B., Sloan, L. M., Schriefer, M. E., Replogle, A. J., Bjork, J., et al. (2016). *Borrelia mayonii* sp. nov., a member of the *Borrelia burgdorferi* sensu lato complex, detected in patients and ticks in the upper midwestern United States. *International Journal of Systematic and Evolutionary Microbiology, 66*, 4878−4880.

Ragagli, C., Bertolotti, L., Giacobini, M., Mannelli, A., Bisanzio, D., Amore, G., et al. (2011). Transmission dynamics of *Borrelia lusitaniae* and *Borrelia afzelii* among *Ixodes ricinus*, lizards, and mice in Tuscany, central Italy. *Vector-borne Zoonotic Dis, 11*, 21−28.

Randolph, S. E. (1995). Quantifying parameters in the transmission of *Babesia microti* by the tick *Ixodes trianguliceps* amongst voles (*Clethrionomys glareolus*). *Parasitology, 110*, 287−295.

Randolph, S. E. (2001). The shifting landscape of tick-borne zoonoses: Tick-borne encephalitis and Lyme borreliosis in Europe. *Philosophical Transactions of the Royal Society of London Series B: Biological Sciences, 356*, 1045−1056.

Randolph, S. E., Gern, L., & Nuttall, P. A. (1996). Co-feeding ticks: Epidemiological significance for tick-borne pathogen transmission. *Parasitology Today, 12*, 472−479.

Randolph, S. E., Miklisová, D., Lysy, J., Rogers, D. J., & Labuda, M. (1999). Incidence from coincidence: Patterns of tick infestations on rodents facilitate transmission of tick-borne encephalitis virus. *Parasitology, 118*, 177−186.

Randolph, S. E., Green, R. M., Peacey, M. F., & Rogers, D. J. (2000). Seasonal synchrony: The key to tick-borne encephalitis foci identified by satellite data. *Parasitology, 121*, 15−23.

Randolph, S. E., & Sumilo, D. (2007). Tick-borne encephalitis in Europe: Dynamics of changing risk. In W. Takken, & B. G. J. Knols (Eds.), *Emerging pests and vector-borne diseases in Europe* (pp. 187−206). Wageningen, Netherlands: Wageningen University Publishers.

Reeves, W. K., Streicker, D. G., Loftis, A. D., & Dasch, G. A. (2006). Serologic survey of *Eptesicus fuscus* from Georgia, U.S.A. for *Rickettsia* and *Borrelia* and laboratory transmission of a *Rickettsia* by bat ticks. *Journal of Vector Ecology, 31*, 386−389.

Ribeiro, J. M. C. (1989). Role of saliva in tick/host interactions. *Experimental & Applied Acarology, 7*, 15−20.

Ribeiro, J. M., Alarcon-Chaidez, F., Francischetti, I. M., Mans, B. J., Mather, T. N., Valenzuela, J. G., et al. (2006). An annotated catalog of salivary gland transcripts from *Ixodes scapularis* ticks. *Insect Biochemistry and Molecular Biology, 36*, 111−129.

Ribeiro, J. M., Slovak, M., & Francischetti, I. M. (2017). An insight into the sialome of *Hyalomma excavatum*. *Ticks and Tick-borne Diseases, 8*, 201−207.

Riemersma, K. K., & Komar, N. (2015). Heartland virus neutralizing antibodies in vertebrate wildlife, United States, 2009−2014. *Emerging Infectious Diseases, 21*, 1830−1833.

Roberts, F. H. S. (1970). *Australian ticks*. Melbourne: Commonwealth Scientific and Industrial Organization, 267 pp.

Rodriquez-Vivas, R. I., Jonsson, N. N., & Bhuhan, C. (2018). Strategies for the control of *Rhipicephalus microplus* ticks in a world of conventional acaricide and macrocyclic lactone resistance. *Parasitology Research, 117*, 3−29.

Rogers, D. J., & Randolph, S. E. (2006). Climate change and vector-borne diseases. *Advances In Parasitology, 62*, 345−381.

Rudenko, N., Golovchenko, M., Clark, K., Oliver, J. H., & Grubhoffer, L. (2016). Detection of *Borrelia burgdorferi* sensu stricto in *Amblyomma americanum* ticks in the southeastern United States: the case of selective compatibility. *Emerging Microbes and Infections, 5*, e48.

Rudenko, N., Golovchenko, M., Edwards, M. J., & Grubhoffer, L. (2005). Differential expression of *Ixodes ricinus* tick genes induced by blood feeding or *Borrelia burgdorferi* infection. *Journal of Medical Entomology, 42*, 36−41.

Schetters, T. P. M., & Brown, W. C. (Eds.). (2006), *Vet. Parasitol: Vol. 138. Special issue: Babesiosis* (pp. 1−168).

Schulze, T. L., Jordan, R. A., Williams, M., & Dolan, M. C. (2017). Evaluation of the SELECT Tick Control System (TCS), a host-targeted bait box, to reduce exposure to *Ixodes scapularis* (Acari: Ixodidae) in a Lyme disease endemic area of New Jersey. *Journal of Medical Entomology, 54*, 1019−1024.

Schutzer, S. E., Fraser-Liggett, C. M., Casjens, S. R., Qiu, W. G., Dunn, J. J., Mongodin, E. F., et al. (2011). Whole-genome sequences of thirteen isolates of *Borrelia burgdorferi*. *Journal of Bacteriology, 193*, 1018−1020.

Schutzer, S. E., Fraser-Liggett, C. M., Qiu, W. G., Kraiczy, P., Mongodin, E. F., Dunn, J. J., et al. (2012). Whole-genome sequences of *Borrelia bissettii*, *Borrelia valaisiana*, and *Borrelia spielmanii*. *Journal of Bacteriology, 194*, 545−546.

Schwan, T. G., Schrumpf, M. E., Hinnebusch, B. J., Anderson, D. E., Jr., & Konkel, M. E. (1996). GlpQ: an antigen for serological discrimination between relapsing fever and Lyme borreliosis. *Journal of Clinical Microbiology, 34*, 2483−2492.

Schwan, T. G., Policastro, P. F., Miller, Z., Thompson, R. L., Damrow, T., & Keirans, J. E. (2003). Tick-borne relapsing fever caused by *Borrelia hermsii*, Montana. *Emerging Infectious Diseases, 9*, 1151−1154.

Schwan, T. G., Raffel, S. J., Schrumpf, M. E., & Porcella, S. F. (2007). Diversity and distribution of *Borrelia hermsii*. *Emerging Infectious Diseases, 13*, 436−442.

Schwartz, A. M., Hinckley, A. F., Mead, P. S., Hook, S. A., & Kugeler, K. J. (2017). Surveillance for Lyme Disease − United States, 2008−2015. *MMWR Surveillance Summary, 66*, 1−12.

Scoles, G. A., & Ueti, M. W. (2015). Vector ecology of equine piroplasmosis. *Annual Review of Entomology, 60*, 561−580.

Scott, J. C., Wright, D. J. M., & Cutler, S. J. (2005). Typing African relapsing fever spirochetes. *Emerging Infectious Diseases, 11*, 1722−1729.

Sidi, G., Davidovitch, N., Balicer, R. D., Anis, E., Grotto, I., & Schwartz, E. (2005). Tickborne relapsing fever in Israel. *Emerging Infectious Diseases, 11*, 1784−1786.

Silaghi, C., Beck, R., Oteo, J. A., Pfeffer, M., & Sprong, H. (2016). Neoehrlichiosis: an emerging tick-borne zoonosis caused by *Candidatus* Neoehrlichia mikurensis. *Experimental & Applied Acarology, 68*, 279−297.

Solano-Gallego, L., Sainz, Á., Roura, X., Estrada-Peña, A., & Miró, G. (2016). A review of canine babesiosis: the European perspective. *Parasites & Vectors, 9*, 336.

Sonenshine, D. E. (1991). *Biology of ticks* (Vol. I). New York and Oxford: Oxford University Press, 447 pp.

Sonenshine, D. E., Adams, T., Allan, S. A., McLaughlin, J. R., & Webster, F. X. (2003). Chemical composition of some components of the arrestment pheromone of the black-legged tick, *Ixodes scapularis* (Acari: Ixodidae) and their use in tick control. *Journal of Medical Entomology, 40*, 849−859.

Sonenshine, D. E., Allan, S. A., Norval, R. A. I., & Burridge, M. J. (1996). A self-medicating applicator for control of ticks on deer. *Medical and Veterinary Entomology, 10*, 149−154.

Sonenshine, D. E., & Hynes, W. L. (2008). Molecular characterization and related aspects of the innate immune response in ticks. *Frontiers in Bioscience, 13*, 7046−7063.

Sonenshine, D. E., & Roe, R. M. (Eds.). *Biology of ticks* (2nd ed., Vol. 1). New York, NY: Oxford University Press, 540 pp.

Sonenshine, D. E., & Roe, R. M. (Eds.). *Biology of ticks* (2nd ed., Vol. 2). New York, NY: Oxford University Press, 491 pp.

Spielman, A., Wilson, M. L., Levine, J. F., & Piesman, J. (1985). Ecology of *Ixodes dammini*-borne human babesiosis and Lyme disease. *Annual Review of Entomology, 30*, 439−460.

Stafford, K. C., III (2007). Tick management handbook: An integrated guide for homeowners, pest control operators, and public health officials for the prevention of tick-associated diseases (revised edition). *Connecticut Agricultural Experiment Station Bull. No. 1010*, 84 pp. Available at: www.ct.gov/caes.

Starkey, L. A., Barrett, A. W., Beall, M. J., Chandrashekar, R., Thatcher, B., Tyrrell, P., et al. (2015). Persistent *Ehrlichia ewingii* infection in dogs after natural tick infestation. *Journal of Veterinary Internal Medicine, 29*, 552−555.

Steere, A. C., Coburn, J., & Glickstein, L. (2005). Lyme borreliosis. In J. L.Goodman, D. T. Dennis, & D. E. Sonenshine (Eds.), *Tick-borne diseases of humans* (pp. 176−206). Washington, D.C: ASM Press.

Steere, A. C., Malawista, S. E., Snydman, D. R., Shope, R. E., Andiman, W. A., Ross, M. R., et al. (1977). Lyme arthritis: an epidemic of oligoarticular arthritis in children and adults in three Connecticut communities. *Arthritis & Rheumatology, 20*, 7−17.

Steere, A. C., Strle, F., Wormser, G. P., Hu, L. T., Branda, J. A., Hovius, J. W., et al. (2016). Lyme borreliosis. *Nature Reviews. Disease Primers, 2*, 16090.

Suarez, C. E., & Noh, S. (2011). Emerging perspectives in the research of bovine babesiosis and anaplasmosis. *Veterinary Parasitology, 180*, 109−125.

Swei, A., Ostfeld, R. S., Lane, R. S., & Briggs, C. J. (2011). Impact of the experimental removal of lizards on Lyme disease risk. *Proceedings of the Royal Society of London B: Biological Sciences, 278*, 2970−2978.

Tabuchi, N., Kataoka-Ushijima, Y., Talbert, A., Mitani, H., & Fukunaga, M. (2008). Absence of transovarial transmission of *Borrelia duttonii*, a tick-borne relapsing fever agent, by the vector tick *Ornithodoros moubata*. *Vector Borne and Zoonotic Diseases, 8*, 607−613.

Telford, S. R., 3rd, Goethert, H. K., Molloy, P. J., Berardi, V. P., Chowdri, H. R., Gugliotta, J. L., et al. (2015). *Borrelia miyamotoi* disease: neither Lyme disease nor relapsing fever. *Clinics in Laboratory Medicine, 35*, 867−882.

Telford, S. R., 3rd, & Goethert, H. K. (2011). Toward an understanding of the perpetuation of the agent of tularemia. *Frontiers in Microbiology, 1*, 150.

Thomas, N. J., Bunikis, J., Barbour, A. G., & Wolcott, M. J. (2002). Fatal spirochetosis due to a relapsing fever-like *Borrelia* sp. in a northern spotted owl. *Journal of Wildlife Diseases, 38*, 187−193.

Thompson, D. M., Khalil, S. M., Jeffers, L. A., Ananthapadmanaban, U., Sonenshine, D. E., Mitchell, R. D., et al. (2005). In vivo role of 20-hydroxyecdysone in the regulation of the vitellogenin mRNA and egg development in the American dog tick, *Dermacentor variabilis* (Say). *Insect Physiology, 51*, 1105−1116.

Thompson, D. M., Khalil, S. M., Jeffers, L. A., Sonenshine, D. E., Mitchell, R. D., Osgood, C. J., et al. (2007). Sequence and the developmental and tissue-specific regulation of the first complete vitellogenin messenger RNA from ticks responsible for heme sequestration. *Insect Biochemistry and Molecular Biology, 37*, 363−374.

Tijsse-Klasen, E., Fonville, M., Reimerink, J. H., Spitzen-van der Sluijs, A., & Sprong, H. (2010). Role of sand lizards in the ecology of Lyme and other tick-borne diseases in the Netherlands. *Parasites & Vectors, 3*, 42.

Trager, W. (1939). Acquired immunity to ticks. *Journal of Parasitology, 25*, 57−81.

Trape, J. F., Diatta, G., Arnathau, C., Bitam, I., Sarih, M., Belghyti, D., et al. (2013). The epidemiology and geographic distribution of relapsing fever borreliosis in West and North Africa, with a review of the *Ornithodoros erraticus* complex (Acari: Ixodida). *PLoS One, 8*, e78473.

Uilenberg, G. (1995). International collaborative research: significance of tick-borne hemoparasitic diseases to world animal health. *Veterinary Parasitology, 57*, 19−41.

Uilenberg, G. (2006). *Babesia* − a historical overview. *Veterinary Parasitology, 138*, 3−10.

Van Nunen, S. (2015). Tick-induced allergies: mammalian meat allergy, tick anaplylaxis, and their significance. *Asia Pacific Allergy, 5*, 3−16.

Van Wye, J. E., Hsu, Y.-P., Terr, A. I., Lane, R. S., & Moss, R. B. (1991). Anaphylaxis from a tick bite. *New England Journal of Medicine, 324*, 777−778.

Valenzuela, J. G., Charlab, R., Mather, T. N., & Ribeiro, J. M. C. (2000). Purification, cloning and expression of a novel salivary anticomplement protein from the tick, *Ixodes scapularis*. *Journal of Biological Chemistry, 275*, 18717−18723.

Vaughn, M. F., Funkhouser, S. W., Lin, F.-C., Fine, J., Juliano, J. J., Apperson, C. S., et al. (2014). Long-lasting permethrin impregnated uniforms: a randomized-controlled trial for tick bite prevention. *American Journal of Preventive Medicine, 46*, 473−480.

Vázquez, M., Muehlenbein, C., Cartter, M., Hayes, E. B., Ertel, S., & Shapiro, E. D. (2008). Effectiveness of personal protective measures to prevent Lyme disease. *Emerging Infectious Diseases, 14*, 210−216.

Vial, L., Diatta, G., Tall, A., Ba, E. H., Bouganali, H., Durand, P., et al. (2006). Incidence of tick-borne relapsing fever in West Africa: longitudinal study. *Lancet, 368*, 37−43.

Waladde, S. M., & Rice, M. J. (1982). The sensory basis of tick feeding behavior. In F. D. Obenchain, & R. Galun (Eds.), *Physiology of ticks* (pp. 71−118). Oxford: Pergamon Press.

Waldenström, J., Lundkvist, Å., Falk, K. I., Garpmo, U., Bergström, S., Lindegren, G., et al. (2007). Migrating birds and tickborne encephalitis virus. *Emerging Infectious Diseases, 13*, 1215−1218.

Walker, A. R., & Lloyd, C. M. (1993). Experiments on the relationship between feeding of the tick *Amblyomma variegatum* (Acari: Ixodidae) and dermatophilosis skin disease in sheep. *Journal of Medical Entomology, 30*, 136−143.

Walker, J. B., Kierans, J. E., & Horak, I. G. (2000). *The genus* Rhipicephalus *(Acari: Ixodidae): A guide to the brown ticks of the world*. Cambridge: Cambridge University Press, 643 pp.

Wikel, S. K. (2014). Chapter 4. Tick-host interactions. In D. E. Sonenshine, & R. M. Roe (Eds.), *Biology of ticks* (2nd ed., pp. 88−128). New York, NY: Oxford University Press.

Williams, S. C., Stafford, K. C., III, Molaei, G., & Linske, M. A. (2018). Integrated control of nymphal *Ixodes scapularis*: effectiveness of white-tailed deer reduction, the entomopathogenic fungus *Metarhizium anisopliae*, and fipronil-based rodent bait boxes. *Vector-borne Zoonotic Dis, 18*, 55−64.

Wise, L. N., Kappmeyer, L. S., Mealey, R. H., & Knowles, D. P. (2013). Review of equine piroplasmosis. *Journal of Veterinary Internal Medicine, 27*, 1334−1346.

Wormser, G. P., Masters, E., Liveris, D., Nowakowski, J., Nadelman, R. B., Holmgren, D., et al. (2005). Microbiologic evaluation of patients from Missouri with erythema migrans. *Clinical Infectious Diseases, 40*, 423−428.

Yabsley, M. J., Parsons, N. J., Horne, E. C., Shock, B. C., & Purdee, M. (2012). Novel relapsing fever *Borrelia* detected in African penguins (*Spheniscus demersus*) admitted to two rehabilitation centers in South Africa. *Parasitology Research, 110*, 1125−1130.

Yabsley, M. J., & Shock, B. C. (2013). Natural history of zoonotic *Babesia*: role of wildlife reservoirs. *Intl. J. Parasitology: Parasites & Wildl, 2*, 18−31.

Zung, J. L., Lewengrub, S., & Rudzinska, M. A. (1989). Fine structural evidence for the penetration of the Lyme disease spirochete *Borrelia burgdorferi* through the gut and salivary tissues of *Ixodes dammini*. *Canadian Journal of Zoology, 67*, 1737−1748.

Chapter 28

Molecular Tools Used in Medical and Veterinary Entomology

Dana Nayduch[1], Rebecca Trout Fryxell[2], and Pia Untalan Olafson[3]

[1]*USDA-ARS, Arthropod-borne Animal Diseases Research Unit, Manhattan, KS, United States;* [2]*Department of Entomology and Plant Pathology, University of Tennessee, Knoxville, TN, United States;* [3]*USDA-ARS, Livestock Arthropod Pests Research Unit, Kerrville, TX, United States*

Researchers are increasingly applying molecular tools to conventional medical and veterinary entomology research areas such as phylogenetics, vector competence and vector–host–pathogen interactions, pesticide resistance, behavior, ecology, physiology, reproduction, and development. Many of these techniques have been adapted for use in applied research, including rapid and simple detection of vector-borne pathogens, in both arthropods and their hosts, and methods for controlling and mitigating vectors and vector-borne diseases. This chapter provides an introduction to molecular techniques commonly used in medical and veterinary entomological research at the time of publication, including examples of current applications and a list of references as illustrative studies.

Using Koch's postulates to confirm vector-borne pathogens and diseases Criteria used to incriminate a microbe as a causative agent of a disease are known as Koch's postulates. Here, we have modified the postulates to demonstrate their use in implicating an arthropod as a vector of a human-borne pathogen with molecular techniques.

- *First, infected vectors must be associated with all cases of the disease*. The suspected vector must be collected in locations associated with infected hosts.
- *Second, the pathogen must be isolated and identified within the vector*. Molecular or nonmolecular techniques are used to isolate and identify the pathogen, including its genotype, from suspected vectors. Genotypes should be identical to those in the infected host population.
- *Third, a naïve, noninfected vector must be able to acquire the pathogen from an infected host*. The naïve vector must become infected with the same pathogen present in the infected host. Vector infection can be confirmed via standard or molecular techniques.
- *Fourth, the infected vector must transmit the pathogen to a naïve host and cause disease*. Transmission of the identical pathogen is confirmed by genotyping techniques. Molecular signatures of the pathogen isolated from the infected host must be identical to the infected vector. Infection can also be confirmed directly or indirectly by immunological techniques.

For human diseases, satisfying Koch's postulates for vector-borne diseases is often very difficult because infected vectors are likely to appear before the first disease case occurs, and experimentally testing infection would involve participation of human volunteers. For these reasons, established surveillance measures for detecting infected vectors and hosts are paramount in preventing and managing potential outbreaks. Surveillance data originate from a variety of sources that use the techniques described later and benefit from protocols that enable detection of infected vectors that often are in low abundance, even during outbreaks.

The world is experiencing an alarming increase in the number of vector-borne disease cases (WHO, 2014). Consequently, rapid and accurate detection of vector-borne pathogens is imperative in human and veterinary medicine. Techniques used for pathogen detection include traditional, immunological, molecular, and computational. Many of these techniques such as gene amplification and sequencing, detecting pesticide resistance, engineering DNA, evaluating efficacy of control, and evolutionary analyses are used in other medical and veterinary entomology applications. Here, we focus on detection of pathogens in vectors and hosts using molecular tools, and briefly touch on other nonmolecular techniques.

MOLECULAR TECHNIQUES

Polymerase chain reaction The most common methods for the detection of pathogens use the polymerase chain

Medical and Veterinary Entomology. https://doi.org/10.1016/B978-0-12-814043-7.00028-5

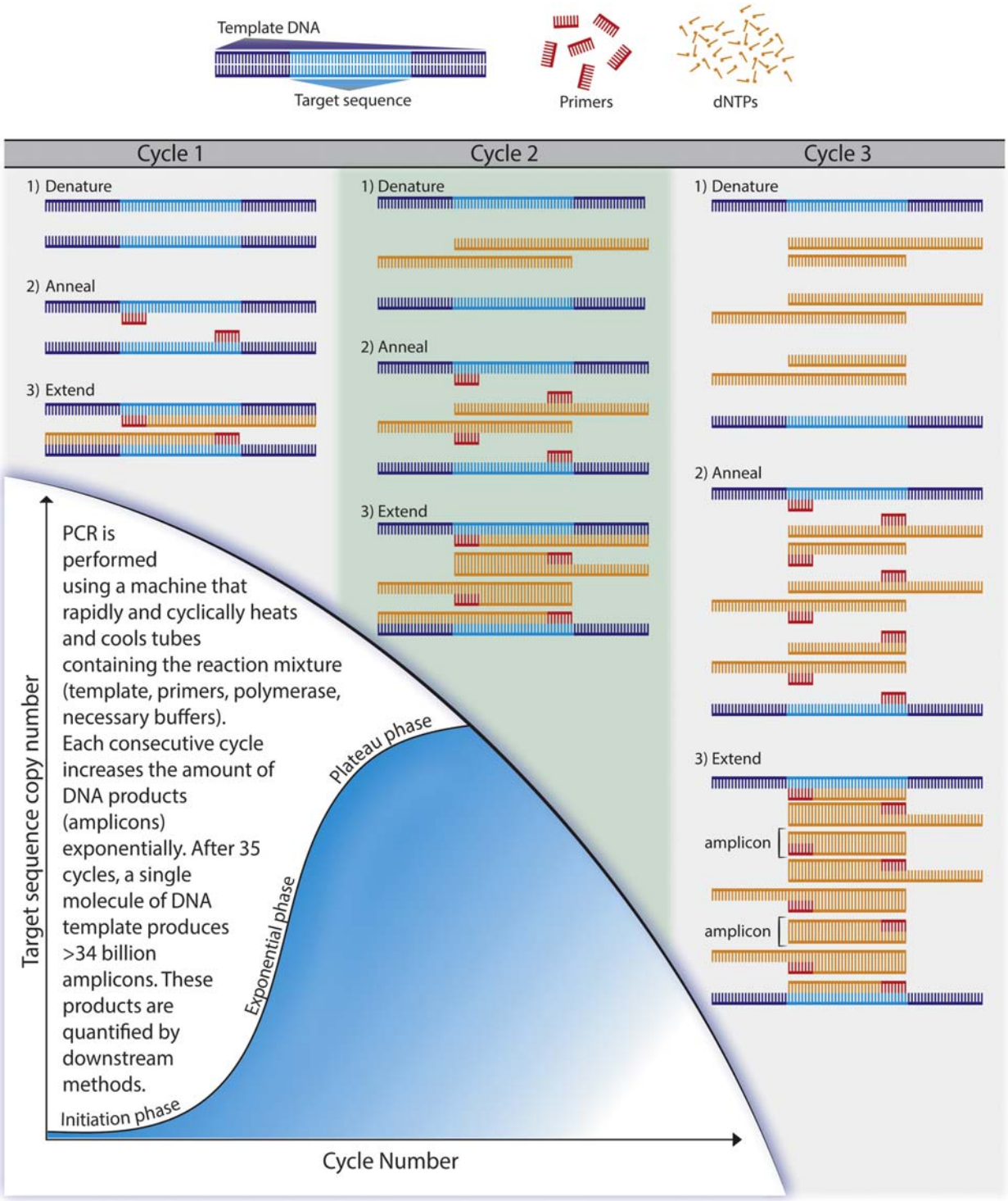


FIGURE 28.1 **The polymerase chain reaction (PCR).** PCR is a method that has numerous applications in the molecular studies of organisms. The technique is used to amplify DNA from a template, and it includes all the key players in cellular DNA replication: nucleotides (*dNTPs*), the building blocks of DNA; *primers*, short oligonucleotides that "prime" DNA synthesis to begin in certain locations on the template (flanking the target); a heat-stable polymerase enzyme (such as *Taq* polymerase) to perform the DNA synthesis (not shown). Sequential cycles of replication are achieved in three steps: (1) *denaturation*, in which DNA is heated to separate the template strands; (2) *annealing*, in which the reaction is cooled to allow primer binding to the template by base-pairing rules; and (3) *extension*, in which the polymerase will add complementary nucleotides to the growing strand, using the template as a guide. The "chain reaction" involves consecutive rounds of amplification cycles, with each cycle increasing the amount of DNA product (*amplicon*) exponentially ($1 \rightarrow 2 \rightarrow 4 \rightarrow 8$). PCR amplicons are analyzed in downstream techniques outlined in this chapter. *Illustration by Victoria Rhodes.*

reaction (PCR). A detailed description of the technique is provided in Fig. 28.1. PCR is based on in vivo DNA replication, a biological process that uses a DNA template and DNA polymerase to synthesize new and complementary DNA. In the laboratory, PCR is used to detect pathogens harbored within a vector or host by specifically amplifying products (**amplicons**) of the targeted DNA (i.e., the pathogen's gene) amid the total DNA extracted from the specimen (i.e., host, pathogen, and any other DNA in the specimen); this specificity is made possible by target-specific **primers**. In addition to detecting pathogens, PCR has been used to identify host DNA in mosquito bloodmeals (Ngo and Kramer, 2003). PCR has benefits compared with more classic approaches, like culture or microscopy. The samples (1) do not need to be fresh or intact, (2) can be stored frozen and/or in ethanol for later analysis, and (3) can be derived from small pieces of precious voucher or museum specimens because sensitivity and specificity are hallmarks of PCR. PCR followed by amplicon purification and **DNA sequencing** (discussed later in this chapter) can be used on its own to detect and identify pathogens in a specimen. Other applications of PCR include amplified fragment length polymorphisms (AFLP-PCR), random amplification of polymorphic DNA (RAPD), multiplex PCR, real-time PCR (quantitative), and reverse transcription−PCR (RT-qPCR). Table 28.1 summarizes these techniques.

AFLP-PCR uses **restriction enzymes (REs)** to recognize certain DNA sequences and only cut at those restriction sites. Each RE has a specific recognition sequence (e.g., *EcoRI* cuts DNA at 5′-G/AATTC-3′), and the presence/absence of these sites varies between organisms. Thus, DNA cut with REs yields variable fragment sizes across individuals. Once cut, small DNA adaptors are joined (**ligated**) to the ends of these fragments that are subsequently PCR-amplified using a primer set complementary to the adaptor and restriction site. The fragments are then size-separated by electrophoresis, resulting in unique sizes of fragments (banding patterns) on the gel, which are often termed "fingerprints" because the pattern is specific to that organism or strain. For diagnostics, banding patterns of the specimen can be compared with a known control to identify pathogens of interest in a sample. AFLP is also used in population genetics, because organisms with bands in common likely share sequences (alleles) and therefore are more closely related.

PCR-RFLP is commonly used to identify single nucleotide polymorphisms **(SNPs)** that may be associated with specific populations of pathogens or vectors, including distinct species, biotypes, or genotypes. The technique uses PCR to first amplify a genetic region and a restriction enzyme to make cuts in that amplicon. The RE-digested product is separated on a gel, and the banding pattern is observed. As with AFLP, the pattern can aid in pathogen/organism detection. Prior knowledge of the initial target genetic region being amplified is necessary for assay development (primer identification, cycling conditions, and selection of an appropriate restriction enzyme to give unique banding patterns). Previous research used PCR-RFLP to genotype Old and New World *Leishmania* spp. (Marfurt et al., 2003).

Alternatively, **RAPD-PCR** is a convenient technique that does not require any specific knowledge of the DNA sequence of the organism; however, the specimen must be considered "clean" of any other competing templates or contaminants such as a pathogen or bloodmeal. RAPD-PCR is not typically used to detect pathogens in vectors but rather to study the vectors themselves and can be important in population genetics and surveillance. A mixture of random primers (∼8−12 nucleotides long) are used to amplify genetic regions of a specimen's DNA. The position where primers anneal to the target DNA varies due to polymorphisms across individuals, resulting in unique amplicon sizes that are reflected as DNA fingerprints. The applications are similar to AFLP-PCR and PCR-RFLP.

Multiplex PCR is a PCR technique in which multiple sets of primers are used to target two or more genetic regions for simultaneous amplification in a single reaction. Typically, multiplex PCR is used in (1) genotyping, when simultaneous analysis of multiple markers is required, (2) detecting pathogens in vectors, and (3) **microsatellite analysis** in population genetics. This method was used to identify *Cuterebra* of *Peromyscus* mice (Noël et al., 2004). Primers can be designed to detect genetic deletions, mutations, and polymorphisms in genotyping assays (Domingues et al., 2014; Guerrero et al., 2001, 2002). By constructing primers that target more than one pathogen-associated gene, the user can screen and identify multiple pathogens in a single sample reaction. Microsatellites are small stretches of repeating units of DNA (e.g., CACACA or CAGCAGCAG); the numbers of repeats in a unit can differ between individuals and serve as molecular markers in population genetic studies. Thus, primers are designed to flank these regions and PCR results in distinct amplicon sizes; results from a collection of different microsatellite regions can then be used to estimate population relatedness. Multiplex assays require tedious and lengthy optimization procedures because the primers, reagents, and cycling conditions must be optimized for the genetic regions being analyzed.

Nested PCR involves secondary PCRs on amplicons from a primary PCR. The first PCR is conducted with a gene-specific set of primers, and this amplicon serves as the template for a second PCR with primers designed to a genetic region internal to those primary PCR primers—hence, "nested" within the primary amplicon and smaller in fragment size. The goals of nested PCR are to maximize product yield and confirm results of the primary reaction.

TABLE 28.1 Advantages and Disadvantages of Techniques Used to Identify Pathogens in Biological Samples

Technique	Approach	Advantages	Disadvantages
PCR	Primers amplify targeted genetic region	• Sensitive and specific	• Requires previous sequence information • Need sequencing to confirm
AFLP-PCR	Restriction enzyme digest followed by PCR for fingerprinting	• Does not require previous sequence information	• Only generates dominant markers • Needs clean sample
PCR-RFLP	PCR followed by a restriction digest for unique pattern	• Can screen mutations quickly and efficiently	• Requires sequence knowledge
RAPD-PCR	Random primers used in PCR give unique pattern	• Does not require previous sequence information	• Need clean and high-quality DNA
Multiplex-PCR	Simultaneous amplification of more than one gene fragment	• Reduces time, cost, and increases efficiency • Can distinguish more than one genetic region	• Challenging to develop because of specificity and sensitivity in assay design
Nested PCR	Two PCRs, use amplicon of first reaction as template in second	• Increased specificity and sensitivity	• Extra step (time and reagents) • Need to develop two primer sets
Real-time PCR	Uses florescent probe or SYBR Green to detect amplicon	• Increased specificity, sensitivity, speed • Visually detect samples in real time • Can quantify product	• Expensive equipment and reagents • Requires sequence knowledge • Can be challenging to develop efficient primers
LAMP	Uses *Bst* polymerase and 4–6 primers to synthesize DNA/RNA	• Single temperature, visually detect samples • Specific and sensitive • Relatively inexpensive	• Requires sequence knowledge • Difficult to develop because of specificity and sensitivity in assay design
Slide Mount	Visually detect pathogen under a microscope	• Relatively inexpensive • Rapid with very few reagents	• Requires specialized training in pathogen identification • Must have high-quality specimen • Reduced sensitivity and specificity • Can be subjective
Culture	Culture pathogen in selective media or cell culture	• Expensive molecular reagents not needed • Viable pathogen cultures available for downstream testing	• Not useable for unculturable organisms • Reduced sensitivity compared to molecular • Chance for cross-contamination • Exposes user to live pathogen
ELISA	Use antibody to detect pathogen or pathogen antigen to detect antibody	• Time efficient • Can standardize and quantify	• False positive rate, may indicate previous exposure not infection • Antibodies may cross-react (reduced specificity)

This assumes that the laboratory provides the appropriate personnel and equipment.

These assays are highly sensitive and specific, making them useful in detecting pathogenic microbes in vectors and hosts. Further, a primary PCR can reveal the presence of a bacterial genus in an initial screen (e.g., amplifies any *Ehrlichia* spp.), while a nested PCR using species-specific primers can be used to confirm the species identity (e.g., primers that amplify only *Ehrlichia chaffeensis*; Inayoshi et al., 2004).

Real-time PCR monitors the abundance of amplicons accumulating during PCR cycling in "real time," and it can be used to precisely quantify the amount of amplicon being produced (quantitative PCR [qPCR]). A specialized instrument, known as a real-time PCR or qPCR machine or cycler, simultaneously runs the PCR amplification and monitors amplicon production virtually (e.g., on a monitor) during each cycle of the reaction. Two benefits of real-time PCR are (1) the results are obtained quickly and sensitively and (2) the technique can be used to quantify the starting amount of target-gene template in the sample by comparison with a standard curve of known amounts of DNA. The instrument monitors amplicon production resulting from either probe-based or SYBR Green–based assays. In probe-based real-time assays, short DNA probes (e.g., a TaqMan Probe) anneal to a specific region of the template that is internal to the forward and reverse PCR primers. This probe can be constructed several ways, but most commonly it is designed to contain a high-energy dye (a fluorescent "reporter") at its 5′ end and a low-energy molecule ("quencher") at the 3′ end. When this probe is intact and bound to template, and excited by a light source, emission by the reporter dye is suppressed by being adjacent to the quencher dye. However, if primers anneal and DNA polymerase starts to copy DNA during the PCR extension phase, the annealed probe that sits in the polymerization path is cleaved by exonuclease activity of the polymerase and the quencher and reporter are separated. The separated reporter transmits a fluorescent signal that is subsequently detected and quantified by the instrument and software. An increase in the reporter signal over time indicates continued (exponential) amplification of product. Further, the amount of reporter signal increase is proportional to the amount of product, which is directly proportional to the amount of starting material (template) for a given sample.

Amplicon products also can be quantified by using SYBR Green chemistry. This fluorescent dye binds the smaller of the two grooves along the outer surface of a DNA molecule (**minor groove**) in double-stranded DNA. Thus, the SYBR Green fluorescent signal increases as more double-stranded product is synthesized in the reaction mixture. However, SYBR Green dye binds to *any* double-stranded DNA and therefore does not differentiate between specific and nonspecific PCR products. Ultimately, the choice of assay will depend on the user's experimental objectives and budget. Real-time PCR techniques also are used for gene expression analyses, termed qRT-PCR (described later in this chapter) and can be designed as multiplex assays. Multiplex real-time PCR has been used for pathogen detection in ticks (Venczel et al., 2016). Recently, droplet digital PCR has been used to detect rare and low-abundance molecules due to its enhanced sensitivity and resolution compared with RT-PCR (Baker, 2012; Hindson et al., 2013; Wilson et al., 2015).

Loop-mediated isothermal amplification (**LAMP**) and reverse-transcriptase loop-mediated isothermal amplification (RT-LAMP) have three distinct advantages over conventional PCR methods: (1) LAMP uses a single temperature instead of three temperatures, (2) detection is highly specific, and (3) results are visually detected without the use of expensive, sophisticated equipment, making it amenable for field use. In LAMP protocols, four primers and accompanying reagents are mixed with the DNA template and held at a single temperature for strand displacement. While PCR relies on heat denaturation of double-stranded DNA before the activity of DNA polymerase (*Taq*), LAMP uses a polymerase (*Bst*) that can displace double-stranded DNA and initiate complementary strand synthesis without denaturation. A byproduct of *Bst* polymerase DNA synthesis is magnesium pyrophosphate, produced from the reaction between pyrophosphates (released after nucleotide incorporation) and magnesium ions present in the solution buffers. The magnesium pyrophosphate byproduct forms a white precipitate whose increasing turbidity can be visually detected. Fluorescent molecules can also be incorporated that bind to LAMP byproducts, and these can be excited using a handheld ultraviolet (UV) light source (Fischbach et al., 2015). In contrast to qRT-PCR (described later in this chapter), reverse-transcriptase LAMP (RT-LAMP) uses six highly specific primers and reverse transcriptase for RNA virus detection to form cDNA from RNA. This is then followed by standard LAMP at the same temperature as the cDNA synthesis step. RT-LAMP has been used to detect dengue (Parida et al., 2005), Japanese encephalitis (Toriniwa and Komiya, 2006), and West Nile (Parida et al., 2004) viruses in mosquitoes.

NONMOLECULAR TECHNIQUES

Other techniques used to detect pathogens within vectors include traditional microscopy methods and immunological techniques. Traditional methods are relatively inexpensive and involve visually identifying the pathogen in the specimen. As a result, these techniques require technical and advanced training in specimen handling, processing, and pathogen identification. Simple light microscopy still is routinely used to visualize larger microbes in vector and host tissues. Examples include viewing protozoa such as

trypanosome flagellates or malaria parasites in insect midguts or host blood smears. While slide mounts can be used to detect larger pathogens, as they can be distinguished by their morphology, microbes such as viruses and bacteria are much smaller and less visually distinguishable, and need to be identified by laboratory culture and biochemical tests. Culturing vector-borne microbes can be especially laborious since many are obligate parasites that require cell culture techniques (rather than simple liquid or solid media preparations), as well as appropriate biosafety-level training and equipment.

Immunological techniques involve either direct or indirect detection of pathogens within a host or vector. Enzyme-linked immunosorbent assay (**ELISA**) is used to detect either host antibodies to pathogens or pathogen antigen in biological samples. To test for host antibodies, which indicates previous or current exposure to a pathogen, wells in a polystyrene microtiter plate are coated with antigens from the pathogen and host serum (containing antibodies) is added to the wells. If antibodies produced toward a specific pathogen are present in the host's serum (primary antibody), they will recognize and bind to the plated antigen. A second antibody is added that binds to the primary antibody, if present. An enzyme is typically incorporated into the second antibody, and its reaction with a color- or fluorescence-producing substrate allows for colorimetric or fluorescent visualization of the antibody—antibody binding. Presence of the visible product indicates that the serum sample contained anti-pathogen antibodies. Serum antibodies can be serially diluted to generate a quantitative result, known as a **titer**, which reflects the level of pathogen infection or exposure. A **sandwich ELISA** is used to detect the presence of the pathogen in a sample. Here, the wells are coated with an anti-pathogen antibody that "captures" the pathogen from the specimen, such as serum. A second enzyme-linked antibody that recognizes another region of the pathogen is added to the plate and visualized as just described. Both tests can be standardized with known quantities of antibody or antigen, respectively, and can be automated with microplate readers that measure the color or fluorescence intensity, quantify the reaction, and compare these quantities with predetermined cut-off values that represent infection. ELISAs have been commonly used for *Plasmodium* detection in mosquitoes (Noedl et al., 2006).

GENOMICS: SEQUENCING, CATALOGING, AND STUDY OF AN ORGANISM'S GENES

DNA carries a living organism's genetic information, located on chromosomes that are tightly compacted within the nucleus of almost all cells. DNA is composed of four different nucleic acids called **bases**, and these four bases, adenine (A), guanine (G), thymine (T) and cytosine (C), are sequentially arranged into genes, some of which ultimately encode proteins. A **genome** (Fig. 28.2) is a complete collection of an organism's DNA encompassing both coding and noncoding regions. A number of methods have been developed to enable **whole genome sequencing**, or cataloging the exact arrangement of bases on chromosomes (Fig. 28.3). Why sequence a gene or a genome? Sequence data can provide insight into an organism's biology by defining the genes that comprise biochemical pathways and cell structures and machinery that are vital for its survival; whole genome sequencing allows us to understand the role an organism's genetic makeup plays in its form and function (Richards, 2015). Genetic data can be used in comparisons within and between phyla to identify the molecular basis for traits that are unique or shared across the tree of life, further defining evolutionary relationships (**phylogenomics**). At a finer scale, genome sequence availability enhances identification of single-base differences (**SNPs**) within and between arthropod populations (**population genomics**).

Genome Projects of Important Arthropods

Genome projects for arthropods of medical and veterinary importance are listed in Table 28.2. The i5K insect genome sequencing initiative (http://i5k.github.io/) aims to sequence the genomes of 5,000 arthropods that are of agricultural and medical importance (i5K Consortium, 2013). Another resource for arthropod genomes is VectorBase (https://www.vectorbase.org/), a bioinformatics resource center funded by the National Institute of Allergy and Infectious Diseases (NIAID) that provides access to genome sequence data of arthropods that vector human pathogens (Giraldo-Calderon et al., 2015). Access to genome sequences of important arthropod species fosters research related to insecticide resistance, blood-feeding, vector competence, and novel targets for control efforts, as well as comparisons across species and even genera. For example, the comparative genome analysis of 16 *Anopheles* mosquito species included representatives from broad geographical locations that occupy varied ecological niches and have different vectorial capacities, providing a means to understand why some species are more competent vectors of human malaria than others (Neafsey et al., 2015). Increasing availability of whole genome sequences from nonmodel organisms has revealed expansions in gene families that appear to be associated with lineage-specific adaptations. For example, the house fly genome has expanded immune-system gene families encoding antimicrobial receptors and effectors, which is likely an adaptation to the septic niches and microbe-rich substrates in which they live and breed (Scott et al., 2014).

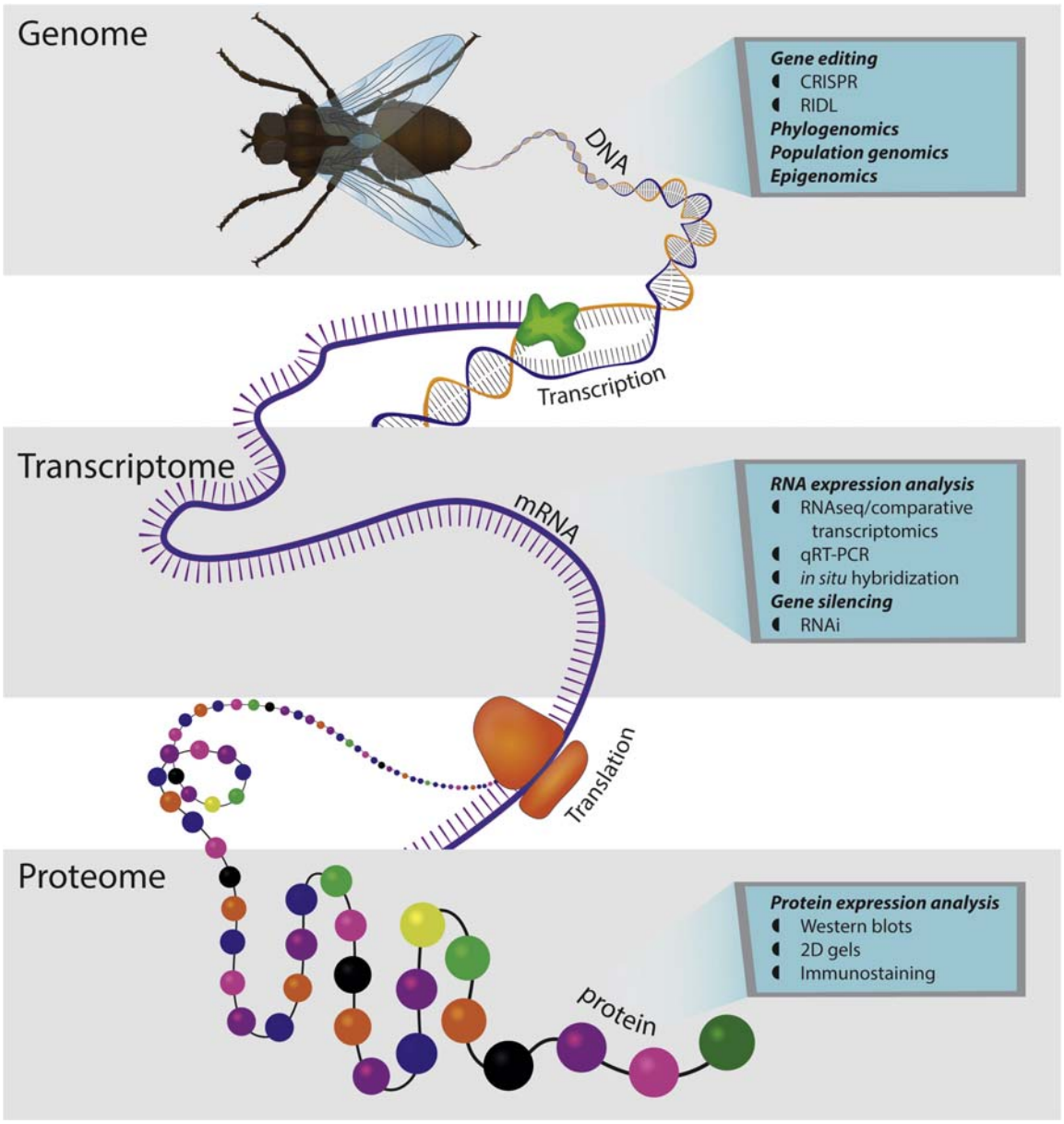


FIGURE 28.2 **From genomes to gene expression.** The canonical pathway of gene expression involves genes (housed in the genome) being transcribed into RNA templates that are translated into proteins, the molecules that underlie an organism's phenotype. Molecular tools facilitate investigation at all stages of these processes. Genomic tools help in understanding gene sequence structure and modifications. With this information, researchers can engineer genomes, via CRISPR and RIDL, to understand gene function or control arthropods and determine genetic relatedness of populations. Broad views of gene expression (transcriptomes) are snapshots of an organism's genetic responses and functions. Experimental tools like qRT-PCR and in situ hybridization allow for finer-scale inference of gene expression, while RNAi is used to silence genes and infer function by reverse genetics. The proteome, a collection of proteins produced by an organism, gives insight into phenotypic results of gene expression. Associated applications, such as Western blots, two-dimensional gels, and immunostaining, are used in laboratory studies of protein expression in organisms or their tissues. *Illustration by Victoria Rhodes.*

Genome Sequencing

Genome size Several factors inform the approach used to sequence an arthropod genome: genome size, availability of inbred lines, quantity of DNA that can be isolated, and funding (Richards and Murali, 2015). A **genome size**, or the number of nucleotide bases in a genome, varies greatly and, like chromosome number, does not necessarily reflect an organism's complexity. A standard method for genome size estimation involves staining cell nuclei of the organism with propidium iodide, which is a sequence-independent, fluorescent dye that stains nucleic acids (DNA and RNA). RNAs are removed from the sample, and intensity of the fluorescent-stained DNA is measured on a flow cytometer. The genome size of the sample is measured by comparing

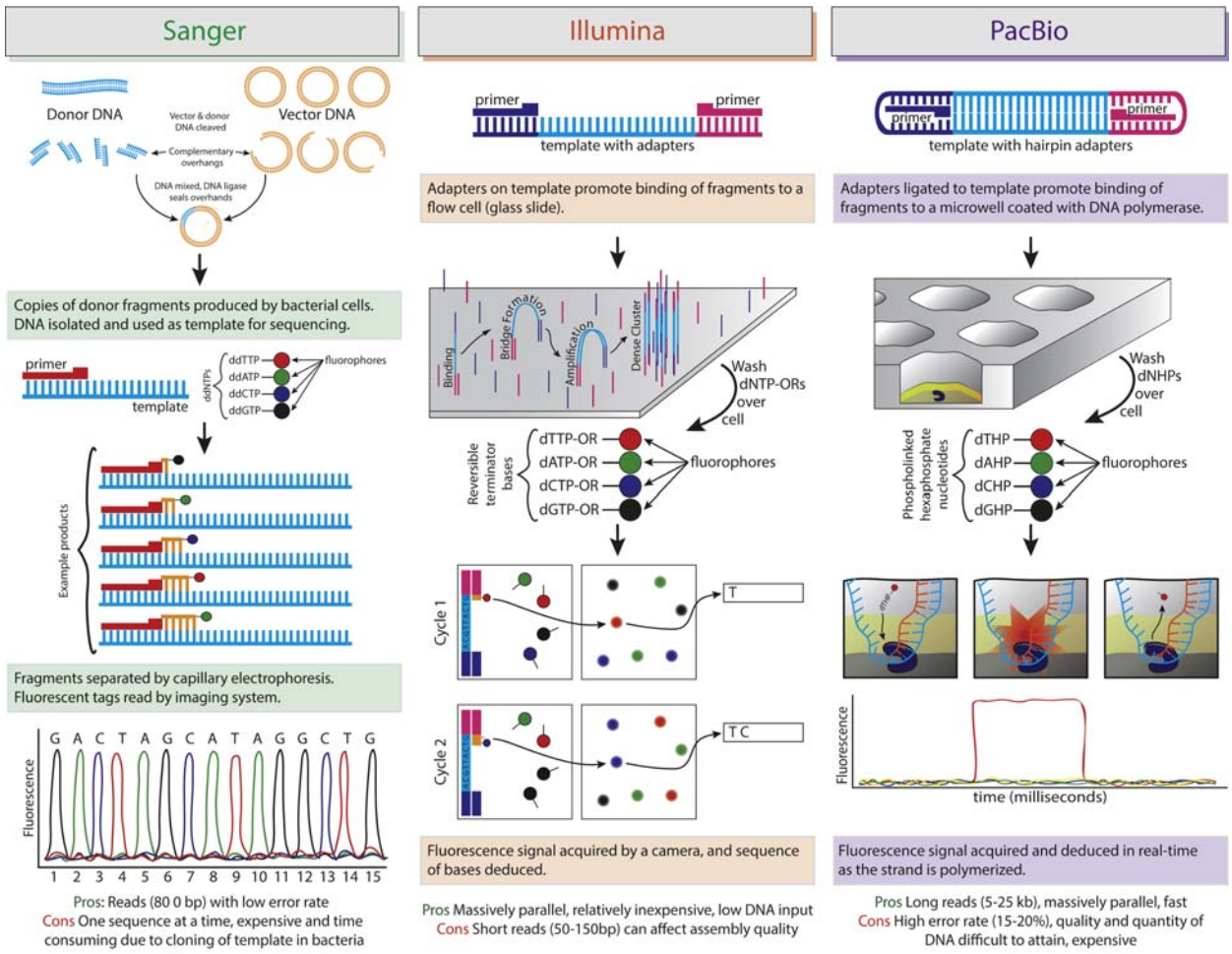


FIGURE 28.3 Evolution of sequencing platforms. In *Sanger sequencing*, DNA is fragmented into pieces and copies of these fragments are produced in bacteria. DNA polymerase incorporates fluorophore-labeled nucleotides, and the resulting products are electrophoretically separated. The order of bases is resolved by capturing the sequence of fluorophores on laser excitation. The *Illumina platform* employs a sequencing strategy in which millions of DNA fragments (50−150 bp) are sequenced in parallel. A flow cell, in this case a glass slide, is coated with oligonucleotides that are complementary to those on the library adapters promoting binding of the fragments across the flow cell surface. The fragments are clonally amplified by bridge PCR, producing thousands of copies of each fragment that are clustered across the flow cell. These clusters serve as templates for sequencing in the presence of a polymerase and fluorophore-labeled reversible terminator bases (dNTP-OR). Polymerization occurs across the length of the flow cell. Base fluorescence is measured by a coupled-charge device (CCD) camera, and arrangement of bases is interpreted from patterns of fluorescence. The *PacBio platform* sequencing libraries contain long DNA fragments (5−25 kb) with hairpin adapters at each end. The flow cell is composed of >300 reaction chambers, each containing a single DNA polymerase immobilized at its base. Adapters used in library preparation enable docking of a single DNA molecule in the chamber without the need for clonal amplification. Polymerization of the DNA molecule occurs in the presence of fluorophore-labeled phospholinked hexaphosphate bases (dNHP), and data are captured in real time as a series of light pulses. Sequence acquisition occurs rapidly, as DNA is polymerized at a rate of 10 bases per second. *Illustration by Victoria Rhodes.*

the intensity to the relative fluorescence of a standard sample of known genome size. Determining genome size is a first step in commencing a sequencing project, as larger genome sizes require more extensive sequencing, thus affecting overall cost of the project. Genome sizes of arthropods vary greatly, as shown in Table 28.2.

Genome source material Genetic variation within an arthropod population, whether field collected or laboratory colonized, is reflected in the genome as regions of **DNA polymorphism**, or alternate DNA sequences (alleles), that differ between individuals. Randomly pooling individuals from a population to use in a sequencing effort increases the prevalence of these polymorphisms, which causes difficulties in downstream genome assembly algorithms. Variation is reduced by either sequencing a single individual or producing an **inbred line,** a strain resulting from 10 or more successive generations of full-sibling paired matings. Hurdles related to production of inbred lines include the complexities of colonizing arthropods such as length of life cycle, refractoriness to colonization, and the potential for negative effects of inbreeding. In the absence of inbred lines, the size of the arthropod can affect the

TABLE 28.2 Complete or Nearly Complete Genome Projects for Arthropods of Medical and Veterinary Importance

Organism	Common Name	Genome Size (Mb)	Significance
Chelicerata			
Ixodes scapularis	Black-legged tick	2,262	Vector, Lyme disease, anaplasmosis, Powassan virus, babesiosis
Ixodes ricinus	Castor bean tick	2,100	Vector, Lyme disease, anaplasmosis, babesiosis in Europe
Rhipicephalus microplus	Southern cattle tick	7,335	Vector, bovine babesiosis, anaplasmosis
Sarcoptes scabiei (human, pig varieties)	Scabies mite	56	Causes scabies in humans, mange in other animal species
Dermatophagoides farinae	American house-dust mite	53.5	Common cause of human allergy
Blattodea: Blattellidae			
Blatella germanica	German cockroach	1,960	Urban pest, mechanical vector
Diptera: Calliphoridae			
Calliphora vicina	European blow fly	748	Forensic importance
Cochliomyia hominivorax	New World screwworm	443	Myiasis
Lucilia cuprina	Australian sheep blow fly	458	Sheep strike, food safety
Lucilia sericata	Common green bottle fly	679	Forensic entomology, food safety
Phormia regina	North American blow fly	523	Forensic entomology, food safety
Diptera: Ceratopogonidae			
Culicoides sonorensis	Biting midge	200	Vector, Bluetongue, African horse sickness, epizootic hemorrhagic disease, Schmallenberg viruses
Diptera: Culicidae			
Aedes albopictus	Asian tiger mosquito	606–1,623	Vector, dengue, Chikungunya, LaCrosse viruses
Aedes aegypti	Yellow fever mosquito	1,380	Vector, dengue, Yellow fever, Zika viruses
Anopheles gambiae[a]	African malaria mosquito	278	Vector, African malaria
Culex quinquefasciatus	Southern house mosquito	540	Vector, West Nile virus

Continued

TABLE 28.2 Complete or Nearly Complete Genome Projects for Arthropods of Medical and Veterinary Importance—cont'd

Organism	Common Name	Genome Size (Mb)	Significance
Diptera: Drosophilidae			
Phortica variegata	Zoophilic fruit fly	183	Intermediate host of *Thelazia callipaeda* (eye worm) in Europe
Diptera: Hippoboscidae			
Glossina morsitans morsitans[b]	Tsetse fly	366	Vector, African sleeping sickness, Nagana (*Trypanosoma brucei* spp.)
Diptera: Muscidae			
Musca domestica	House fly	998	Filth fly, food safety
Stomoxys calcitrans	Stable fly	1,133	Human and animal pest/parasite
Haematobia irritans	Horn fly	1,186	Cattle parasite
Diptera: Psychodidae			
Phlebotomus papatasi	Old World sand fly	170	Vector, leishmaniasis
Lutzomyia longipalpis	New World sand fly	300	Vector, leishmaniasis
Diptera: Sarcophagidae			
Neobellieria bullata	Grey flesh fly	396	Forensic entomology
Hemiptera: Cimicidae			
Cimex lectularius	Common bed bug	864	Human parasite, urban pest
Hemiptera: Reduviidae			
Rhodnius prolixus	Kissing bug	733	Vector, Chagas disease
Phthiraptera: Pediculidae			
Pediculus humanus humanus	Human body louse	106	Vector, typhus fever (*Rickettsia prowazekii*)

[a] *Genome sequencing of 16 additional* Anopheles *sp. has been completed, and 8 more species are near completion (Neafsey et al., 2015; www.vectorbase.org).*
[b] *Five additional* Glossina *genomes have been sequenced, representing species from the Fusca, Palpalis, and Morsitans subgroups (www.vectorbase.org).*

quantity of DNA available for the genome sequencing effort; however, DNA isolation approaches have improved substantially over the past several decades, and the availability of commercial kits enable scientists to successfully isolate high-molecular-weight, high-purity DNA from a small amount of biological material.

Sequencing methods A **whole-genome shotgun sequencing** method is routinely used in current genome sequencing efforts, as computer algorithms are available that allow *de novo* assembly of a genome without the need for physical genetic maps. The method involves fragmenting chromosomal DNA into manageable lengths, assembling these into DNA libraries representing the various fragment sizes, sequencing the fragments, and assembling or reconstructing the sequencing reads into contiguous stretches of the genome. The evolution of sequencing technologies is depicted in Fig. 28.3. In traditional **Sanger sequencing**, DNA synthesis is initiated from a primer and a mixture of nucleotides (dNTPs) and modified nucleotides (fluorophore-labeled **dideoxynucleotides [ddNTPs]**, which are missing a hydroxyl group) are used to extend the growing complementary strand. DNA synthesis is halted when a ddNTP is incorporated, producing an array of fragments that incrementally differ in length and terminate in a single ddNTP base. The fragments are resolved by size on an instrument that is equipped with a laser (to excite the fluorophore), and a computer acquires the fluorescent signal and translates these into the respective nucleotide.

Currently, **high-throughput sequencing** platforms have revolutionized access to whole-genome sequencing, expanding capabilities for nonmodel arthropods (van Dijk et al., 2014; Reuter et al., 2015). In general, the process involves DNA isolation, DNA fragmentation into shorter lengths, attachment of sequencer platform-specific adapters, and delivery to the platform. At the time of this publication, two popular high-throughput sequencing platforms are those from Illumina Inc. (HiSeq, MiSeq) and Pacific Biosciences (PacBio RSII) (Fig. 28.3). Each platform alone has its benefits, and current trends are leaning toward using them in combination to harness the advantages of each. For example, whole genomes are sequenced using PacBio, and data from the Illumina platform are incorporated to improve downstream assembly and correct for inherent sequencing errors. An up-and-coming sequencing platform from Oxford Nanopore Technologies (MinION) is also beginning to make traction in whole-genome sequencing. The nanopore instrument itself is quite small, enabling use in the field, and the low cost for sample preparation has made it more accessible outside of the research community (reviewed in Jain et al., 2016). Cell-free preparation and amplification of DNA sequencing libraries, and the implementation of massively parallel sequencing by synthesis (SBS) methods, have decreased the cost of whole-genome sequencing substantially, resulting in a marked rise in the number of available sequenced arthropods of medical and veterinary importance (Table 28.2).

Library preparation for high-throughput **sequencing platforms** involves five steps: (1) isolation of high-molecular-weight DNA, (2) sample DNA fragmentation, (3) short oligonucleotide adapter ligation, (4) fragment size selection, and (5) library quantitation. High-molecular-weight DNA is fragmented into pools of different lengths that are used in high-throughput sequencing platforms. Examples of physical fragmentation (shearing) methods include high-frequency acoustic shearing, low-frequency sonication, dispersed acoustic energy, and high-pressure nebulization that uses compressed air to force DNA through a small opening. Platform-specific oligonucleotide adapters are ligated to the 5′ and 3′ ends of the fragments. Depending on the sequencer platform, the adapters may contain primer sequences for library enrichment and polymerase binding sites, as well as sequences that promote binding of the fragments to a solid surface where DNA sequencing will occur. Additional, unique sequence tags can be included to enable multiple samples to be combined, deciphered, and demultiplexed in downstream data processing steps. The adapter-linked products are size selected by agarose gel excision or immobilized magnetic beads to ensure optimum fragment lengths for the different sequencer platforms, and the resulting library is subjected to quality-control metrics to ensure only high-quality preparations are used. The library preparation is delivered to a flow cell solid matrix, which can take the form of a glass slide, surface of a magnetic bead, or nanochamber, depending on the sequencing platform.

Bioinformatics Currently, a major bottleneck in genome sequencing is the processing and analysis of sequence data, which is the focus of scientists in the field of **bioinformatics**. Bioinformatics involves using computer algorithms and statistical functions to collect, catalog, and analyze DNA and protein sequences. In general, quality-control criteria are applied to whole-genome sequence data, ensuring only high-quality reads are incorporated into downstream analyses. Algorithms for assembly of reads into contiguous sequences representing the genome are used, and these differ based on whether short-read (Illumina; e.g., ALL-PATHS, DISCOVAR) or long-read (PacBio; e.g., HGAP, Falcon, MECAT) data are generated. Researchers publish newly reported sequences in databases, such as GenBank at the National Institutes of Health, in Bethesda, Maryland (USA), and the European Molecular Biology Laboratory (EMBL) Sequence Database at the European Bioinformatics Institute in the United Kingdom. These are freely available to the public via the Internet (www.ncbi.nlm.nih.gov, www.ebi.ac.uk/Databases). After assembly of the whole-genome sequence data, regions of coding sequence within the genome are predicted

(**annotated**) by a combined application of (1) aligning known coding sequence information to the genome assembly to aid in identification of intron/exon junctions and (2) using algorithms that are trained (using known data sets) to identify the hallmarks of a gene/gene model (e.g., Gnomon, Augustus, or all algorithms combined into a single pipeline, as in MAKER).

For functional annotation, protein-coding regions of genes are translated and compared with protein databases, which also are publicly available. Alignment programs (e.g., BLAST at NCBI) have wide-ranging capabilities such as determining putative identities of sequences of interest by aligning with other sequences in the database. Once a predicted gene set is available, a community of interested researchers manually curates these *in silico* predictions to verify or edit models within gene families of interest using evidence from literature or from experiments. The massive quantity of data generated by genome sequencing efforts has created data storage issues for which a perfect solution has yet to be identified (Stephens et al., 2015). Further, computational power required for these "big data" analyses can be cost prohibitive, and the availability of cloud-based computing networks have helped in easing this burden (Muir et al., 2016).

ANALYZING GENE EXPRESSION

While genes constitute the genetic makeup and phenotypic potential of an organism, **gene expression** contributes to an arthropod's observed phenotype. Gene expression begins with synthesis of an RNA transcript (a process called **transcription**; Fig. 28.2) from a DNA template (gene). For some genes, the information in these RNA transcripts is decoded during **translation** to generate peptide sequences that compose proteins. Therefore, gene expression is measured by analyzing products of transcription (such as messenger RNA [mRNA]) and translation (protein). Gene expression analysis is a step beyond genomic sequencing that allows researchers to explore the biological function of genes, a type of "functional genomics."

RNA Analysis

Some of the techniques described next involve the molecular detection of mRNA in an organism and use many of the same methods mentioned in the previous section. The presence of mRNA associated with particular genes indicates that the gene has been transcriptionally activated or conditionally "turned on" in the organism. Synthesis of mRNAs under certain conditions gives insight into the roles that the mRNA's encoded proteins play in the organism's biology.

Transcriptomics: a broad view of gene expression
Catalogs of mRNAs from an organism that are sequenced, categorized (annotated), and counted collectively constitute a **transcriptome**. Transcriptomes are constructed by first extracting total RNA from the biological specimen, enriching for mRNA, synthesizing a complementary DNA (cDNA) copy of the mRNA by **reverse transcription**, and sequencing short pieces ("reads") from these cDNAs using high-throughput sequencing methods called RNA-Seq, which incorporate sequencing platforms like Illumina (discussed earlier). Short reads are aligned and assembled to reconstruct full mRNA transcripts, either with the guidance of an organism's genome or via *de novo* assembly in the absence of an available genome. As with genomics, the assembled transcripts are annotated and assigned a putative function based on their protein-coding regions. Both assembly and annotation are facilitated by comparing the mRNA sequences to similar (orthologous) annotated sequences in a database, like GenBank. Because the transcriptome essentially captures a snapshot of gene expression under a certain condition, its information can be used on its own to elucidate the role of genes or gene networks associated with that condition. For example, one may be interested in capturing and comparing the transcriptional landscape of adult female mosquitoes that have fed on animals to those fed on sugar solution, in order to identify genes that might be involved specifically in blood digestion.

Transcriptome snapshots are compared across different conditions in a process called **comparative transcriptomics**, where read counts representing differences in expression of the same population of genes (actually, transcripts) are analyzed by using bioinformatics software and visualized a variety of ways. For example, Fig. 28.4 shows a scatter plot of transcripts that are differentially expressed across two conditions: unfed female biting midges and female biting midges fed blood containing orbivirus. Each "dot" represents a differentially expressed transcript, with those to the left of zero being downregulated in orbivirus-infected midges and those to the right being upregulated in orbivirus-infected midges, both relative to unfed midges. Also shown is the color-coded fold change in expression across the two conditions (horizontal axis) and the statistical significance of that change (vertical axis). Examples of comparative transcriptomics include studying gene expression across life stages (e.g., larvae, pupae, adults), among different tissues (e.g., gut, salivary gland, antenna), or in response to different stimuli (e.g., attractants, hosts, pesticides, temperature) and biological state (fed vs. fasted, male vs. female, infected vs. uninfected). For further reading on the broad applications of transcriptomics in arthropod studies, see references listed at the end of this chapter by Valenzuela et al. (2003), Campbell et al. (2005), Anderson et al. (2006), Koutsos et al. (2007), Wang et al. (2009), Price et al. (2011), Li et al. (2013b), Rinker et al. (2013) and Nayduch et al. (2014a,b).

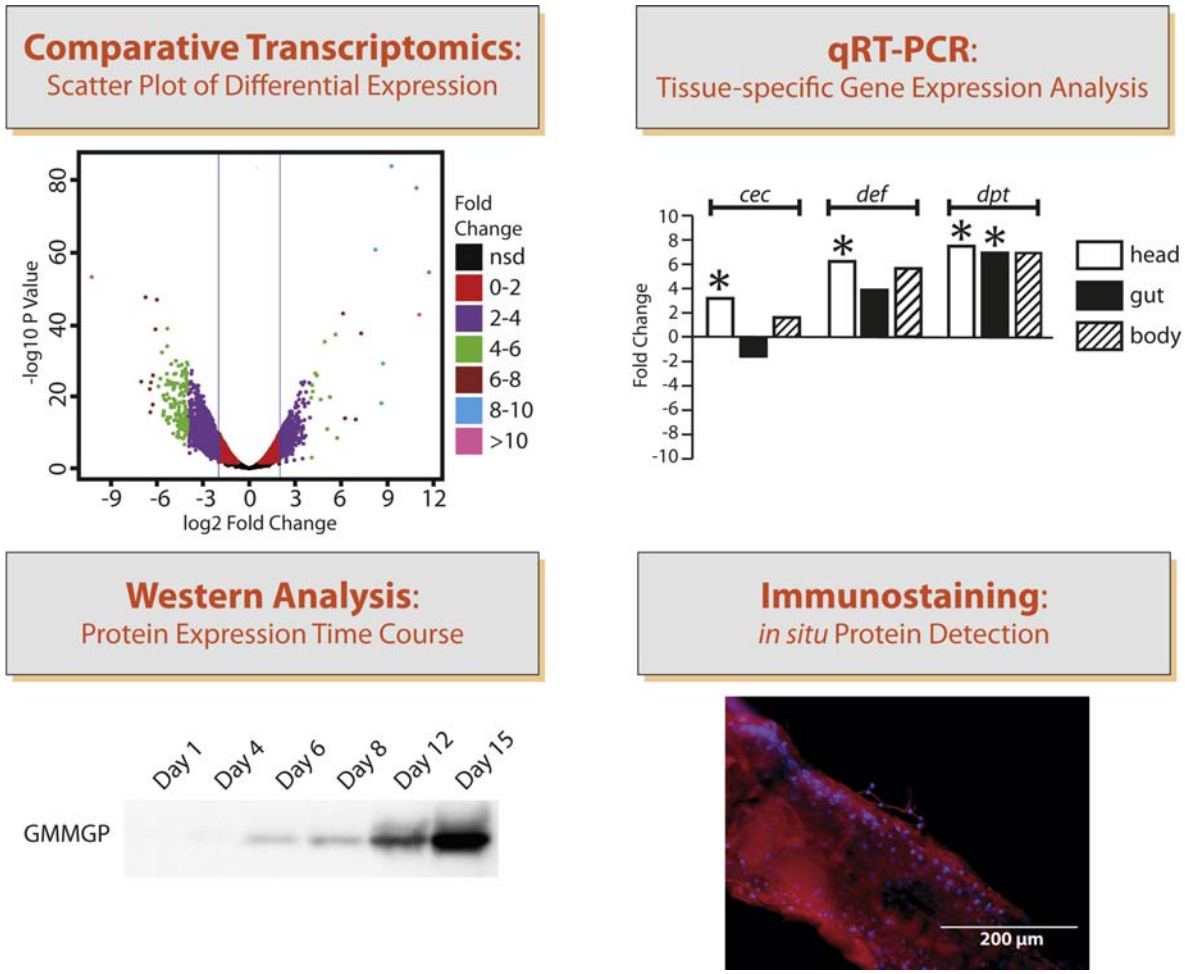


FIGURE 28.4 **Methods for analyzing gene expression in arthropods.** Top left: *Comparative transcriptomics* scatter plot of gene expression across two conditions in biting midges (*Culicoides sonorensis*, see text for details). Each dot represents a differentially expressed gene, color coded to show fold changes in expression. Top right: *qRT-PCR* analysis of antimicrobial peptide expression (fold change) across body tissues from adult house flies (*M. domestica*) that were fed bacteria. *Gene expression was significantly different from broth-fed control flies. Bottom left: *Western blot* of tsetse fly (*G. m. morsitans*) milk protein (GMMGP) expression in days after eclosion (figure credit: G. Attardo, Yale University). Bottom right: *Immunofluorescent staining* of tissues to detect lysozyme protein expression (red) in the midgut of a bacteria-fed house fly. Blue = DAPI-stained cell nuclei. *Illustrations by Dana Nayduch and Victoria Rhodes.*

qRT-PCR As described earlier, RT is the process by which cDNA is made from an mRNA template. qRT-PCR is a process used to quantify the abundance of cDNA across different conditions relative to a reference gene and a calibrator (control) condition. Reference genes are genes whose transcript abundance tends to stay constant across the conditions being tested and serve as normalization tools to facilitate comparisons of genes of interest (target genes) across the treatment(s) and calibrator (control). To perform qRT-PCR, RNA is extracted from specimens in the experimental and calibrator groups. cDNA is generated from this RNA, and gene-specific primers designed to one or several target and reference genes are used in subsequent PCR reactions. PCR products can be visualized with electrophoretic separation on an agarose gel, where intensity of the band is correlated with the amount of product and, therefore, the amount of starting template. In modern qRT-PCR, the PCR amplicons are measured in real time by using intercalating dyes like SYBR Green or internal probes and qPCR cyclers, as discussed earlier in this chapter. Subsequent algorithms or programs are used to quantify gene expression by comparing the relative expression of the targets to the reference gene across the experimental and calibrator samples. Fig. 28.4 shows the expression of three antimicrobial peptide genes (cecropin, *cec*; defensin, *def*; diptericin, *dpt*) across three tissue samples from house flies (head, gut, body) that had been fed either bacteria (experimental) or sterile broth (calibrator). The expression of these genes was quantified by SYBR Green qRT-PCR, and expression values were compared by using the reference gene *rps18*, a ribosomal protein gene whose expression is similar across house fly tissues, to

generate fold changes in expression between calibrator and experimental flies. The asterisks indicate genes whose expression was significantly different compared with controls, calculated by using the methods of Pfaffl (2002), similar to the methods of Joyner et al. (2013).

In situ hybridization is a technique applied to determine the spatial expression of genes in an organism. This technique allows researchers to observe the cellular localization of mRNA transcripts in a tissue section. After experimental treatment, the organism or its tissues are harvested, fixed, and embedded in a matrix (such as wax) to facilitate sectioning. Then, very thin sections are sliced and mounted on a slide. Short DNA or RNA oligonucleotide probes with sequences complementary to the target transcript of interest are added and hybridize, by base-pairing rules, to the intracellular mRNA transcripts in the tissue section. The probes incorporate a colorimetric or radioactive label to facilitate detection by the user. The researcher can then compare gene expression within or between tissue samples as they appeared in that organism during the experimental treatment. In situ hybridization also is used to detect nucleic acid of pathogens, such as viruses, in the tissues of arthropod vectors (Drolet et al., 2005).

Protein Analysis

Gene expression can vary posttranscriptionally, where the mRNA level detected by the techniques described here does not reflect the amount of protein produced by the organism on a translational level. Consequently, researchers sometimes use additional techniques for detecting gene expression at the protein level. Many of these techniques use labeled antibodies generated to specifically detect proteins of interest, just as DNA or RNA primers and probes are used to detect nucleic acids.

Proteomics: a broad view of protein expression A proteome is a set of proteins produced by an organism or its tissues. Like the transcriptome, described earlier, the proteome is a snapshot of proteins being expressed in that specimen at the time of collection. Therefore, the proteome varies across tissues, treatments, and conditions such as time. Proteomes are generated by collecting the total protein from a specimen and fractionating them with electrophoresis. The fractions are enzymatically digested to produce shorter peptides, which then are separated by the use of liquid chromatography or ion exchange. Categorized fractions are then ionized via various techniques and subject to mass spectrometry either alone or combined with subsequent sequencing techniques (e.g., Edman degradation of N-terminus amino acids). For annotation, resulting sequences are compared with protein databases such as SWISS-PROT, TrEMBL, and PIR-PSD (or UniProt, which consolidates all the protein databases for easy queries; www.uniprot.org/). For further reading on the methods used to generate proteomes, see the review by Steen and Mann (2004). Proteomics has been used to characterize saliva of blood-feeding flies (e.g., Ribeiro and Francischetti, 2003; Valenzuela et al., 2004; Wang et al., 2009; Ribeiro et al., 2010; King et al., 2011; Lehiy and Drolet, 2014) and ticks (e.g., Madden et al., 2004; Valenzuela, 2004) to elucidate proteins involved in hematophagy and pathogen transmission.

Western analysis Western analysis (of which, Western blotting, is part) is used to detect and quantify protein expression across samples. Total proteins are extracted from a specimen, denatured with a detergent (e.g., sodium dodecyl sulfate [SDS]), and size-separated through the use of polyacrylamide gel electrophoresis (SDS-PAGE). Size-fractionated proteins are visualized by staining the gel with a dye (e.g., Coomassie blue) and quantified. The separated proteins are transferred ("blotted") to a nitrocellulose or polyvinylidene difluoride (PVDF) membrane. Blots are exposed to a solution containing specific polyclonal or monoclonal antibodies that have been labeled with detectable markers and designed to bind to the protein of interest (**Western blot**). Molecular-weight ladders also are run on the gel, ensuring that the correct size can be confirmed if antibodies bind to the protein of interest. Western blotting and proteomics have been used to identify important salivary-gland proteins used by blood-feeding arthropods such as ticks, biting midges, sand flies, and mosquitoes. Fig. 28.4 shows a Western blot of milk protein expression in female tsetse flies (*Glossina morsitans morsitans*) over time (1−15 days) after eclosion. For further reading on hematophagous arthropod sialomes (salivary gland proteomes), see the review by Ribeiro and Francischetti (2003).

Two-dimensional gels (2D gels) Two-dimensional gels first separate proteins by **isoelectric focusing** (by charge in a pH gradient) and then, at right angles to the first separation, by their mass. As a result, the proteins migrate across the gel in two dimensions by their isoelectric point (pI) and size. Each "spot" on a stained gel likely represents a unique protein that can be mapped with x and y coordinates (i.e., mass and pI). Greater resolution is achieved compared with SDS-PAGE alone, because proteins will be distinguishable by the second parameter (pI). Two-dimensional gels can be compared across different samples to gain information on broad differences in protein expression. Spots present on one specimen's gel may not be present on another or may be present but with higher or lower intensity, representing differentially-expressed proteins. Computer software can be used to scan gels and overlay gel images and detect these differences for comparative analyses.

Immunostaining Similar to in situ hybridization methods, used to detect RNA expression in cells, immunostaining techniques like immunofluorescence and

immunohistochemistry facilitate in situ observation and localization of protein expression within tissues or cells. Tissues are either frozen or embedded in a matrix, then cut into thin sections and mounted on a slide. The tissue section is exposed to a labeled antibody that binds to the protein of interest. If the tissues are very small and thin, they can be fixed and permeabilized without subsequent sectioning. In immunofluorescence, the antibody is conjugated to a fluorescent marker, and special microscopes are used to excite the marker so it emits fluorescence that is visualized in the magnified tissue. In immunohistochemistry, the antibody is coupled to an enzyme that converts a colorless substrate into a visible reaction product. In either case, the presence of the visible marker indicates the cytolocalization of the protein of interest and allows a comparison of both location and intensity of these markers across tissues or cells within tissues. Fig. 28.4 shows immunofluorescent detection of the antimicrobial enzyme lysozyme in the midgut of house flies fed bacteria. In this example, the anti-lysozyme antibody was not labeled, so a second antibody, generated against the primary anti-lysozyme antibody, was labeled with a red fluorescent dye. All cells in the tissue that appear bright red were expressing the lysozyme protein. Nuclei are stained with DAPI blue that binds and stains DNA and helps the user visualize cells in the specimen.

MOLECULAR TOOLS TO MANIPULATE ARTHROPODS

After genes of interest are identified by the methods discussed in this chapter, researchers typically proceed to assess gene function in biological processes by altering these genes or their products in organisms and observing the phenotypic result (**reverse genetics**).

These genetically engineered or genetically modified organisms (GMOs) are generated by "knock out" (deletion of nucleotides or entire genes) or "knock in" (addition of nucleotides, including entire genes) methods. Both methods either can be directed toward a certain region of the genome (e.g., gene-specific insertions and deletions) or random (e.g., inserting a transgene anywhere in the genome and selecting for recombinant mutants). Other methods involve knocking out or knocking down mRNA, instead of genes. Boxes 28.1–28.3 describe some approaches to genetic manipulation of arthropods.

Editing Genomes With CRISPR-Cas9

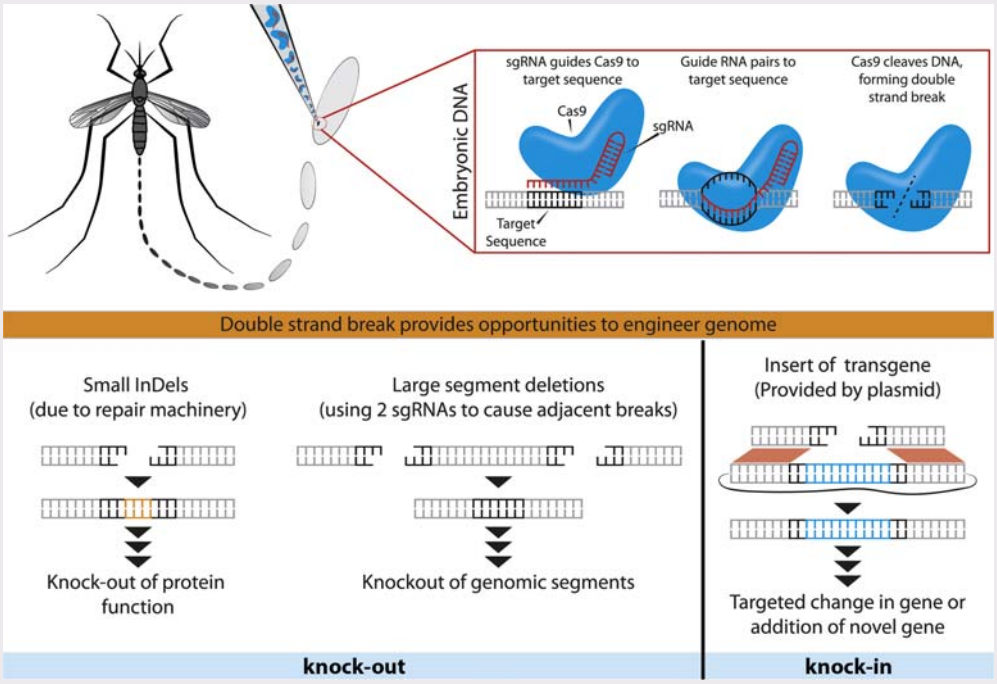
Box 28.1 explains a gene-editing technique called CRISPR-Cas9 that is used to modify an organism's genes at the genome level, as reviewed by Barrangou and Doudna (2016). **CRISPR** (clustered regularly interspersed short palindromic repeats) are present in bacterial genomes and, in combination with CRISPR-associated (Cas) proteins, are part of the bacterial immune response to viral infection (Sontheimer and Barrangou, 2015). On viral infection, bacteria acquire segments of viral DNAs, whose signatures enable recognition by bacteria on subsequent viral infection (a rudimentary type of immunological memory). When bacteria later recognize these same signature sequences, Cas proteins are recruited to the site and cleave the viral DNA via nuclease activity to destroy the virus. Components of the CRISPR-Cas system have been simplified and commercialized to provide researchers with a tool to modify genomes across a variety of taxa, from cattle to plants to insects. The CRISPR-Cas9 system, used most often for genome editing research at the time this chapter was written, uses genome-specific guide RNA sequences to direct the Cas9 nuclease activity (Doudna and Charpentier, 2014; Wang et al., 2016). This method (Box 28.1) provides a means to delete target regions of the genome (knock-out) or insert sequences of interest (knock-in). Recent modifications to Cas proteins have further provided a means to activate or repress expression of genes to study the impacts of up-/down-regulation within pathways, and the incorporation of fluorophores to Cas proteins has enabled real-time, sequence-specific visualization of cellular processes (gene imaging) (Barrangou and Doudna, 2016).

CRISPR has been used to engineer the *Ae. aegypti* genome for genetic studies (Kistler et al., 2015) and for functionally characterizing elements of mosquito sex determination (Hall et al., 2015) and reproduction (Zhang et al., 2016). The technique is being explored as a method for insect population suppression (Alphey, 2016a). It shows promise as a tool for genome engineering and functional characterization in newly sequenced genomes from nonmodel organisms. These efforts have been otherwise problematic due to the variable success of gene silencing (see RNAi later) in flies.

Insect Population Suppression

The sterile insect technique (SIT) is a control strategy that uses radiation to produce genetic mutations or chromosomal breaks to generate sterile adult insects. These sterile insects are released into the wild to suppress and eventually eradicate wild pest populations (reviewed in Reichard, 2002). An SIT program was successfully used in 1957 by releasing sterile adult female and male New World screwworm flies, *Cochliomya homnivorax*, eradicating this important pest from the United States by 1966 (reviewed in Mastrangelo and Welch, 2012). Unfortunately, SIT has been less successful in controlling populations of mosquito vectors of arboviruses (e.g., dengue, Chikungunya virus, Zika, and West Nile viruses) and protozoa (e.g., malaria) due, in part, to reduced performance of irradiated mosquitoes (Dame et al., 2009).

BOX 28.1 Manipulating Arthropods: CRISPR-Cas9 Genome Editing System



Genome-specific guide RNA sequences (sgRNA) hybridize to complementary regions within the target genome and direct the CRISPR-associated 9 (Cas9) nuclease activity to the region of interest to create double-stranded breaks in the genomic DNA. Use of single guide RNAs can create small insertion/deletions resulting from inherent DNA repair machinery (InDels), while use of several guide RNAs can direct deletion of large genome segments, both approaches of which result in gene knock-out. Alternatively, a segment of the genome can be targeted for alteration by providing a sequence that can be inserted into the cleaved region, resulting in a gene knock-in. *Illustration by Victoria Rhodes.*

The release of insects carrying dominant lethals (**RIDL**) is an alternative method for producing sterile insect lines and is based on genetically modifying insects to harbor a repressible lethal gene (Heinrich and Scott, 2000; Thomas et al., 2000). A comparison between SIT and RIDL is depicted in Box 28.2. Elements of the prokaryotic tetracycline gene expression system are used in RIDL genetic constructs. The system harnesses the ability to upregulate a transactivator protein, the production of which is turned "off" in the presence of tetracycline but proceeds in its absence (system is "on"). Uncontrolled expression of the transactivator protein results in organismal lethality from "transcriptional squelching," in which overproduction of the transactivator results in global suppression of gene expression in the organism. The precise mechanism is currently unclear. Genetic constructs are delivered via the microinjection of insect embryos, and they are integrated into the germ line using a transposable element vector transformation system.

Alphey and colleagues produced RIDL Mediterranean fruit flies, *Ceratitis capitata* (Gong et al., 2005), and later RIDL *Aedes aegypti* mosquitoes (Fu et al., 2010) and RIDL *Aedes albopictus* mosquitoes (Labbe et al., 2012). The technology was used by Oxitec Ltd (England) to produce a line of genetically modified *Ae. aegypti* mosquitos for use in suppressing wild populations. Male pupae are separated from females, and Friendly™ (transgenic) adult male mosquitoes are released to mate with females in the wild, passing on the transgene to their progeny; the absence of tetracycline in the wild affects the lethality phenotype. Oxitec conducted field studies in the Cayman Islands (Harris et al., 2012) and Brazil [municipality of Itaberaba] (Carvalho et al., 2015), reporting an 80%–95% reduction of *Ae. aegypti* in the treatment areas. Further, Oxitec reported a 90% reduction in dengue cases for one treatment area in Brazil (Piracicaba) compared with 50% reduction where sanitary methods alone were used (e.g., eliminating standing water) (Oxitec, 2016a). In the United States, the Florida Keys Mosquito Control District of Florida voted in favor of partnering with Oxitec to complete a field trial of the genetically modified mosquitoes to reduce *Ae. aegypti* populations at an as-yet-unidentified test site (Oxitec, 2016b).

RIDL genetic constructs have been designed using life stage-specific promoters and/or sex-specific genetic signatures. These tools can be exploited for development-targeted or female-specific lethality; for example, the latter being the basis for production of male-only insect lines.

Such an approach was used to develop male-only lines of *Lucilia cuprina* (Scott, 2014) and *C. hominivorax* (Concha et al., 2016) by Max Scott's laboratory. Field studies were recently approved to assess these genetically modified New World screwworm lines in Panama. If successful, mass production of male-only lines would save the screwworm eradication program millions of dollars by eliminating the need to rear the flies to adults for the sterile insect release program (Concha et al. 2016; Alphey, 2016b).

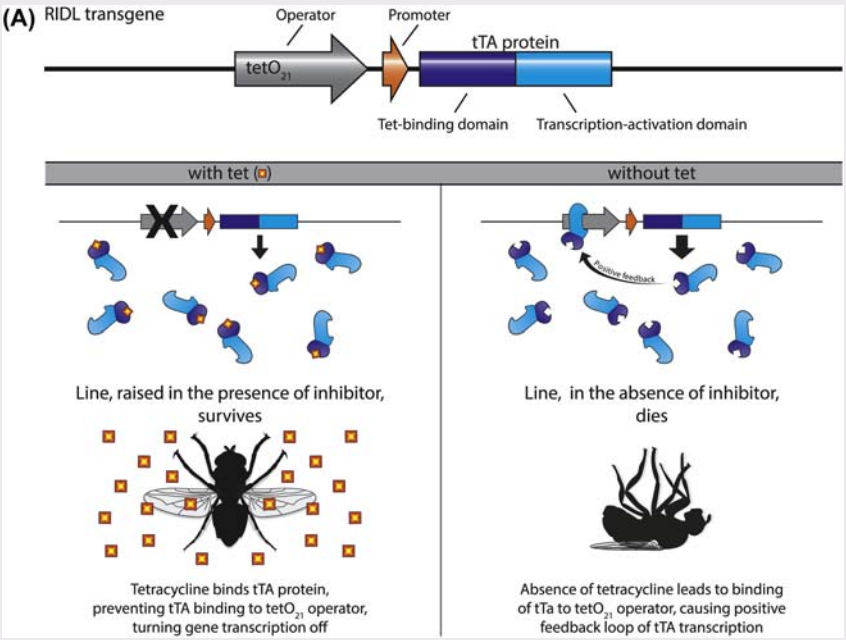
Posttranscriptional Gene Silencing With RNAi

While both CRISPR and RIDL involve altering an organism's genome, RNAi (Box 28.3) is a mechanism used to modify the phenotype posttranscriptionally, at the mRNA level. RNAi is an innate antiviral mechanism present in many metazoan organisms that has been coopted as a laboratory tool to silence genes by destroying mRNA and inhibiting translation (Fire, 1999). This technique does not require the generation of GMOs and can be induced to study transient effects of gene function. RNAi has been used in large-scale screens to assess the role of genes and biological pathways in cellular or organismal function such as the insect immune response, feeding processes, olfaction, reproduction, and vector–parasite interactions (Belles, 2010; Scott et al., 2013). For example, RNAi silencing of genes expressed in the salivary gland has revealed proteins involved in blood-feeding (Chalaire et al., 2011). The technique also has been used to elucidate the components of vector competence in dipteran vectors (Nayduch and Aksoy, 2007; Barnard et al., 2012; Mills et al., 2015). The utility of RNAi via feeding dsRNA or siRNA is being explored as a novel pesticide in ants and other important arthropod pests (Baum and Roberts, 2014).

CONCLUSIONS AND FUTURE DIRECTIONS

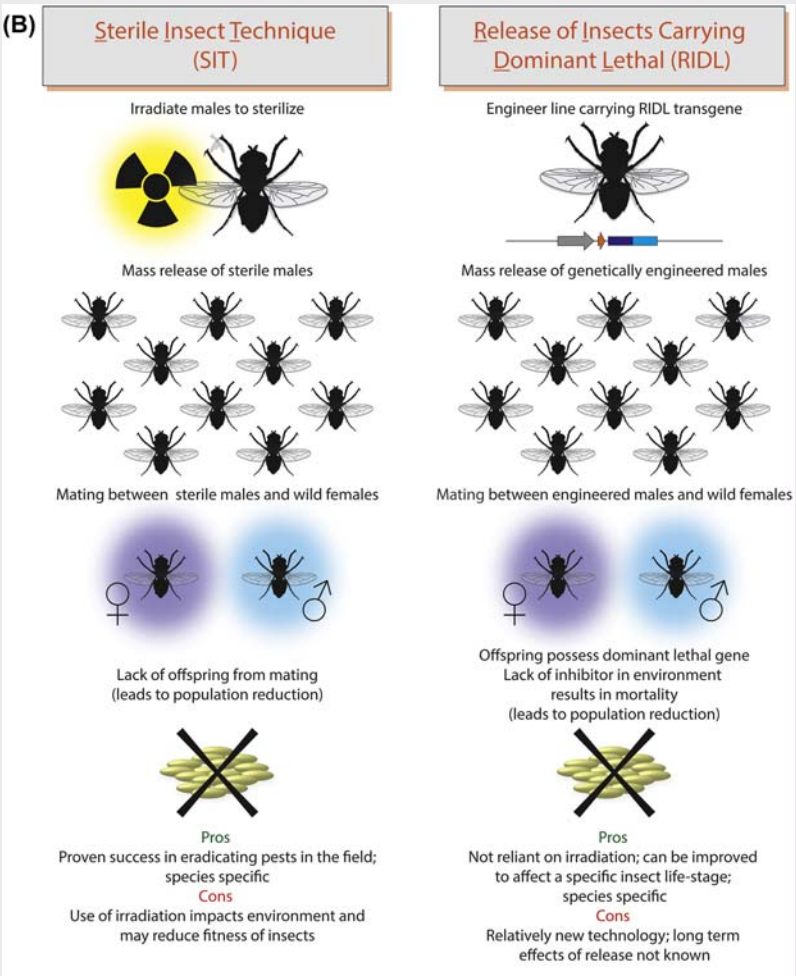
Like other disciplines in the biological sciences, molecular tools provide opportunities for medical and veterinary entomologists to investigate vectors, pathogens, and hosts in innovative and novel ways. There are many examples of the broad impact of molecular tools: (1) genomic databases can reveal novel targets for pest control existing in an insect's genome; (2) expression analysis and RNAi help in determining the function of these genes within organisms; (3) CRISPR and RIDL show promise in manipulating

BOX 28.2 Manipulating Arthropods: Insect Population Suppression



Continued

BOX 28.2 Manipulating Arthropods: Insect Population Suppression—cont'd

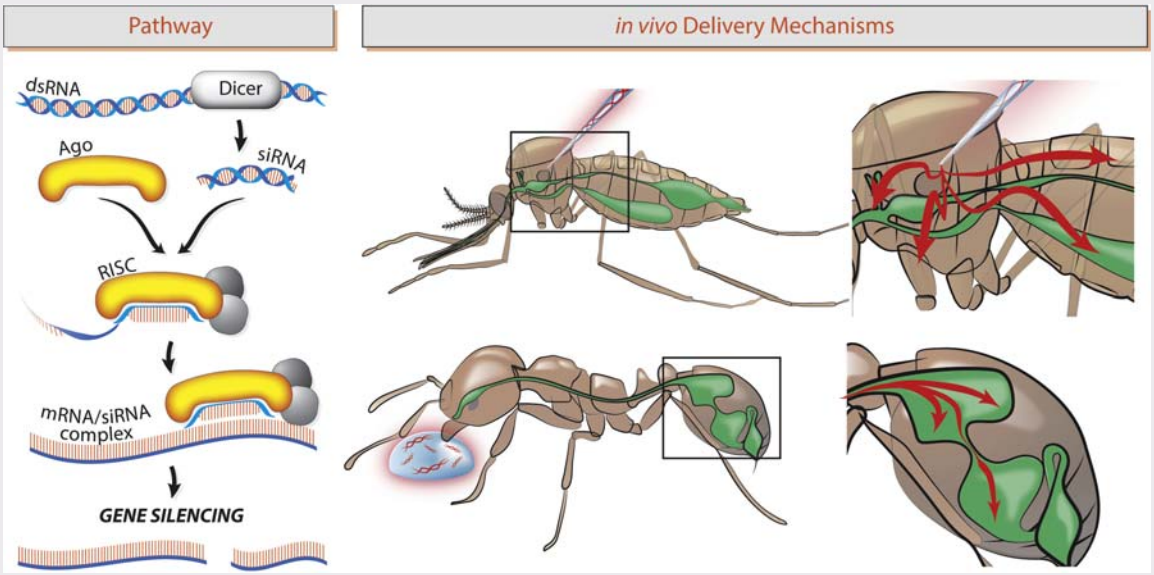


(A) RIDL constructs. The tetracycline transactivator protein (tTA) is placed under the control of an insect-specific, core promoter that directs where tTA will be expressed. The core promoter is fused to the tetracycline operator (tetO), and these are located upstream of the tTA. Operators are sites where transcription factors typically bind to repress transcription. The tTA protein, however, is composed of a transcription activation domain fused to a tet repressor, which allows tTA to bind at tetO and induce expression rather than repress. Tetracycline (inhibitor) can bind tTA and, when present, turns the system "off." In the absence of tetracycline, tTA circulates and binds at the tetO site, resulting in increased expression and production of more tTA (positive feedback). This conditional upregulation of the transactivator protein results in organismal lethality from "transcriptional squelching."
(B) Comparison of the sterile insect technique (SIT) and repressible induced dominant lethality (RIDL). Both techniques are used to suppress insect populations. *Illustration by Victoria Rhodes.*

vectors and preventing the spread of devastating diseases; (4) rapid diagnostic techniques aid clinicians in assessing infection and saving lives; and (5) population genetics and methods for detecting pathogens in arthropod vectors in the field can improve predictive models for the spread of vector-borne diseases.

Next-generation sequencing (NGS) techniques have transformed clinical diagnostics and revolutionized the discovery of novel pathogens in vector species. Adapting current sequence analysis pipelines to medical and veterinary entomology will improve the design and optimization of PCR primers for diagnostically informative genes. **Targeted-sequencing** platforms are being used in metagenomics (genomic analysis of microbial populations present in environmental samples), small genome sequencing, and amplicon sequencing. *De novo* sequencing

BOX 28.3 Manipulating Arthropods: Silencing Genes With RNAi



To perform RNAi, a dsRNA is generated that is homologous in sequence to the target mRNA. When this dsRNA is introduced to the organism in vivo, usually by injection or feeding, an RNase called Dicer cleaves the dsRNA into small interfering RNA (siRNA) fragments. This initiates assembly of the RNA-induced silencing complex (RISC) including the essential component argonaute (Ago). Antisense fragments of siRNAs have nucleotide sequences complementary to the targeted mRNA strand, which the RISC uses to bind mRNAs containing those sequences, either inhibiting or inactivating them in the process. The result is that the target mRNA transcripts are not translated into proteins (gene silencing). Gene function can be inferred by observing subsequent changes to the phenotype. RNAi is used in reverse genetics studies in arthropods by introducing dsRNA (by microinjection into the hemocoel or feeding) and looking at in vivo effects. Oral delivery of RNAi is also being investigated as novel insecticidal treatments in several species of arthropods. Here, the dsRNA or siRNA is provided in a bait, which is ingested by the insect pest. *Illustration by Victoria Rhodes.*

(e.g., Illumina, PacBio) allows identification of multiple species from a single sample and aids in distinguishing new strains. Sample preparation kits are being developed and tested to permit sequencing of specimens in the field for real-time biosurveillance.

Computational biology helps researchers identify trends and patterns in their data that may not be easy to distinguish using standard methods. In vector-borne disease surveillance, for example, large data sets are compiled and analyzed in order to identify patterns and predict outcomes. These data may come from unexpected sources such as medical claims, mobile phone call data records, geographically tagged Twitter posts, and other social media platforms (Brownstein et al., 2009; Schmidt, 2012; Mekaru and Brownstein, 2014; Aslam et al., 2014). The future is likely to include improved computational approaches to mine "big data" generated by these sources and the impact they can have on vector-borne disease surveillance.

Medical and veterinary entomologists are exploiting this bourgeoning arsenal of techniques and procedures to answer questions about, and provide solutions for, problems involving medical and veterinary pests of humans and other animals. Not only are entomology students coevolving with these innovative technologies, but this interplay is likely to redefine the field of medical and veterinary entomology.

REFERENCES AND FURTHER READING

Alphey, L. (2016a). Can CRISPR-Cas9 gene drives curb malaria? *Nature Biotechnology, 34*, 149–150.

Alphey, L. (2016b). SIT 2.0: 21st century genetic technology for the screwworm sterile-insect program. *BMC Biology, 14*, 80.

Anderson, J. M., Oliveira, F., Kamhawi, S., Mans, B. J., Reynoso, D., Seitz, A. E., et al. (2006). Comparative salivary gland transcriptomics of sandfly vectors of visceral leishmaniasis. *BMC Genomics, 7*, 52.

Aslam, A. A., Tsou, M. H., Spitzberg, B. H., An, L., Gawron, J. M., Gupta, D. K., et al. (2014). The reliability of tweets as a supplementary method of seasonal influenza surveillance. *Journal of Medical Internet Research, 16*, e250.

Baker, M. (2012). Digital PCR hits its stride. *Nature Methods, 9*, 541–544.

Barnard, A. C., Nijhof, A. M., Fick, W., Stutzer, C., & Maritz-Olivier, C. (2012). RNAi in arthropods: Insight into the machinery and applications for understanding the pathogen-vector interface. *Genes (Basel), 3*, 702−741.

Barrangou, R., & Doudna, J. A. (2016). Applications of CRISPR technologies in research and beyond. *Nature Biotechnology, 34*, 933−941.

Baum, J. A., & Roberts, J. K. (2014). Progress towards RNAi-mediated insect pest management. *Advances in Insect Physiology, 47*, 249−295.

Belles, X. (2010). Beyond Drosophila: RNAi in vivo and functional genomics in insects. *Annual Review of Entomology, 55*, 111−128.

Brownstein, J. S., Freifeld, C. C., & Madoff, L. C. (2009). Digital disease detection−harnessing the Web for public health surveillance. *New England Journal of Medicine, 360*, 2153−2155, 2157.

Campbell, C. L., Vandyke, K. A., Letchworth, G. J., Drolet, B. S., Hanekamp, T., & Wilson, W. C. (2005). Midgut and salivary gland transcriptomes of the arbovirus vector *Culicoides sonorensis* (Diptera: Ceratopogonidae). *Insect Molecular Biology, 14*, 121−136.

Carvalho, D. O., McKemey, A. R., Garziera, L., Lacroix, R., Donnelly, C. A., Alphey, L., et al. (2015). Suppression of a field population of *Aedes aegypti* in Brazil by sustained release of transgenic male mosquitoes. *PLoS Neglected Tropical Diseases, 9*, e0003864.

Chalaire, K. C., Kim, T. K., Garcia-Rodriguez, H., & Mulenga, A. (2011). *Amblyomma americanum* (L.) (Acari: Ixodidae) tick salivary gland serine protease inhibitor (serpin) 6 is secreted into tick saliva during tick feeding. *Journal of Experimental Biology, 214*, 665−673.

Concha, C., Palavesam, A., Guerrero, F. D., Sagel, A., Li, F., Osborne, J. A., et al. (2016). A transgenic male-only strain of the New World screwworm for an improved control program using the sterile insect technique. *BMC Biology, 14*, 72.

Dame, D. A., Curtis, C. F., Benedict, M. Q., Robinson, A. S., & Knols, B. G. (2009). Historical applications of induced sterilisation in field populations of mosquitoes. *Malaria Journal, 8*(Suppl. 2), S2.

Domingues, L. N., Guerrero, F. D., & Foil, L. D. (2014). Simultaneous detection of pyrethroid, organophosphate, and cyclodiene target site resistance in *Haematobia irritans* (Diptera: Muscidae) by multiplex polymerase chain reaction. *Journal of Medical Entomology, 51*, 964−970.

Doudna, J. A., & Charpentier, E. (2014). Genome editing. The new frontier of genome engineering with CRISPR-Cas9. *Science, 346*, 1258096.

Drolet, B. S., Campbell, C. L., Stuart, M. A., & Wilson, W. C. (2005). Vector competence of *Culicoides sonorensis* (Diptera: Ceratopogonidae) for vesicular stomatitis virus. *Journal of Medical Entomology, 42*, 409−418.

Fire, A. (1999). RNA-triggered gene silencing. *Trends in Genetics, 15*, 358−363.

Fischbach, J., Xander, N. C., Frohme, M., & Glokler, J. F. (2015). Shining a light on LAMP assays − a comparison of LAMP visualization methods including the novel use of berberine. *Biotechniques, 58*, 189−194.

Fu, G., Lees, R. S., Nimmo, D., Aw, D., Jin, L., Gray, P., et al. (2010). Female-specific flightless phenotype for mosquito control. *Proceedings of the National Academy of Sciences of the United States of America, 107*, 4550−4554.

Giraldo-Calderon, G. I., Emrich, S. J., MacCallum, R. M., Maslen, G., Dialynas, E., Topalis, P., et al. (2015). VectorBase: An updated bioinformatics resource for invertebrate vectors and other organisms related with human diseases. *Nucleic Acids Research, 43*, D707−D713.

Gong, P., Epton, M. J., Fu, G., Scaife, S., Hiscox, A., Condon, K. C., et al. (2005). A dominant lethal genetic system for autocidal control of the Mediterranean fruitfly. *Nature Biotechnology, 23*, 453−456.

Guerrero, F. D., Alison, M. W., Jr., Kammlah, D. M., & Foil, L. D. (2002). Use of the polymerase chain reaction to investigate the dynamics of pyrethroid resistance in *Haematobia irritans irritans* (Diptera: Muscidae). *Journal of Medical Entomology, 39*, 747−754.

Guerrero, F. D., Davey, R. B., & Miller, R. J. (2001). Use of an allele-specific polymerase chain reaction assay to genotype pyrethroid resistant strains of *Boophilus microplus* (Acari: Ixodidae). *Journal of Medical Entomology, 38*, 44−50.

Hall, A. B., Basu, S., Jiang, X., Qi, Y., Timoshevskiy, V. A., Biedler, J. K., et al. (2015). Sex Determination. A male-determining factor in the mosquito *Aedes aegypti*. *Science, 348*, 1268−1270.

Harris, A. F., McKemey, A. R., Nimmo, D., Curtis, Z., Black, I., Morgan, S. A., et al. (2012). Successful suppression of a field mosquito population by sustained release of engineered male mosquitoes. *Nature Biotechnology, 30*, 828−830.

Heinrich, J. C., & Scott, M. J. (2000). A repressible female-specific lethal genetic system for making transgenic insect strains suitable for a sterile-release program. *Proceedings of the National Academy of Sciences of the United States of America, 97*, 8229−8232.

Hill, C. A., Kafatos, F. C., Stansfield, S. K., & Collins, F. H. (2005). Arthropod-borne diseases: Vector control in the genomics era. *Nature Reviews Microbiology, 3*, 262−268.

Hindson, C. M., Chevillet, J. R., Briggs, H. A., Gallichotte, E. N., Ruf, I. K., Hindson, B. J., et al. (2013). Absolute quantification by droplet digital PCR versus analog real-time PCR. *Nature Methods, 10*, 1003−1005.

Holt, R. A., Subramanian, G. M., Halpern, A., Sutton, G. G., Charlab, R., Nusskern, D. R., et al. (2002). The genome sequence of the malaria mosquito *Anopheles gambiae*. *Science, 298*, 129−149.

Hoy, M. A. (2003). *Insect molecular Genetics: An introduction to principles and application* (2nd ed.). Burlington, Massachusetts: Academic Press.

i5K Consortium. (2013). The i5K initiative: Advancing arthropod genomics for knowledge, human health, agriculture, and the environment. *Journal of Heredity, 104*, 595−600.

Inayoshi, M., Naitou, H., Kawamori, F., Masuzawa, T., & Ohashi, N. (2004). Characterization of *Ehrlichia* species from *Ixodes ovatus* ticks at the foot of Mt. Fuji, Japan. *Microbiology and Immunology, 48*, 737−745.

Jain, M., Olsen, H. E., Paten, B., & Akeson, M. (2016). The Oxford nanopore MinION: Delivery of nanopore sequencing to the genomics community. *Genome Biology, 17*, 239.

Jones, K. E., Patel, N. G., Levy, M. A., Storeygard, A., Balk, D., Gittleman, J. L., et al. (2008). Global trends in emerging infectious diseases. *Nature, 451*, 990−993.

Joyner, C., Mills, M. K., & Nayduch, D. (2013). *Pseudomonas aeruginosa* in *Musca domestica* L.: Temporospatial examination of bacteria population dynamics and house fly antimicrobial responses. *PLoS One, 8*, e79224.

King, J. G., Vernick, K. D., & Hillyer, J. F. (2011). Members of the salivary gland surface protein (SGS) family are major immunogenic components of mosquito saliva. *Journal of Biological Chemistry, 286*, 40824–40834.

Kistler, K. E., Vosshall, L. B., & Matthews, B. J. (2015). Genome engineering with CRISPR-Cas9 in the mosquito *Aedes aegypti*. *Cell Reports, 11*, 51–60.

Koutsos, A. C., Blass, C., Meister, S., Schmidt, S., MacCallum, R. M., Soares, M. B., et al. (2007). Life cycle transcriptome of the malaria mosquito *Anopheles gambiae* and comparison with the fruitfly *Drosophila melanogaster*. *Proceedings of the National Academy of Sciences of the United States of America, 104*, 11304–11309.

Labbe, G. M., Scaife, S., Morgan, S. A., Curtis, Z. H., & Alphey, L. (2012). Female-specific flightless (fsRIDL) phenotype for control of *Aedes albopictus*. *PLoS Neglected Tropical Diseases, 6*, e1724.

Lehiy, C. J., & Drolet, B. S. (2014). The salivary secretome of the biting midge, *Culicoides sonorensis*. *PeerJ, 2*, e426.

Li, F., Vensko, S. P., 2nd, Belikoff, E. J., & Scott, M. J. (2013a). Conservation and sex-specific splicing of the transformer gene in the calliphorids *Cochliomyia hominivorax, Cochliomyia macellaria* and *Lucilia sericata*. *PLoS One, 8*, e56303.

Li, M., Reid, W. R., Zhang, L., Scott, J. G., Gao, X., Kristensen, M., et al. (2013b). A whole transcriptomal linkage analysis of gene co-regulation in insecticide resistant house flies, *Musca domestica*. *BMC Genomics, 14*, 803.

Madden, R. D., Sauer, J. R., & Dillwith, J. W. (2004). A proteomics approach to characterizing tick salivary secretions. *Experimental and Applied Acarology, 32*, 77–87.

Maleki-Ravasan, N., Oshaghi, M., Javadian, E., Rassi, Y., Sadraei, J., & Mohtarami, F. (2009). Blood meal identification in field-captured sand flies: Comparison of PCR-RFLP and ELISA assays. *Iranian Journal of Arthropod-Borne Diseases, 3*, 8–18.

Marfurt, J., Niederwieser, I., Makia, N. D., Beck, H. P., & Felger, I. (2003). Diagnostic genotyping of Old and new world *Leishmania* species by PCR-RFLP. *Diagnostic Microbiology and Infectious Disease, 46*, 115–124.

Mastrangelo, T., & Welch, J. B. (2012). An overview of the components of AW-IPM campaigns against the New World Screwworm. *Insects, 3*, 930–955.

Mekaru, S. R., & Brownstein, J. S. (2014). One Health in social networks and social media. *Revue Scientifique et Technique, 33*, 629–637.

Mills, M. K., Nayduch, D., & Michel, K. (2015). Inducing RNA interference in the arbovirus vector, *Culicoides sonorensis*. *Insect Molecular Biology, 24*, 105–114.

Muir, P., Li, S., Wang, D., Spakowicz, D. J., Salichos, L., Zhang, J., et al. (2016). The real cost of sequencing: Scaling computation to keep pace with data generation. *Genome Biology, 17*, 53.

Nayduch, D., & Aksoy, S. (2007). Refractoriness in tsetse flies (Diptera: Glossinidae) may be a matter of timing. *Journal of Medical Entomology, 44*, 660–665.

Nayduch, D., Lee, M. B., & Saski, C. A. (2014a). Gene discovery and differential expression analysis of humoral immune response elements in female *Culicoides sonorensis* (Diptera: Ceratopogonidae). *Parasites and Vectors, 7*, 388.

Nayduch, D., Lee, M. B., & Saski, C. A. (2014b). The reference transcriptome of the adult female biting midge (*Culicoides sonorensis*) and differential gene expression profiling during teneral, blood, and sucrose feeding conditions. *PLoS One, 9*, e98123.

Neafsey, D. E., Waterhouse, R. M., Abai, M. R., Aganezov, S. S., Alekseyev, M. A., Allen, J. E., et al. (2015). Mosquito genomics. Highly evolvable malaria vectors: The genomes of 16 *Anopheles* mosquitoes. *Science, 347*, 1258522.

Ngo, K. A., & Kramer, L. D. (2003). Identification of mosquito bloodmeals using polymerase chain reaction (PCR) with order-specific primers. *Journal of Medical Entomology, 40*, 215–222.

Noedl, H., Yingyuen, K., Laoboonchai, A., Fukuda, M., Sirichaisinthop, J., & Miller, R. S. (2006). Sensitivity and specificity of an antigen detection ELISA for malaria diagnosis. *The American Journal of Tropical Medicine and Hygiene, 75*, 1205–1208.

Noel, S., Tessier, N., Angers, B., Wood, D. M., & Lapointe, F. J. (2004). Molecular identification of two species of myiasis-causing *Cuterebra* by multiplex PCR and RFLP. *Medical and Veterinary Entomology, 18*, 161–166.

Olafson, P. U. (2013). Molecular characterization and immunolocalization of the olfactory co-receptor Orco from two blood-feeding muscid flies, the stable fly (*Stomoxys calcitrans*, L.) and the horn fly (*Haematobia irritans irritans*, L.). *Insect Molecular Biology, 22*, 131–142.

Oxitec. (2016a). *Dengue fever cases drop 91% in neighborhood of Piracicaba, Brazil, where Oxitec's Friendly™ Aedes were released, July 14*. http://www.oxitec.com/dengue-fever-cases-drop-91-percent-neighbourhood-piracicaba-brazil-oxitecs-friendly-aedes-released/.

Oxitec. (2016b). *Board of Florida keys mosquito control District approves investigational agreement for effectiveness trial of Oxitec's Friendly (TM) mosquitoes in Monroe county, Florida, November 21*. http://www.oxitec.com/board-florida-keys-mosquito-control-district-approves-investigational-agreement-monroe-county/.

Parida, M., Horioke, K., Ishida, H., Dash, P. K., Saxena, P., Jana, A. M., et al. (2005). Rapid detection and differentiation of dengue virus serotypes by a real-time reverse transcription-loop-mediated isothermal amplification assay. *Journal of Clinical Microbiology, 43*, 2895–2903.

Parida, M., Posadas, G., Inoue, S., Hasebe, F., & Morita, K. (2004). Real-time reverse transcription loop-mediated isothermal amplification for rapid detection of West Nile virus. *Journal of Clinical Microbiology, 42*, 257–263.

Paupy, C., Orsoni, A., Mousson, L., & Huber, K. (2004). Comparisons of amplified fragment length polymorphism (AFLP), microsatellite, and isoenzyme markers: Population genetics of *Aedes aegypti* (Diptera: Culicidae) from Phnom Penh (Cambodia). *Journal of Medical Entomology, 41*, 664–671.

Pfaffl, M. W., Horgan, G. W., & Dempfle, L. (2002). Relative expression software tool (REST) for group-wise comparison and statistical analysis of relative expression results in real-time PCR. *Nucleic Acids Research, 30*, e36.

Phuc, H. K., Andreasen, M. H., Burton, R. S., Vass, C., Epton, M. J., Pape, G., et al. (2007). Late-acting dominant lethal genetic systems and mosquito control. *BMC Biology, 5*, 11.

Price, D. P., Nagarajan, V., Churbanov, A., Houde, P., Milligan, B., Drake, L. L., et al. (2011). The fat body transcriptomes of the yellow fever mosquito *Aedes aegypti*, pre- and post- blood meal. *PLoS One, 6*, e22573.

Reichard, R. E. (2002). Area-wide biological control of disease vectors and agents affecting wildlife. *Revue Scientifique et Technique, 21*, 179–185.

Reuter, J. A., Spacek, D. V., & Snyder, M. P. (2015). High-throughput sequencing technologies. *Molecular Cell, 58*, 586–597.

Ribeiro, J. M., & Francischetti, I. M. (2003). Role of arthropod saliva in blood feeding: Sialome and post-sialome perspectives. *Annual Review of Entomology, 48*, 73−88.

Ribeiro, J. M., Mans, B. J., & Arca, B. (2010). An insight into the sialome of blood-feeding Nematocera. *Insect Biochemistry and Molecular Biology, 40*, 767−784.

Richards, S. (2015). It's more than stamp collecting: How genome sequencing can unify biological research. *Trends in Genetics, 31*, 411−421.

Richards, S., & Murali, S. C. (2015). Best practices in insect genome sequencing: What works and what doesn't. *Current Opinion in Insect Science, 7*, 1−7.

Rinker, D. C., Zhou, X., Pitts, R. J., Consortium, A. G. C., Rokas, A., & Zwiebel, L. J. (2013). Antennal transcriptome profiles of anopheline mosquitoes reveal human host olfactory specialization in *Anopheles gambiae*. *BMC Genomics, 14*, 749.

Schmidt, C. W. (2012). Trending now: Using social media to predict and track disease outbreaks. *Environmental Health Perspectives, 120*, A30−A33.

Scott, J. G., Michel, K., Bartholomay, L. C., Siegfried, B. D., Hunter, W. B., Smagghe, G., et al. (2013). Towards the elements of successful insect RNAi. *Journal of Insect Physiology, 59*, 1212−1221.

Scott, J. G., Warren, W. C., Beukeboom, L. W., Bopp, D., Clark, A. G., Giers, S. D., et al. (2014). Genome of the house fly, *Musca domestica* L., a global vector of diseases with adaptations to a septic environment. *Genome Biology, 15*, 466.

Scott, M. J. (2014). Development and evaluation of male-only strains of the Australian sheep blowfly, *Lucilia cuprina*. *BMC Genetics, 15*(Suppl. 2), S3.

Sontheimer, E. J., & Barrangou, R. (2015). The Bacterial origins of the CRISPR genome-editing revolution. *Human Gene Therapy, 26*, 413−424.

Steen, H., & Mann, M. (2004). The ABC's (and XYZ's) of peptide sequencing. *Nature Reviews Molecular Cell Biology, 5*, 699−711.

Stephens, Z. D., Lee, S. Y., Faghri, F., Campbell, R. H., Zhai, C., Efron, M. J., et al. (2015). Big Data: Astronomical or genomical? *PLoS Biology, 13*, e1002195.

Thomas, D. D., Donnelly, C. A., Wood, R. J., & Alphey, L. S. (2000). Insect population control using a dominant, repressible, lethal genetic system. *Science, 287*, 2474−2476.

Toriniwa, H., & Komiya, T. (2006). Rapid detection and quantification of Japanese encephalitis virus by real-time reverse transcription loop-mediated isothermal amplification. *Microbiology and Immunology, 50*, 379−387.

Trout, R. T., Steelman, C. D., & Szalanski, A. L. (2010). Population genetics of *Amblyomma americanum* (Acari: Ixodidae) collected from Arkansas. *Journal of Medical Entomology, 47*, 152−161.

Valenzuela, J. G. (2004). Exploring tick saliva: From biochemistry to 'sialomes' and functional genomics. *Parasitology, 129*(Suppl.), S83−S94.

Valenzuela, J. G., Francischetti, I. M., Pham, V. M., Garfield, M. K., & Ribeiro, J. M. (2003). Exploring the salivary gland transcriptome and proteome of the *Anopheles stephensi* mosquito. *Insect Biochemistry and Molecular Biology, 33*, 717−732.

Valenzuela, J. G., Garfield, M., Rowton, E. D., & Pham, V. M. (2004). Identification of the most abundant secreted proteins from the salivary glands of the sand fly *Lutzomyia longipalpis*, vector of Leishmania chagasi. *Journal of Experimental Biology, 207*, 3717−3729.

van Dijk, E. L., Auger, H., Jaszczyszyn, Y., & Thermes, C. (2014). Ten years of next-generation sequencing technology. *Trends in Genetics, 30*, 418−426.

Venczel, R., Knoke, L., Pavlovic, M., Dzaferovic, E., Vaculova, T., Silaghi, C., et al. (2016). A novel duplex real-time PCR permits simultaneous detection and differentiation of *Borrelia miyamotoi* and *Borrelia burgdorferi* sensu lato. *Infection, 44*, 47−55.

Wang, H., La Russa, M., & Qi, L. S. (2016). CRISPR/Cas9 in genome editing and beyond. *Annual Review of Biochemistry, 85*, 227−264.

Wang, X., Ribeiro, J. M., Broce, A. B., Wilkerson, M. J., & Kanost, M. R. (2009). An insight into the transcriptome and proteome of the salivary gland of the stable fly, *Stomoxys calcitrans*. *Insect Biochemistry and Molecular Biology, 39*, 607−614.

WHO-World Health Organization. (2014). *A global brief on vector-borne diseases*. Available at: http://apps.who.int/iris/bitstream/10665/111008/1/WHO_DCO_WHD_2014.1_eng.pdf.

Wilson, M., Glaser, K. C., Adams-Fish, D., Boley, M., Mayda, M., & Molestina, R. E. (2015). Development of droplet digital PCR for the detection of *Babesia microti* and *Babesia duncani*. *Experimental Parasitology, 149*, 24−31.

Zhang, Y., Zhao, B., Roy, S., Saha, T. T., Kokoza, V. A., Li, M., et al. (2016). microRNA-309 targets the Homeobox gene SIX4 and controls ovarian development in the mosquito *Aedes aegypti*. *Proceedings of the National Academy of Sciences of the United States of America, 113*, E4828−E4836.

Appendix

Arthropod-Related Viruses of Medical and Veterinary Importance

Michael J. Turell

Virology Division, United States Army Medical Research Institute of Infectious Diseases, Fort Detrick, Frederick, MD, United States (retired)

Hundreds of different viruses have been associated with arthropods. Although each of these viruses can be classified phylogenetically into various orders, families, genera, and species, it has been useful to characterize them ecologically by their association with arthropods. These viruses can be placed in four ecological groups: (1) arthropod-borne viruses, (2) arthropod-transmitted animal viruses, (3) arthropod viruses, and (4) arthropod-transmitted plant viruses. The first two groups include most of the viruses of medical and/or veterinary importance. **Arthropod-borne viruses** are usually referred to by the shortened term **arboviruses**. These viruses replicate in both arthropod vectors and vertebrate hosts and can be transmitted between vertebrate hosts by the arthropod vector (Table A.1). Infections of vertebrates with these viruses may result in clinical disease or death. **Arthropod-transmitted animal viruses** are similar to arboviruses except that they do not replicate in the arthropod vector (Table A.2). Transmission is mechanical rather than biological. **Arthropod viruses** replicate only in an arthropod. Because they do not replicate in a vertebrate host, they do not cause disease in humans or other vertebrates, although infection of the arthropod may be deleterious to that arthropod. **Arthropod-transmitted plant viruses** can be transmitted either mechanically or, in some cases, biologically, by a variety of arthropods, including aphids, leafhoppers, plant bugs, and certain plant mites. Because viruses in the latter two groups do not directly affect vertebrates, they are not included in the tables provided here.

Arboviruses are often named for (1) the disease that they cause (e.g., chikungunya, yellow fever, and bluetongue viruses); (2) the location where the virus or disease was first described (e.g., Barmah Forest, West Nile, Zika, and Sindbis viruses); or (3) a combination of the two (e.g., Crimean-Congo hemorrhagic fever, eastern equine encephalitis, and Colorado tick fever viruses). When the full name of a virus is written out, only proper nouns should be capitalized, such as western equine encephalitis virus, Venezuelan equine encephalitis virus, and Rift Valley fever virus. Unlike family names for other groups of biological organisms, the current convention is to italicize virus family as well as genus names. A standard two- to four-letter abbreviation has been approved for each of these viruses, as indicated in the tables.

Arboviruses are members of the following five families: *Togaviridae* (genus *Alphavirus*), *Flaviviridae* (genus *Flavivirus*), *Bunyaviridae* (various genera), *Rhabdoviridae* (genus *Vesiculovirus*), and *Reoviridae* (genus *Orbivirus*). Although viruses can contain either RNA or DNA, arboviruses all have nucleic acid in the form of RNA that can be either single or double stranded; most, however, are single stranded. They can be either positive or negative sense. The RNA of a positive-sense virus is infectious if it enters a cell, while reverse transcriptase is required to transcribe negative-sense RNA to enable it to infect a cell. The viruses can have either a single strand of RNA or be multisegmented. Some viruses, such as members of the families *Togaviridae* and *Flaviviridae,* contain a single strand of RNA, while other viruses, such as the members of the *Bunyaviridae*, contain multiple strands of RNA, each encoding separate genes. If two closely related viruses with multiple strands of RNA infect the same cell, it is possible for the viruses to reassort to produce a new virus that contains genes from each of the two initial viruses. Some arboviruses, such as Japanese encephalitis and the various serotypes of dengue virus, currently cause millions of infections in humans and tens of thousands of fatalities each year, while infections with Zika virus have been associated with severe birth defects, including microcephaly. Others, such as Rift Valley fever

TABLE A.1 Selected Arthropod-Borne Viruses (Arboviruses) (Viruses That Replicate in Arthropods and are Dependent on Arthropod Vectors for Transmission)

Virus	Abbreviation	Family	Genus	Serogroup	Animals Affected	Principal Vectors	Disease[a]	Distribution	Comments
African horse sickness	AHS	*Reoviridae*	*Orbivirus*	Bluetongue	Wild/domestic equids, Horses, Mules	*Culicoides* midges *C. imicola*	F	Africa, Europe, India Middle East	
African swine fever	ASF	*Asfarviridae*	*Asfivirus*	Ungrouped	Swine	*Ornithodoros* ticks	F, H	Sub-Saharan Africa Eurasia	Also mechanical transmission
Barmah Forest	BF	*Togaviridae*	*Alphavirus*	Barmah Forest	Marsupials, Humans	*Culex* mosquitoes *Cx. annulirostris, Ae. vigilax*	F, A	Australia	Similar disease as caused by RR virus
Bluetongue	BLU	*Reoviridae*	*Orbivirus*	Bluetongue	Wild Ruminants, Sheep, Cattle, Goats	*Culicoides* midges *C. sonorensis, C. imicola*	F, V, H	Worldwide	Can be confused with foot and mouth disease
Bunyamwera	BUN	*Bunyaviridae*	*Orthobunyavirus*	Bunyamwera	Rodents	Mosquitoes	F, A	Africa	
Cache Valley	CV	*Bunyaviridae*	*Orthobunyavirus*	Bunyamwera	Ungulates, Sheep	Mosquitoes *An. quadrimaculatus*	F	North America	Causes congenital malformations in sheep
Caraparu	CAR	*Bunyaviridae*	*Orthobunyavirus*	Group C	Rodents, Humans	Mosquitoes	F	North/South America	
California encephalitis	CE	*Bunyaviridae*	*Orthobunyavirus*	California	Rodents, Rabbits, Humans	*Aedes* mosquitoes *Ae. dorsalis, Ae. melanimon*	F, N	Western US	
Chandipura	CHP	*Rhabdoviridae*	*Vesiculovirus*	VS	Hedgehogs, Humans	Sand flies *P. papatasi*	F, N	India, Africa	
Changuinola	CGL	*Reoviridae*	*Orbivirus*	Changuinola	Sloths, Humans	Sand flies *Lutzomyia* spp.	F	Central/South America	
Chikungunya	CHIK	*Togaviridae*	*Alphavirus*	Semliki Forest	Humans	*Aedes* mosquitoes *Ae. aegypti, Ae. albopictus*	F, A	Africa, Asia, North/South America	Spread to the New World in 2013

Colorado tick fever	CTF	*Reoviridae*	*Orbivirus*	Colorado tick fever	Rodents, Humans	Ticks *D. andersoni*	F	Western Canada, US, Northern Mexico	200–300 cases/year in US
Crimean-Congo hemorrhagic fever	CCHF	*Bunyaviridae*	*Nairovirus*	CCHF group	Hares, Hedgehogs, Cattle, Humans	*Hyalomma* ticks *H. marginatum, H. asiaticum*	F, H, N	Africa, Asia	
Dengue 1, 2, 3, and 4	DEN	*Flaviviridae*	*Flavivirus*	Dengue	Humans	*Aedes* (*Stegomyia*) mosquitoes *Ae. aegypti, Ae. albopictus*	F, A, H	Tropics Worldwide	~50 million cases worldwide/year
Eastern equine encephalitis	EEE	*Togaviridae*	*Alphavirus*	EEE	Birds, Horses, Humans	Mosquitoes *Cs. melanura*	F, N	Eastern Canada to Argentina	
Epizootic hemorrhagic disease	EHD	*Reoviridae*	*Orbivirus*	Bluetongue	Wild Ruminants, Deer, Cattle	*Culicoides* midges *C. variipennis*	F, H	Worldwide	
Highlands J	HJ	*Togaviridae*	*Alphavirus*	WEE	Birds	Mosquitoes *Cs. melanura*	None	Eastern US	Often confused with EEE virus
Itaqui	ITQ	*Bunyaviridae*	*Orthobunyavirus*	Group C	Rodents, Humans	Mosquitoes	F	South America	
Jamestown Canyon	JC	*Bunyaviridae*	*Orthobunyavirus*	California	Rodents, Humans	Mosquitoes *Aedes* and *Culiseta* spp.	F, N	Southern Canada Temperate US	Includes SSH virus
Japanese encephalitis	JBE	*Flaviviridae*	*Flavivirus*	Japanese encephalitis	Birds, Swine, Horses, Humans	*Culex* mosquitoes *Cx. tritaeniorhynchus*	F, N	Asia	~50,000 cases/year in Asia
Karshi	KSI	*Flaviviridae*	*Flavivirus*	Mammalian tick-borne flavivirus group	Rodents?, Humans	Ticks *Ornithodoros* spp.	F, N	Western Asia	
Kunjin	KUN	*Flaviviridae*	*Flavivirus*	Japanese encephalitis	Birds, Humans	*Culex* mosquitoes *Cx. annulirostris*	F, N	Australia	Considered a subtype of WNV
Kyasanur Forest disease	KFD	*Flaviviridae*	*Flavivirus*	Mammalian tick-borne flavivirus group	Rodents, Monkeys, Birds, Humans	Ticks, *Haemaphysalis* spp.	F, H, N	Southern Asia	

Continued

TABLE A.1 Selected Arthropod-Borne Viruses (Arboviruses) (Viruses That Replicate in Arthropods and are Dependent on Arthropod Vectors for Transmission)—cont'd

Virus	Abbreviation	Family	Genus	Serogroup	Animals Affected	Principal Vectors	Disease[a]	Distribution	Comments
La Crosse	LAC	*Bunyaviridae*	*Orthobunyavirus*	California	Rodents, Humans	*Aedes* mosquitoes *Ae. triseriatus*	F, N	Midwestern-Mid Atlantic states in US	~70 cases/ year in US
Langat	LGT	*Flaviviridae*	*Flavivirus*	Mammalian tick-borne flavivirus group	Rodents	Ticks, both ixodid and argasid	F, N	Asia	
Louping ill	LI	*Flaviviridae*	*Flavivirus*	Mammalian tick-borne flavivirus group	Rodents, Sheep, Humans	Ticks *I. ricinus*	F, N	United Kingdom	
Mayaro	MAY	*Togaviridae*	*Alphavirus*	Semliki Forest	Primates, Rodents, Birds, Humans	Mosquitoes *Haemagogus* spp.	F, A	South America Caribbean islands	
Murray Valley encephalitis	MVE	*Flaviviridae*	*Flavivirus*	Japanese encephalitis	Birds, Humans	*Culex* mosquitoes *Cx. annulirostris*	F, N	Australia, New Guinea	
Nairobi sheep disease	NSD	*Bunyaviridae*	*Nairovirus*	NSD group	Rodents, Sheep, Goats	Ixodid ticks *R. appendiculatus*	F, H	East Africa	
Omsk hemorrhagic fever	OMSK	*Flaviviridae*	*Flavivirus*	Mammalian tick-borne flavivirus group	Rodents, Goat, Sheep, Humans	Ixodid ticks *Dermacentor* and *Ixodes* spp.	F, H	Western Siberia	Also spread from contaminated milk
O'nyong'nyong	ONN	*Togaviridae*	*Alphavirus*	Semliki Forest	Humans	*Anopheles* mosquitoes *An. funestus, An. gambiae*	F, A	Africa	Closely related to CHIKV
Oropouche	ORO	*Bunyaviridae*	*Orthobunyavirus*	Simbu group	Primates, Sloths, Humans	*Culicoides* spp., Mosquitoes? *C. paraensis*	F, A	South America Caribbean	
Powassan	POW	*Flaviviridae*	*Flavivirus*	Mammalian tick-borne flavivirus group	Rodents, Humans	Ticks *I. cookei*	F, N	Northern US, Canada	

Rift Valley fever	RVF	*Bunyaviridae*	*Phlebovirus*	Rift Valley fever	Cattle, Sheep, Goats, Humans	Mosquitoes *Ae. macintoshi*, *Culex* spp.	F, H, N	Africa	Major outbreaks every 10—15 years
Rocio	ROC	*Flaviviridae*	*Flavivirus*	Ntaya	Birds, Humans	Mosquitoes	F, N	South America	
Ross River	RR	*Togaviridae*	*Alphavirus*	Semliki Forest	Marsupials, Humans	Mosquitoes *Aedes* and *Culex* spp.	F, A	Australia, South Pacific	
Russian spring-summer encephalitis	RSSE	*Flaviviridae*	*Flavivirus*	Mammalian tick-borne flavivirus group	Rodents, Humans	Ticks *I. ricinus*	F, N	Europe, Western Asia	
Sandfly fever Naples	SFN	*Bunyaviridae*	*Phlebovirus*	Phlebotomus fever	Rodents?	Sand flies *Phlebotomus* spp.	F	Southern Europe India, North Africa	
Sandfly fever Sicilian	SFS	*Bunyaviridae*	*Phlebovirus*	Phlebotomus fever	Rodents?	Sand flies *Phlebotomus* spp.	F	Southern Europe India, North Africa	
Semliki Forest	SF	*Togaviridae*	*Alphavirus*	Semliki Forest	Rodents, Humans	Mosquitoes *Aedes* and *Culex* spp.	F, N	Africa, India-SE Asia	
Sindbis	SIN	*Togaviridae*	*Alphavirus*	WEE	Birds, Humans	Mosquitoes *Culex* spp.	F, A	Africa, Europe, Asia, Australia	
St. Louis encephalitis	SLE	*Flaviviridae*	*Flavivirus*	Japanese encephalitis	Passerine, Birds, Humans	*Culex* mosquitoes *Cx. pipiens* complex, *Cx. tarsalis*	F, N	North/South America	
Snowshoe hare	SSH	*Bunyaviridae*	*Orthobunyavirus*	California	Rabbits, Humans	Mosquitoes *Aedes* and *Culiseta* spp.	F, N	Northern US and southern Canada	Closely related to La Crosse virus
Tahyna	TAH	*Bunyaviridae*	*Orthobunyavirus*	California	Rabbits, Humans	Mosquitoes *Aedes* spp.	F, N	Europe, Asia, Africa	
Tick-borne encephalitis	TBE	*Flaviviridae*	*Flavivirus*	Mammalian tick-borne flavivirus group	Rodents, Humans	Ticks *I. ricinus*	F, N	Europe Western Asia	

Continued

TABLE A.1 Selected Arthropod-Borne Viruses (Arboviruses) (Viruses That Replicate in Arthropods and are Dependent on Arthropod Vectors for Transmission)—cont'd

Virus	Abbreviation	Family	Genus	Serogroup	Animals Affected	Principal Vectors	Disease[a]	Distribution	Comments
Toscana	TOS	*Bunyaviridae*	*Phlebovirus*	Phlebotomus fever	Rodents, Humans	Sand flies *Phlebotomus* spp.	F, N	Europe	
Venezuelan equine encephalitis	VEE	*Togaviridae*	*Alphavirus*	VEE	Rodents, Horses, Humans	Mosquitoes *Cx.* (*Melanoconion* spp.) and *Aedes* spp.	F, N	South/ Central America	Both epizootic and enzootic strains
Vesicular stomatitis Indiana	VSI	*Rhabdoviridae*	*Vesiculovirus*	VS	Mammals, Cattle, Swine, Horses, Humans	Sand flies, Black flies? *Lutzomyia* spp.	F, V	South/ Central/ North America	Can be confused with foot and mouth disease
Vesicular stomatitis New Jersey	VSNJ	*Rhabdoviridae*	*Vesiculovirus*	VS	Mammals, Cattle, Swine, Horses, Humans	Sand flies, Black flies *Lutzomyia* spp.	F, V	South/ Central/ North America	Can be confused with foot and mouth disease
Western equine encephalitis	WEE	*Togaviridae*	*Alphavirus*	WEE	Birds, Horses, Humans	*Culex* mosquitoes *Cx. tarsalis*	F, N	Western Canada through South America	
West Nile	WNV	*Flaviviridae*	*Flavivirus*	Japanese encephalitis	Birds, Horses, Humans	Many mosquito species primarily *Culex* (*Culex*) spp.	F, N	Worldwide	Spread to the New World in 1999
Yellow fever	YF	*Flaviviridae*	*Flavivirus*	Ungrouped	Primates, Humans	*Aedes* (*Stegomyia*) mosquitoes *Ae. aegypti, Ae. albopictus*	F, H	Africa South America	Formerly found in Central/North America
Zika virus	ZIK	*Flaviviridae*	*Flavivirus*	Spondweni	Primates, Humans	*Aedes* (*Stegomyia*) mosquitoes *Ae. aegypti, Ae. albopictus*	F, H, N	Africa, Asia South America	Spread to the New World in 2014

[a]*Disease:* A, *arthritis;* F, *fever;* H, *hemorrhagic;* N, *neurologic (i.e., meningoencephalitis);* V, *vesicular rash.*

TABLE A.2 Selected Arthropod-Transmitted Animal Viruses (Viruses that are Mechanically Transmitted by Arthropods, but that Do Not Replicate in the Arthropod Vector, i.e., Not an Arbovirus)

Virus	Abbreviation	Family	Genus	Animals Affected	Principal Vectors	Disease[a]	Distribution
Bovine leukemia	BL	*Retroviridae*	*Deltaretrovirus*	Cattle	Biting insects		Worldwide
Equine infectious anemia	EIA	*Retroviridae*	*Lentivirus*	Horses	Horse flies, Deer flies, Mosquitoes, Lice	F	Americas, Europe, Middle East, Africa
Hog Cholera (classic swine fever)	CSF	*Flaviviridae*	*Pestivirus*	Swine	Deer flies, Stable flies	F, H	Europe, Africa South/Central America
Myxoma	MYX	*Poxviridae*	*Leporipoxvirus*	Rabbits	Mosquitoes, Fleas	V	Europe, Australia, The Americas
Swinepox	SWP	*Poxviridae*	*Suipoxvirus*	Swine	Hog louse, Mange mites	F, V	Worldwide

[a]*Disease:* F, *fever;* H, *hemorrhagic;* V, *vesicular rash.*

and Venezuelan equine encephalitis viruses, can cause outbreaks that can devastate livestock and domestic animal populations as well as cause severe disease in human populations.

Arboviruses and arthropod-transmitted animal viruses can be transmitted by members of a number of different arthropod groups, ranging from mosquitoes and sand flies to argasid and ixodid ticks. These include viruses that replicate in the arthropod vector as well as those that are merely mechanically transmitted by arthropods. Most of the arboviruses are biologically transmitted, that is, replicate in their arthropod host prior to being transmitted, by mosquitoes, sand flies, or ticks (Table A.1). In contrast, arthropod-transmitted viruses are transmitted mechanically, that is, without replication in the arthropod, by tabanids, mosquitoes, black flies, lice, and mange mites (Table A.2).

Although there have been reports that some of the hemorrhagic fever viruses, such as Hantaan virus (the causative agent of Korean hemorrhagic fever) and Sin Nombre virus (a causative agent of hantavirus pulmonary syndrome), have been transmitted by mites, these reports are generally unsubstantiated (Table A.3).

In order to consolidate information on arboviruses, the Subcommittee on Information Exchange (SIE) of the American Committee on Arthropod-borne Viruses published the *International Catalogue of Arboviruses Including Certain Other Viruses of Vertebrates* in 1967. A second edition was published in 1975 and a third edition in 1985. The catalog is now accessible on-line in electronic format and is maintained by the Centers for Disease Control and Prevention (Fort Collins, Colorado, USA). It can be accessed at https://wwwn.cdc.gov/arbocat/VirusBrowser.aspx. The number of viruses listed in the catalogue has continued to increase. Whereas the original 1967 edition listed 204 viruses, the second edition in 1975 and the third edition in 1985 listed 359 and 504 viruses, respectively, and the online version listed more than 520 viruses as of 2017.

The *International Catalogue of Arboviruses* includes viruses believed to be arboviruses as well as those "thought to be of interest to arbovirologists," such as Hantaan, Ebola, Marburg, and Lassa viruses. These latter viruses are now believed to be rodent- or bat-associated, rather than transmitted by arthropods and are therefore listed separately in Table A.3.

When a potentially new arbovirus is discovered, information about its structure (i.e., enveloped or not, RNA single or double stranded, RNA segmented or not, etc.), relationship with other known arboviruses, and isolation history is submitted to two subcommittees of the ACAV: the Subcommittee on Inter-Relationships Among Catalogued Arboviruses (SIRACA) and the Subcommittee on the Evaluation of Arthropod-borne Status (SEAS). Based on the findings of the SIRACA and SEAS, the SIE decides if the virus should be included as a new virus in the catalog. If so, a decision is then made as to whether it should be designated as an arbovirus, a rodent-associated virus, or a virus for which additional information is needed before the SEAS can make a final decision.

TABLE A.3 Selected Hemorrhagic Fever Viruses Found in the Catalog of Arboviruses (Now Believed to be Rodent- or Bat-Associated Viruses rather than Arboviruses)

Virus	Abbreviation	Family	Genus	Serogroup	Animals Affected	Principal Vectors	Disease[a]	Distribution	Comments
Ebola	EBO	*Filoviridae*	*Filovirus*	Ungrouped	Rodents?, Bats?, Humans, Primates	None	F, H	Africa	Very high case-fatality rate
Hantaan	HTN	*Bunyaviridae*	*Hantavirus*	Ungrouped	Rodents, Humans	None	F, H	Asia	Korean hemorrhagic fever
Junin	JUN	*Arenaviridae*	*Arenavirus*	Ungrouped	Rodents, Humans	None	F, H	Argentina	Argentinean hemorrhagic fever
Machupo	MAC	*Arenaviridae*	*Arenavirus*	Ungrouped	Rodents, Humans	None	F, H	Bolivia	Bolivian hemorrhagic fever
Marburg	MBG	*Filoviridae*	*Filovirus*	Ungrouped	Humans	None	F, H	Africa	Very high case-fatality rate
Prospect Hill	PH	*Bunyaviridae*	*Hantavirus*	Ungrouped	Rodents	None	?	Eastern US	
Puumala	PUU	*Bunyaviridae*	*Hantavirus*	Ungrouped	Rodents, Humans	None	F, H	Europe	Nephropathia epidemica
Seoul	SEO	*Bunyaviridae*	*Hantavirus*	Ungrouped	Rodents, Humans	None	?	Worldwide	Possible cause of kidney disease/hypertension
Sin nombre	SN	*Bunyaviridae*	*Hantavirus*	Ungrouped	Rodents, Humans, *P. maniculatus*	None	F, R	North America	Caused an outbreak in southwestern US in 1993

[a]*Disease:* F, *fever;* H, *hemorrhagic;* R, *respiratory.*

REFERENCES AND FURTHER READING

Berge, T. O. (Ed.). (1975). *International catalogue of arboviruses including certain other viruses of vertebrates* (2nd ed.). U.S. Department of Health, Education, and Welfare, DHEW Publication No. (CDC) 75−8301.

Fauquert, C. M., Mayo, M. A., Maniloff, J., Desselberger, U., & Ball, L. A., (Eds.). (2005). *Virus taxonomy. 8th report of the International Committee on Taxonomy of Viruses*. New York: Academic Press. 1162 p.

International catalog of arboviruses including certain other viruses of vertebrates. https://wwwn.cdc.gov/arbocat/VirusBrowser.aspx.

Karabatsos, N. (Ed.). (1985). *International catalogue of arboviruses including certain other viruses of vertebrates* (3rd ed.). San Antonio, TX: American Society of Tropical Medicine and Hygiene.

Monath, T. P. (Ed.). (1988). *The arboviruses: epidemiology and ecology* (Vols. 1−5). Boca Raton, FL: CRC Press.

Moore, C. G., McLean, R. G., Mitchell, C. J., Nasci, R. S., Tsai, T. F., Calisher, C. H., et al. (1993). *Guidelines for arbovirus surveillance in the United States*. Fort Collins, Colorado, Centers for Disease Control and Prevention. U.S. Dept. of Health and Human Services.

Taylor, R. M. (Ed.). (1967). *The catalog of arthropod-borne and selected vertebrate viruses of the World*. Washington, DC: U.S. Public Health Service Publication No. 1760, U.S. Government Printing Office.

This page intentionally left blank

Glossary

The following definitions are provided for words and terms used in this book. They are intended to help the reader understand terminology with which they may not be familiar, given the breadth of interests and professions of the intended readers.

Abamectin An analogue of the antiparasitic group of compounds called avermectins.

Abatement A reduction in the amount or degree; e.g., abatement of mosquito populations, or an abatement district or program.

Abrade (-ed, -sion) To wear away by scraping, rubbing, or friction; e.g., skin abrasion.

Abscess Localized accumulation of pus, typically with associated inflammation.

Abscond (-ing) Behavior in bees in which an entire colony abandons an established hive to seek another suitable nest site.

Absolute The actual, or real, numerical value of something; as opposed to a relative number or value; e.g., absolute abundance.

Acalyptrate Lacking a calypter; e.g., certain muscid flies; sometimes spelled acalypterate.

Acariasis An infestation with mites; also any disease or other medical condition caused by mites.

Acaricide (-al) A compound or substance that kills mites.

Acarine Referring to mites (Acari).

Acarology The science or study of mites (Acari).

Acarophobia Abnormal fear of mites and ticks; also used in medical community with reference to a delusion that the skin is infested with mites (not to be confused with acrophobia, a fear of heights).

Acetylcholine A chemical, an ester of choline, which plays a role in transmission of nerve impulses at synapses and myoneural junctions.

Acne cosmetica An acne condition resulting from repeated applications of cosmetics; usually mild and noninflammatory.

Acquired immunity (Acquired resistance) Resistance resulting from previous exposure of the individual in question to an infectious agent or antigen.

Action threshold The level or magnitude at which action should be, or is, initiated, e.g., when mosquito population densities reach a particular level (action threshold) such that control measures should be implemented.

Aculeate Insects in the order Hymenoptera that possess a stinger; wasps, bees and ants.

Acute (1) Sharp or severe; sudden onset and usually of short duration; as opposed to chronic; e.g., acute symptoms or illness. (2) a morphological description of a structure that forms a point.

Adenolymphangitis Inflammation of lymphatic glands and vessels.

Adenopathy Swelling or unusual enlargement of lymph nodes.

Adenosine phosphate A compound of adenosine (nucleotide containing adenine and ribose) and one or more phosphoric acid groups; a hydrolytic product of nucleic acids.

Adenosine triphosphate A compound of adenosine (nucleotide containing adenine and ribose) and three phosphoric acid groups; energy of muscle is stored in this compound; abbrev. ATP.

Adenotrophic (viviparity) A form of viviparity in which larvae are fully nourished within the parent female by maternal glands and are larviposited immediately prior to pupation; occurs in some Diptera such as tsetse flies and some members of the Hippoboscoidea.

Adrenaline (*See* Epinephrine).

Adrenergic blocking agent A compound that interferes with the synaptic release of epinephrine (adrenaline) when nerve fibers are stimulated.

Adulticide A chemical compound or substance that kills the adults; e.g., insects or mites.

Aedeagus The intromittent organ of male arthropods, used to transfer sperm to female.

Aedine Taxonomic term that refers to mosquitoes of the subfamily Aedinae (e.g., genus *Aedes*).

Aerial treatment Application of insecticides, or other pesticides, from the air, typically by fixed-wing airplanes or helicopter; e.g., for mosquito control.

Aerial In the air, as in flying insects; above the ground, e.g., aerial nest of yellowjackets, in trees; see *aerial treatment*.

Aerobic Living only where oxygen is present; e.g., aerobic bacteria.

Aeropyle A minute respiratory opening in the eggs of some insects; e.g., lice.

Aerosol (-ized) A suspension of fine particles in air or other gases.

Aerosol transmission The transfer of an infectious agent, or aerosolized particles, from one individual to another via air; e.g., by sneezing or coughing

Aestivate (ion) To pass the summer, summer months, or other unfavorable periods (except winter) in a dormant state.

Afebrile Without fever.

Afrotropical region Biogeographic area that includes Africa south of the Sahara and the southwestern part of Arabia; also called Ethiopian region.

Age grading (See *physiological age grading*) a means, or method, for determining the physiological age of an organism; e.g., the reproductive status of mosquitoes and other insects.

Aggregation pheromone A chemical substance produced by an organism that causes individuals of the same species to gather together, or aggregate; e.g., cockroaches and some ticks.

Agonistic Aggressive or threatening, as in agonistic behavior.

Ague A term originally used to indicate fever and chills; formerly with particular reference to malaria (pronounced ā gu).

Alakurt An adult flea belonging to the family Vermipsyllidae, and the genera *Vermipsylla* or *Dorcadia*.

Albendazole A benzimidazole compound used to treat various roundworm and tapeworm infestations; e.g., cases of lymphatic filariasis.

Aliphatic compound An organic compound with carbon atoms joined together in a straight, or branched, open chain rather than a ring; e.g., hydrocarbons, such as alkanes and alkenes.

Alkaloid An alkaline compound in plants that is typically bitter and has a toxic effect on other organisms; e.g., quinine, nicotine, and morphine.

Allantoin A product of purine metabolism that promotes wound healing; e.g., a natural antibiotic produced by certain blow-fly larvae.

Allergen Any substance that induces an allergic reaction; in addition, to proteins and antigens, includes dusts, fungi, pollen, food components, drugs, etc.

Allergic dermatitis Sensitivity reaction of the skin to an allergen.

Allergic reaction A physiological or dermatological response to an allergen.

Allergy A reaction of body tissues of sensitized individuals to a specific substance, or allergen.

Allopatric Occurring in separate geographic areas; usually with reference to the distribution of species.

Alloscutum The dorsal surface of the body (idiosoma) of mites and ticks posterior to the scutum, or dorsal plate.

Alopecia Loss of hair.

Alphavirus An arbovirus belonging to the family *Togaviridae*, genus *Alphavirus*.

Alveolar collapse Compression of the air cells, or alveoli, in the lungs of vertebrates.

Amastigote A cell lacking a flagellum; used to describe a developmental stage of trypanosome protozoans; e.g., *Leishmania* and *Trypanosoma* spp.

Ambient Surrounding, as in ambient temperature or other environmental conditions.

Ambulacrum (-al) The pretarsus, or terminal structures of the tarsus, in mites and ticks; consists of the empodium, pulvillus, and/or tarsal claws, which can be highly modified or reduced.

Ametabolous A type of development in wingless insects in which there is little distinction between the immature stages and adults, other than size and reproductive function; immatures often called juveniles; e.g., springtails or collembolans.

Amplification cycle The sequence of organisms involving arthropods and their vertebrate hosts (reservoirs) by which a pathogen multiplies and is spread from one organism to another.

Amplification The increase of a pathogen in an ecological setting, which can involve the numbers of arthropod vectors and/or hosts infected and the levels of infection; used particularly with reference to viruses.

Amplifying host (amplifier host) A host animal, typically a vertebrate, in which a pathogen multiplies, thereby increasing the potential level of infectivity to competent arthropod vectors.

Ampulla A dilatation or saclike enlargement of a duct or canal; also used to refer to a bulbous enlargement of a structure; e.g., basal portion of the sting at the tip of the tail of scorpions.

Anal groove A curved groove that partially surrounds the anal area, anteriorly and laterally, in ticks of the genus *Ixodes*, but is posterior to the anus in other ixodid (hard) ticks.

Anal papilla (-ae) Thin-walled projections of cuticle at the caudal end of some insects, which typically serve an osmoregulatory and/or respiratory function; e.g., larvae of mosquitoes and other aquatic insects.

Anal wing vein (s) Longitudinal, unbranched vein that extends from the base of the insect wing to the outer margin of the wing, below the cubitus.

Analgesic A medication or chemical that alleviates pain (sometimes secreted by blood-feeding arthropods).

Anaphylactic shock A physiological state of shock (severely depressed vital signs) induced by injection or ingestion of a substance to which the individual has become sensitized (see *anaphylaxis*).

Anaphylatoxin A substance that can trigger anaphylaxis; typically involves release of histamine and other compounds associated with a hypersensitivity reaction.

Anaphylaxis (-tic) A hypersensitivity reaction in which the body responds severely to an injected or ingested protein to which it has been previously exposed (e.g., reaction to bee or wasp sting) an acute antigen-antibody response (see *anaphylactic shock*).

Anaplasmosis A tick-borne disease of cattle, sheep and other ruminants caused by rickettsial bacteria in the genus *Anaplasma* (e.g., *A. marginale*); often characterized by anemia and jaundice.

Anautogeny (-ous) The inability of females of hematophagous arthropods to produce eggs without taking a bloodmeal (see *autogeny*).

Anemia (-ic) A deficiency in the oxygen-carrying capacity of the blood due to reduced levels of hemoglobin, typically caused by too few red blood cells.

Angiomatosis A medical condition characterized by multiple angiomas, i.e., typically benign lesions or tumors involving lymphatic and blood vessels; can be caused by pathogens such as *Bartonella* spp.

Annulate Formed of ringlike segments, or bands representing a secondarily divided segment.

Annulus (-i) A ringlike structure encircling a joint, segment, or other structure; e.g., ring sclerite surrounding the base of an antenna, or annulate terminal part of the antenna, as in horseflies.

Anoplocephalid Referring to taenioid tapeworms in the family Anoplocephalidae.

Anorexia An eating disorder, with loss of appetite or inability to eat, with associated loss of body mass.

Antepygidial Located immediately anterior to the pygidium, or dorsal aspect of the last abdominal segment.

Anterior-station transmission The transfer of a pathogen or parasite from one individual to another via the mouth or mouthparts.

Anthomyiid Refers to members of the dipteran family Anthomyiidae, calyptrate muscoid flies.

Anthroponosis (-es) A disease involving only humans.

Anthropophagic Feeding on humans; e.g., anthropophagic insects and other arthropods.

Anthropozoonosis (-es) An arthropod-borne disease that is transmitted primarily among wild animals (zoonotic disease) but for which humans can become incidentally infected with the associated pathogen or parasite; e.g., East African trypanosomiasis.

Antibody Any of numerous proteins (immunoglobulins) produced in response to specific foreign antigens, including viruses, bacteria,

and protozoans, and capable of reacting specifically with that antigen; an integral part of a body's immune system.

Anticoagulant A substance that prevents, or interferes with, clotting of the blood.

Anticoagulin An anticoagulant, usually referring to substances produced by organisms other than humans that interfere with normal clotting of vertebrate blood; e.g., proteins in saliva of blood-sucking arthropods.

Antiedema Counteracting or reducing the amount of fluids that accumulate in body tissues.

Antigen A substance that can stimulate the production of antibodies.

Antigenic complex A composite of different antigenic structures (e.g., molecule, cell, virus, bacterium) with two or more different antigenic groups and specificities.

Antihistamine A compound that counteracts the effect of histamine.

Antimalarial A drug used to prevent or cure cases of malaria; e.g., chloroquine; also used as an adjective, as in antimalarial agent or drug.

Antithrombin A substance that inhibits clotting of blood by preventing reaction between thrombin and fibrinogen.

Antivenin A substance that counteracts the effects of venom from bites and stings of insects and other organisms; animal serum that contains antivenins, used to treat cases of envenomation; also called antivenom.

Apex The most distal portion of a structure, or farthest from the base; e.g., tip of the insect wing, furthest point from the wing base.

Aphrodisiac A substance or agent that causes sexual arousal or desire.

Apical droplet Small drop of fluid excreted via the anus or specialized glands at the tip of the insect abdomen.

Apocrine gland A coiled, tubular gland that secretes products at the apical end; the latter is, then pinched off as a secretion, including cellular and cytoplasmic components; e.g., apocrine sweat glands.

Apodeme A hollow invagination of the body wall in arthropods that serves primarily as an attachment site for muscle.

Apolysis The separation of old cuticle from the underlying, newly formed epidermis in the initial stages of molting in arthropods.

Apophysis Solid projections of the body wall of arthropods, both internal and external; in addition, to sites for muscle attachment, serve to strengthen the body wall and as both support and protection for various organs.

Aposematic Having a conspicuous structure or color pattern that advertises oneself as a means of defense from potential enemies; e.g., the black-and-yellow warning coloration of some wasps and bees.

Apterous Lacking wings.

Apyrase Any of several enzymes that catalyze the hydrolysis of adenosine triphosphate (ATP), releasing phosphate and energy.

Arachnida A large class of arthropods characterized by two body regions and four pairs of jointed legs, with no wings or antennae; includes spiders, mites, ticks, scorpions, and other arachnids.

Arachnology The science and study of arachnids.

Arachnophobia An abnormal, morbid, or pathological fear of arachnids, most commonly involving spiders.

Araneism A medical condition resulting from the bite of a spider; also called arachnidism, a more general term referring to envenomation by spiders, scorpions, and other arachnids.

Araneomorph Spiders in the suborder Araneomorphae, characterized by opposable chelicerae that move in a transverse plane; as distinguished from tarantulas and their relatives in which the fangs move vertically.

Araneophobia An abnormal fear of spiders (see *arachnophobia*).

Arbovirus A shortened name for arthropod-borne virus, i.e., a virus transmitted by arthropods.

Argasid A member of the tick family Argasidae, or soft ticks.

Arista (-ate) A bristle-like process on the terminal segment of the antennae of adults of certain higher Diptera; e.g., muscoid flies.

Arolium (-a) A median lobe, or cushion-like pad, between the bases of the tarsal claws in insects.

Arrhenotoky Parthenogenesis in which only males are produced, from unfertilized eggs.

Arrhythmia An abnormal rhythm or disturbance of the heartbeat.

Arsenical A preparation or drug containing arsenic.

Arteriole The smaller terminal branches of an artery, particularly those that connect with capillaries.

Arthralgia (-ic) Joint pain.

Arthritis an acute or chronic inflammation of a joint, typically accompanied by pain, swelling and stiffness.

Arthropod Invertebrates of the phylum Arthropoda, possessing a segmented body, jointed appendages, and a chitinous exoskeleton; includes insects and arachnids.

Arthropod-borne virus A virus transmitted by arthropods.

Artiodactyla An order of hoofed mammals with an even number of functional toes on each foot; includes cattle, deer, sheep, goats, and pigs.

Ascites An excessive accumulation of serous fluids in the peritoneal cavity.

Aseptic meningitis A mild form of meningitis characterized by fever, headache, and stiff neck; usually caused by a virus.

Aseptic Free of pathogenic microorganisms or their toxins.

Asexual Lacking evidence of sex organs; involving reproduction without the union of male and female gametes.

Asilid A member of the dipteran family Asilidae, or robber flies.

Asthma (-tic) A chronic respiratory disorder, often caused by allergies; characterized by recurrent bronchial spasms, coughing, wheezing, and constriction in the chest.

Asthmatic bronchitis Inflammation of the mucous membrane lining the bronchial tubes in individuals with asthma.

Asymptomatic Not showing symptoms.

Atopic Relating to certain allergic conditions of a hereditary nature; e.g., atopic dermatitis, eczema, and asthma.

Atrophy A deterioration or reduction in size of tissues or organs of the body as the result of injury, disease, or lack of use.

Attenuate (-ed) To reduce or weaken in strength, amount, or virulence; e.g., attenuated virus.

Attractant A substance that lures insects or other animals to an animal or trapping device; can include pheromones and chemical compounds involved in host-seeking behavior.

Augmentative release Repeated releases of commercially reared biological control agents in the form of predators or parasites for the control of pest species.

Aural Relating to the ear or sense of hearing.

Australasian The region of the southern Pacific Ocean that includes Australia, New Zealand, New Guinea and neighboring islands.

Autoantibody An antibody that acts against tissues, cells, or cell components of the organism that produces it.

Autogeny (-ous) The production and development of eggs without a bloodmeal in insects and other arthropods that usually require blood in order to do so; sufficient protein reserves are carried over from the larval and pupal stages to support one or more gonotrophic cycles.

Autolysis Autodigestion resulting from enzymatic digestion of cells, especially when these are degenerate or dead; especially prevalent in cadavers.

Autonomic Occurring involuntarily or spontaneously, independent of external stimuli; e.g., autonomic nervous system.

Avermectin A group of antiparasitic drugs whose analogues include ivermectin and abamectin.

Avirulent Lacking virulence; nonpathogenic.

Axillary Pertaining to the axilla, or arm pit.

Babesiosis (-es) Any of several tick-borne diseases of humans, livestock, and wild animals caused by blood protozoans of the genus *Babesia.*

Bacillus (-i) Rod-shaped, aerobic bacteria of the genus *Bacillus*, or of other numerous genera.

Back rubber A self-treatment device for livestock in which a padded rope saturated with an insecticide is suspended between two vertical posts through which animals regularly walk; insecticide is applied to the head and upper body by direct contact as the animal passes beneath it.

Bacteremia Presence of bacteria in the blood.

Bacteriome An area in or near the gut of some blood-feeding arthropods containing symbiotic microorganisms (typically bacteria).

Bacteriophage A virus that infects and destroys bacterial cells.

Bacteriostatic A chemical or biological agent that inhibits bacterial growth.

Bagasse Fibrous residue from the process of extracting juice from crushed stalks of sugarcane and other plants; used as a source of cellulose for making paper products.

Bait A food, or other attractive substance, containing, or associated with, a toxicant used to lure insects and other pests as a means of control or extermination.

Bait station A device containing a bait used to attract and kill insects; e.g., ants and cockroaches; also used for attracting rodents and other animals, bringing the latter in contact with insecticides or acaricides that kill certain ectoparasites.

Bandicoot (1) Rat-like marsupials of the family Peramelidae in Australia, Tasmania, New Guinea and adjacent islands. (2) any of several large rats in the family Muridae of the genera *Bandicota* and *Nesokia* of southeast Asia.

Bartonellosis A disease of humans and other mammals caused by bacteria of the genus *Bartonella.*

Basalar flight muscles Muscles in insects that attach to sclerotized plates, or basalar sclerite(s), in the membranous area at the base of the wings; play an important role in flight.

Basis capituli The fused proximal elements of the mouthparts and palps that form the base of the gnathosoma, or capitulum, in ticks.

Basophil A cell, especially white blood cells, with granules that readily stain with methylene blue and other basic dyes.

Bed net A fine-mesh fabric hung tentlike over, or otherwise enclosing, a bed to protect sleeping individuals from biting insects, notably, mosquitoes.

Benign Mild; not progressive or malignant.

Benthic Pertaining to the substrate or community of organisms at the bottom of a body of water, such as a lake or ocean.

Benznidazole An antiparasitic drug containing benzonidazol, used to treat cases of Chagas disease.

Berlese funnel A device used to extract insects and other arthropods from soil, ground litter, and other substrates; consists of a funnel over which is suspended an electric light bulb; the light and heat from the bulb slowly dries the substrate, driving the arthropods down the funnel and into a collecting jar, usually containing alcohol.

Bifid Divided into two parts; e.g., bifid claw (see *bifurcate*).

Bifurcate (-tion) Divided into two parts or branches; forked or cleft.

Bilirubin The orange or yellow pigment in bile, produced by the degradation of heme in red blood cells; excessive titers in the blood can impart yellowish color to skin (jaundice).

Bimodal Having two distinct statistical modes.

Binary fission A form of asexual reproduction in which a parent cell divides into two approximately equal parts.

Biogenic Produced by living organisms or biological processes.

Biological control agent A biological organism (e.g., virus, bacterium, fungus, parasitoid) used to reduce the population density of a pest species.

Biological vector A vector in which biological development (cyclodevelopment) of the vector-borne pathogen/parasite under consideration occurs (as opposed to a mechanical vector in which no biological development occurs).

Biopsy Removal of a piece of tissue for microscopic examination or testing.

Biotic potential The ability of an organism to reproduce optimally.

Biovar A term sometimes applied to different strains of certain bacteria, such as those that cause plague (*Yersinia pestis*) or tularemia (*Francisella tularensis*).

Bivoltine Having two generations per year.

Blepharitis Inflammation of the eyelids, involving the hair follicles and glands; characterized by redness, swelling, and crusty exudates.

Blister A local elevation of the skin containing serum or other watery fluid.

Blood meal Blood taken into the midgut of a hematophagous arthropod at a given feeding.

Blood poisoning Septicemia; a systemic disorder caused by pathogenic organisms or their toxins in the blood; characterized by fever, chills, and prostration.

Bolus A soft mass of ingested food in the alimentary tract; a large pill containing a drug, insecticide, parasiticide, or other compound fed to animals, or otherwise inserted in the alimentary tract.

Boric acid A white or colorless crystalline compound of boron and oxygen (H_3BO_3) prepared from borax; when ingested by cockroaches and other insects, damages epithelial cells of the digestive tract, interfering with nutrient absorption.

Borreliacide (-al) A drug or other compound that kills spirochetes of the genus *Borrelia.*

Borrow pit An excavation from which sand or gravel is moved to use as fill at another location, typically a construction site.

Bot fly Adults of flies in the family Oestridae.

Bot Grublike parasitic larvae of bot flies, family Oestridae.

Botanical A drug, insecticide, or other compound derived from plants.

Bovid A member of the family Bovidae; hoofed, hollow-horned runinants that include cattle, sheep, goats, buffaloes, and antelopes.

Bovine A member of, or pertaining to, the genus *Bos* (cattle, buffaloes, kudus), subfamily Bovinae.

Brachyceran A member, or pertaining to, the dipteran suborder Brachycera.

Brachypterous Possessing very short or rudimentary wings.

Bradykinin A polypeptide that mediates an inflammatory response; causes dilation and increased permeability of peripheral blood vessels, and contraction of smooth muscle.

Bridge vector A vector that transmits a pathogen or parasite from an enzootic cycle to humans or other animals that are often dead-end hosts.

Brisket The breast, or chest, of a quadruped animal.

Bronchiole (-ar) Small, thin-walled, noncartilaginous branches of the bronchi, the two large branches of the trachea.

Bronchitis Inflammation of the bronchial mucous membranes.

Bronchopneumonia Inflammation of the terminal bronchioles and alveoli in the lungs.

Bronchoprovocation Inhalation of an aerosolized chemical to induce a hypersensitivity response as a means of identifying obstructed airway passages in the lungs of asthmatic patients; also called bronchial challenge and broncho provocation.

Brucellosis An infectious disease of cattle, sheep, goats, and some rodents caused by bacteria of the genus *Brucella.*

Bti Abbreviation for the bacterium *Bacillus thuringiensis israelensis.*

Bubo An inflamed swollen or enlarged lymph node, typically in the axilla or groin; as in the bubonic form of plague.

Bug In an entomological context, members of the Order Hemiptera, possessing piercing-sucking mouthparts; e.g., bed bugs and triatomine bugs; used colloquially to imply any kind of arthropod.

Bulbous dermatitis Inflammation of the skin, characterized by bulb-shaped lesion(s).

Bulla (-ae) A large blister or dermal vesicle filled with fluid.

Bunch (-ing) (p. 296) A behavioral response of livestock to the presence of biting or pestiferous flies in which the animals cluster tightly together to minimize the exposure of body surfaces to attack.

Bunyavirus A genus of singled-stranded RNA viruses (mostly arboviruses) in the family Bunyaviridae.

Bursa (-ae) A pouch or saclike cavity, often filled with fluid; e.g., in the vicinity of vertebrate joints where it reduces friction associated with movement of tendons, ligaments and bones.

Cadaver A dead and decaying human body.

Caecum (-a) (cecum) A blind pouchlike extension of the alimentary tract.

Calabash Any of several gourd-bearing plants, including the annual vine *Lagenaria siceraria*, or bottle gourd, and the tropical American tree *Crescentia cujete.*

Calamistrum A row of spines on the metatarsus of cribellate spiders, used to comb tiny strands of silk as they are extruded from the cribellum.

Calamus The hollow basal part of a bird feather; quill.

Calcium gluconate A white water-soluble compound ($CaC_{12}H_{22}O_{14}$), used as a dietary supplement of calcium; in solution is used to counteract the action of black widow venom.

Calliphorid A member of the fly family Calliphoridae, or blow flies.

Callus (-i) A localized thickening and enlargement of the horny layer of skin (vertebrates) or cuticle (arthropods); a callosity.

Calypter (-ate) An enlarged membranous structure (alula or squama) at the base of the dipteran wing, which covers the haltere in some muscoid flies (Calypteratae).

Calytrope Specialized defensive setae in caterpillars that readily break off on contact, causing irritation to skin.

Calyx A cup into which certain structures are set; e.g., egg-calyx in insects, the enlarged portion of the oviduct at the opening of the ovarioles in insects, into which eggs are received before entering the vagina.

Camerostome In soft ticks (family Argasidae), the ventral depression, or cavity, in which the mouthparts (capitulum) are situated; more difficult to discern in engorged specimens.

Campestral Pertaining to fields and open country.

Campodeiform A larval form of insects which, at least in their earlier instars, resembles diplurans; includes larvae of Neuroptera, Trichoptera, Strepsiptera, and some Coleoptera.

Canid A member of the family Canidae; includes dogs, coyotes, wolves, foxes, and jackals.

Canopy trap A tentlike structure elevated above the ground and used to capture flying insects; typically uses a large black ball, or other visual object, to attract certain biting flies (e.g., tabanids).

Canthariasis Invasion of humans and other animals by beetles, as larvae or adults.

Capillary feeder An organism that feeds directly on blood by piercing a capillary with its stylet-like mouthparts.

Capitulum The anterior-most body region of mites and ticks composed of the mouthparts and palps, and their fused bases; gnathosoma.

Capuchin Long-tailed monkeys of the genus *Cebus*, family Cebidae, in Central and South America; with a tuft of hair on the head suggesting a monk's cowl, or hood.

Carapace The sclerotized dorsal portion of the prosoma in some arachnids, e.g., spiders and scorpions.

Caravansary (-ies) A large hotel or inn in central and western Asia, with a courtyard to accommodate caravans.

Carbamate A salt or ester of carbamic acid, with insecticidal properties

Cardiomegaly Enlargement of the heart.

Cardiomyopathy A disease or disorder of heart muscle, especially of unknown cause.

Cardiovascular collapse Retraction of the walls of the heart and blood vessels due to loss of blood, lowered blood pressure, anaphylactic reactions, and other causes.

Carrier A person or other animal that harbors an infectious agent but typically shows no apparent symptoms or signs of the disease, and which is capable of transmitting the pathogen to others.

Carrion Dead and decaying animal remains.

Carton Paper made by hymenopteran insects (e.g., social wasps), used to construct their nests.

Caste One of multiple forms in social insects that perform a specialized function in a colony; e.g., worker, soldier, and queen.

Cataract Opacity of the lens or capsule of the eye, causing impaired vision or blindness.

Catecholamine Any of a group of amines derived from catecol that act as neurotransmitters and hormones; e.g., epinephrine, norepinephrine, and dopamine.

Caudal At or near the posterior end of the body.

Causative agent A biological pathogen or parasite that causes a disease; also sometimes used when referring to a toxin.

Cell In insect morphology, an enclosed area of an insect wing between, or bounded by, veins.

Cell-mediated immunity The reaction to antigens by cells (e.g., T cells) rather than antibodies.

Cellular immune response (*See* cell-mediated immunity).

Cellular immunity (*See* cell-mediated immunity).

Cellulitis Inflammation of cells, especially of subcutaneous and connective tissues; characterized by redness, swelling, pain, and sometimes fever.

Cenozoic (caenozoic) The geologic era from 65 million years ago until the present time; sometimes called "the age of mammals."

Cephalic Pertaining or attached to the head; also, directed toward the head.

Cephalopharyngeal skeleton Internal sclerotized structures in the head region of muscoid fly larvae, serving as muscle attachments for the mouthparts and pharynx; of integumental origin and shed when larva molts.

Cephalothorax A body region in certain arthropods formed by fusion of the head and thorax; e.g., arachnids and crustaceans.

Ceratopogonid A member of the dipteran family Ceratopogonidae, or biting midges.

Cercus (-i) A sensory caudal appendage, usually paired, on the 10th abdominal segment of insects; usually slender or filamentous, but highly variable in form and length.

Cere (-es) A fleshy or waxlike swelling near the base of the upper beak in certain birds (e.g., parrots) through which the nostrils, or nares, open.

Cerebral Pertaining to the cerebrum, or largest part of the vertebrate brain; as in cerebral malaria.

Cerumen (-inal, -nous) Soft, yellow-to-brown, waxlike secretions in the external ear canal; earwax.

Cervid A member of the family Cervidae; includes deer, elk, and moose.

Cervix (-cal) With reference to insects, the "neck" region between the head and thorax.

Cestode A member of the class Cestoda; includes tapeworms.

Chaetotaxy Nomenclature, arrangement, or study of setae and bristles on the integument of insects and other invertebrates.

Chamois A goatlike antelope, *Rupicapra rupicapra*, in the mountainous regions of Europe.

Chapparal An ecological habitat dominated by small-leaved evergreen shrubs, with hot dry summers and cool moist winters; e.g., foothills of California.

Chela (-ae, -ate) A pincer-like structure at the tip of a limb in arthropods, such as arachnids and crustaceans, composed of a fixed and movable digit; e.g., clawlike palps of scorpions and pseudoscorpions and the chelicerae of many mites.

Chelicera (-ae) The pair of pincer-like or fanglike mouthparts in arachnids, variously modified for grasping or piercing.

Chemoprophylaxis (-actic) The use of chemical agents, drugs, or food supplements to prevent disease.

Chemosensillum (-a) A sense organ that responds to chemical agents or chemical cues.

Chigger The parasitic larval stage of mites in the family Trombiculidae.

Chironomid A member of the dipteran family Chironomidae, aquatic midges.

Chitin (-ous) A durable, semitransparent substance, made primarily of nitrogen-containing polysaccharide, that is the principal component of cuticle in arthropods

Chitin synthesis inhibitor A class of pesticides for which the mode of action is interference with the synthesis of chitin; disrupts normal development during formation of the new cuticle in the molting process.

Chloramphenicol An broad-spectrum antibiotic derived from the soil bacterium *Streptomyces venezuelae*, or produced synthetically.

Chlorpyrifos A broad-spectrum organophosphate insecticide, or acaricide, that acts as an acetylcholinesterase inhibitor.

Cholecystitis Inflammation of the gallbladder.

Choriomeningitis Cerebral meningitis with cellular infiltration of the meninges.

Chorion In arthropods, the membrane(s) surrounding the egg, secreted by follicle cells in the ovary; the outer surface is often sculptured, i.e., with a distinct surface pattern.

Chorionic sculpture (-ing) *See* chorion.

Chromatography (-ic) A chemical analysis in which a mixture of substances is separated into fractions by adsorption on a porous surface; e.g., paper chromatography, gel chromatography.

Chronic Persistent, or of long duration; versus acute.

Chrysalis (-ides) The developmental stage in butterflies between the larva and adult; i.e., the butterfly pupa.

Chukar A Eurasian partridge, *Alectoris chukar*; introduced as a game bird in western North America.

Chyluria The presence of fatty globules, or chyle, in the urine; can be caused by blockage of lymphatic vessels by parasites.

Cibarium (-al) A chamber or pocket anterior to the mouth in insects through which fluids are pumped, or otherwise drawn, into the food canal.

Cimicid A member of the insect family Cimicidae; e.g., bed bugs, bat bugs, and bird bugs.

Circadian rhythm A daily activity cycle in living organisms that follows a rhythmic pattern at intervals of approximately 24 h; may be physiological or behavioral.

Civet A catlike mammal of the family Viverridae; in Africa and Asia.

Cladistics An approach to classifying organisms taxonomically based on the branching of descendant lineages from a common ancestor.

Cladistic analysis The quantitative analysis of comparative data used to develop a taxonomic system representing phylogenetic relationships and the evolutionary history of groups of related organisms.

Clasper Variously modified lobes or extensions of abdominal segment 10 in male insects used to grasp the female during mating.

Clavate Clubbed, or thickened toward the tip; e.g., setae, antennae.

Clavus The oblong or triangular anal region of the forewings, or hemelytra (= hemi-elytra), in members of the insect order Hemiptera.

Clinical Based on direct observation of patients and treatment for diseases; e.g., clinical cases, signs and symptoms.

Clubbed *See* clavate.

Clypeus The front of the insect head between the frons and labrum, usually separated from the latter by a groove.

Coalesce To fuse or grow together; e.g., blood vessels, rash and other skin lesions.

Coarctate Referring to an insect pupa that is encased in a hardened cuticle of the next-to-last larval instar, or puparium.

Coccobacillus A microorganism, typically a bacterium that is spherical to ovoid in shape.

Cockle A wrinkle or pucker, as in a fabric; used to refer to blemishes and structural damage of processed sheep skin resulting from feeding by the sheep ked.

Coevolution Biological evolution of two interacting species, with each adapting to changes in the other; e.g., between a parasite and its host.

Colic Muscular spasms in any hollow or tubular organ, such as the intestine; severe abdominal pain caused by such spasms.

Collembolan A member of the insect order Collembola, or springtails.

Colostrum A yellowish secretion from the mammary glands of female mammals a few days before and after they give birth; rich in antibodies, other proteins, and minerals.

Colubrid snake A member of the family Colubridae, represented primarily by nonvenomous species including garter snakes, king snakes, and water snakes.

Columbiform Members of the avian order Columbiformes, pigeons and doves.

Coma A state of deep, often prolonged unconsciousness in which an individual does not respond to external stimuli; usually the result of injury or disease.

Comb scale A specialized spicule, or pointed spine, on the lateral aspect of the seventh abdominal segment of culicine and first-instar anopheline mosquito larvae; usually a patch of such spicules.

Commensal A symbiotic relationship between two species in which one is benefited and the other is generally unaffected.

Communal Organisms living, or performing an activity, together (in a group).

Companion animal A domesticated animal with which humans share a special bond or sentimental relationship; a pet, e.g., a cat, dog, horse.

Competent (host, reservoir) Providing favorable conditions for the survival and/or replication of a particular pathogen or parasite.

Complement A group of proteins in blood serum that act as part of the body's immune response; interact with antibodies and other chemical substances to destroy bacteria and other foreign cells.

Complement-fixing antibody An antibody that, in combination with an antigen and complement, inactivates the complement, interfering with the ability to destroy foreign cells.

Complex In classification, referring to a group of closely related organisms or interacting biologically active molecules; e.g., taxonomic complex, virus complex, antigenic complex.

Compound eye One of the pair of image-forming structures in many insects, and some crustaceans, composed of large numbers of individual light-sensitive units called ommatidia, visible on the eye surface as facets.

Concave Curved inward, like the inner surface of a sphere.

Confirmed (case) A clinically corroborated infection, infestation, or other condition of humans or animals.

Congenital Relating to a condition present at birth; may be hereditary or a result of environmental influences.

Congestion Accumulation of excessive fluid, e.g., blood or other fluids, in tissues or organs.

Congestive heart failure Inability of the heart to maintain adequate circulation of the blood, resulting in distension of the ventricles, congestions in the lungs, and edema in the lower extremities.

Conjunctiva Mucous membrane that lines the inner surface of the eyelid.

Conjunctivitis Inflammation of the conjunctiva of the eye.

Connective tissue Highly vascular tissue derived from embryonic mesoderm that supports and connects various structures of the body; includes elastic and collagenous fibers, mucosal and fat tissues, cartilage and bone.

Connexivum (-al) The lateral margin of the abdomen at the juncture of the dorsal and ventral sclerites in members of the insect order Hemiptera.

Conspecific Belonging to the same species.

Contact dermatitis Inflammation of the skin resulting from contact with an irritating substance or allergen.

Contact pheromone A pheromone that illicits a response after it is touched by a conspecific organism.

Cootie Colloquial term for the human body louse, *Pediculus humanus humanus*; in a less strict sense, also the human head louse, *Pediculus humanus capitis*, and pubic louse, *Pthirus pubis.*

Coprophagous (-y) Feeding on excrement, or dung.

Corbicula Pollen basket of bees; modification of the tibia of the hind leg, characterized by a smoothly concave surface with a fringe of hairs along the tibial margin.

Cornified Having undergone cornification, the conversion of squamous epithelial cells into horny materials, such as nails, hair, feather, or scales; keratinized.

Corolla In mosquito eggs deposited as a raft, notably, of the genera *Culex* and *Culiseta*; a frill-like collar surrounding the micropyle that helps to keep the egg vertical while floating on the water surface.

Coronary band A band of highly vascular tissue at the base of the hoof in horses, ruminants, and other hoofed animals that secretes the horny wall of the hoof.

Corticosteroid A steroid compound produced by the cortex of the adrenal gland.

Corvid A member of the avian family Corvidae; includes crows, ravens, jays, and magpies.

Cosmopolitan Occurring worldwide, or at least in many parts of the world.

Cospeciation As for coevolution but at the species level.

Costa The thickened anterior margin of the wing in insects, usually referring to the forewing.

Cowdriosis A tick-borne disease of cattle, sheep, and goats caused by the rickettsia *Ehrlichia ruminatium*; also called heartwater and veldt disease; *E. ruminatium* formerly in the genus *Cowdria*.

Coxa (ae) The basal-most segment of the leg in insects and other arthropods.

Coxal gland An excretory structure in ticks and other mites that opens via a duct on, or near, the coxa; serves primarily in osmoregulation or to excrete noncellular liquid components of a bloodmeal.

Coxal pore The opening of the duct of a coxal gland.

Crack-and-crevice Protected out-of-the-way places indoors that can harbor insect pests; usually used with regard to application of insecticides, e.g., crack-and-crevice treatments.

Crateriform Hollowed like a bowl or saucer; crater-like.

Creatinine kinase An enzyme in muscle, brain, and other vertebrate tissues that acts as a catalyst for the conversion of ADP and phosphocreatine into ATP and creatine.

Creeping welt Segmental, annular thickenings of the external abdominal wall of the larvae of certain aquatic or semiaquatic flies (e.g., tabanids) that facilitate locomotion; sometimes used to refer to spiny annular bands of maggots that serve a similar purpose.

Crepuscular Pertaining to the twilight hours at dusk and dawn.

Cretaceous period The geological period of the Mesozoic Era, 140 to 65 million years ago, that began with the development of flowering plants and modern insects and ended with the extinction of dinosaurs and many other life forms.

Cribellum (-ate) A transverse plate anterior to the spinnerets in certain spiders through which silk is extruded to produce hackled-band threads characteristic of cribellate spiders.

Crop An enlargement of the foregut, usually the posterior part of the esophagus, in which food is stored; also a diverticulum of the foregut in insects that feed blood and other fluids

Cross-infection The ability of a pathogen or parasite to infect related hosts

Cross-reactivity For immunological testing, indicates a positive reaction to a pathogen or parasite other than the one under consideration; usually cross-reactivity concerns relatively closely related pathogens, such as the spirochete bacteria that cause Lyme disease and syphilis.

Cryptic infection An infection that is difficult, or sometimes impossible, to detect.

Cryptic species Members of a species complex that are not morphologically distinguishable from one another; can be separated only by nonmorphological criteria such as genetic sequencing and life-history studies.

Ctenidium (-a) A row of short, stiff spines resembling the teeth of a comb, used to cling to hair and feathers; found on the head and thorax (rarely also on the apex of the head or on the abdomen) of some fleas and other ectoparasitic insects; e.g., bat bugs (family Polyctenidae) beetles (*Platypsyllus*), and bat flies (families Nycteribiidae and Streblidae).

Culicidology The science and study of mosquitoes.

Culicine Referring to members of the mosquito subfamily Culicinae; includes most mosquito genera other than *Anopheles* and *Toxorhynchites.*

Cuterebrine Referring to members of the subfamily Cuterebrinae, New World skin bots.

Cuticle The noncellular outer layer of the arthropod integument, composed primarily of chitin and protein.

Cuticular hydrocarbon A compound associated with the cuticle, consisting of only carbon and hydrogen.

Cyanosis (-tic) Discoloration of the skin due to reduced hemoglobin in blood; typically bluish, grayish or purplish.

Cyclical development See *cyclodevelopment.*

Cyclodevelopment (-al) The sequence of developmental stages in the life cycle of organisms that undergo metamorphosis; e.g., *Plasmodium* and filarial nematodes in arthropod hosts.

Cyclodiene An organic compound with a chlorinated methylene group bonded to two carbon atoms of a six-member carbon ring; active ingredient in insecticides such as Aldrin, chlordane, and dieldrin.

Cyclorrhapha (-an, -ous) A large group of flies, order Diptera, in the suborder Brachycera, generally characterized as adults by an arista on the third antennal segment; represents a monophyletic group within the infraorder Muscomorpha.

Cynomolgus monkey A primarily arboreal macaque (*Macaca fascicularis*) native to Southeast Asia; also called crab-eating macaque and long-tailed macaque.

Cyst In parasitiology, a structure formed by and enclosing organisms in a larval or dormant stage; a closed sac or pouch with a defined wall that contains fluid, semifluid or solid material.

Cysticercus (-i, -oid) The larva of certain tapeworms in invertebrates that consists of a fluid-filled sac with an invaginated scolex (or multiple scolices), in tissues of an intermediate host.

Cytokine Any of several regulatory proteins released by cells of the immune system in the presence of specific antigen that act as intercellular mediators in generating an immune response.

Cytology The study of structure and function of cells.

Cytopathology The study of cellular changes in diseases.

Cytopenia A deficiency, or lack, of cellular elements in circulating blood.

Cytotaxonomy A branch of taxonomy based on cellular structure and function, particularly the structure and number of chromosomes.

Cytotoxin A substance that has a toxic effect on cells.

Dasyurid Members of the marsupial family Dasyuridae found in Australia and New Guinea; includes banded anteaters, pouched mice, and the Tasmanian devil.

DDT The chlorinated hydrocarbon compound dichloro-diphenyl-trichloroethane, used as an insecticide; now banned for use in many countries including the USA, because of associated environmental problems.

Dead-end host An animal that harbors a pathogen or parasite but does not serve as a source of infection for transmission to another individual.

DEC The chemical compound diethyl carbamate; used to kill filarial nematodes.

DEET The compound diethyl toluamide, used as an insect, tick, and chigger repellent.

Defervescence Abatement of fever; falling of elevated body temperature.

Definitive host An animal in which a parasite develops to the adult stage and in which sexual reproduction of the parasite takes place.

Delayed (reaction, sensitivity) A nonimmediate immunological or skin response to antigens, including those injected by blood-feeding or skin-infesting arthropods.

Delusional parasitosis *See* delusory parasitosis.

Delusory parasitosis An emotional disorder involving the false belief by an individual that he or she is infested by a parasite or that live organisms, usually mites or insects, are crawling on their skin; also called Ekbom syndrome.

Demodicosis A condition resulting from infestation of animal tissues, usually hair follicles or glands, by mites of the genus *Demodex.*

Dentate Resembling a tooth.

Denticle A small toothlike projection.

Depilation The removal of hair from the body or animal skins.

Dermatitis Inflammation of the skin; usually with itching and redness.

Dermatographia A condition in which lightly touching or scratching the skin causes raised reddish marks; also a form of urticaria in which pressure to the skin produces wheals.

Dermatomycosis An infection of the skin caused by certain fungi; e.g., ringworm.

Dermatophytosis A fungal infection of the skin, especially moist skin covered by clothing; e.g., athlete's foot, eczema, and ringworm.

Dermatosis (-es) Any disease of the skin, especially without inflammation.

Dermestid A beetle in the family Dermestidae, or skin beetles (also includes museum beetles and carpet beetles).

Desensitization The process of reducing sensitivity or making an individual nonreactive to an antigen or allergen.

Desiccant A substance that absorbs water and is used as a drying agent; e.g., silica gel and calcium oxide.

Desquamate To shed epithelial cells.

Detritivore An animal that feeds on detritus.

Detritus Partially decomposed, particulate organic matter.

Deutonymph The second of three nymphal stages in the basic life cycle of mites.

Dewlap A fold of loose skin hanging from the throat or neck; e.g., in bovines, and the wattle of certain birds.

Diabetes mellitus A carbohydrate-metabolism disorder resulting from inadequate production or utilization of insulin; characterized by elevated glucose levels in the blood and urine.

Diapause A physiological state of quiescence characterized by reduced metabolic activity without growth or development; may be seasonal or when environmental conditions are unfavorable.

Diatomaceous earth A light-colored porous soil or powder with a high silica content, made from the dried cell walls of diatoms; used as an abrasive, adsorbent, filtering, and insect-killing agent.

Diazinon An organophoshate compound used as an insecticide.

Dichoptic In Diptera, having compound eyes noticeably separated from one another along their midline; *see* holoptic.

Diel periodicity Occurring at a specific time each day, or at intervals of c. 24 h.

Diel Pertaining to a 24-h period; e.g., a daily cycle or activity pattern.

Dieldrin A chlorinated hydrocarbon used as an insecticide.

Die-off A sudden, sharp decline in a population or community as a result of a natural cause, such as disease or extreme weather conditions.

Diethylstilbestrol A synthetic, nonsteroidal compound with estrogen-like properties, used in medical treatments and in feed for livestock and poultry.

Diflubenzuron The chemical benzamide that acts as an insect growth regulator; interferes with the formation of insect cuticle and is used as an insecticide.

Dike An embankment or barrier of earth and rock built to prevent flooding or to retain water, e.g., for agricultural purposes; also, to construct such embankments.

Dilatation The condition of being stretched or expanded beyond the normal size, as in a tubular structure or orifice; e.g., in insects, a dilatation of the stalk of an ovariole where an egg follicle has degenerated.

Dimorphic Occurring in two distinct forms.

Dingo A wild dog, *Canis dingo*, of Australia; often treated as a subspecies, *Canis familiaris dingo*, of the domestic dog.

Diphyletic Derived or descended from two ancestral lines.

Diploid Having two sets of chromosomes, or twice the haploid number; in organisms that reproduce sexually, one pair of chromosomes from each parent.

Dipping vat A large tank of fluid containing an insecticide or acaricide, through which animals are forced to walk or swim as a means of treatment for livestock pests; e.g., ticks.

Disease agent An organism or substance that causes disease; usually refers to a pathogen or parasite; also called the causative agent.

Disease A pathological condition of the body characterized by a group of signs or symptoms.

Dispersal The movement of individuals away from a population center; as distinct from dispersion, the spatial distribution of individuals within a given geographic area.

Disseminate (-ed, -ation) To scatter or become distributed over a considerable geographic area, as in the case of a disease agent; also to spread throughout an organ or the body.

Dissemination barrier A physiological mechanism, typically the immune system, that prevents the spread of a pathogen within the body of an infected arthropod vector.

Distal Toward the free end of an appendage, or farthest from the body.

Distotibial Near the distal end of the tibia.

Diuresis Production and passage of abnormally large amounts of urine.

Diverticulum (-a) A blind, tubular sac or pouch branching from a cavity or canal; usually associated with the alimentary tract.

DNA Deoxyribonucleic acid; the main component of chromosomes that carries the genetic information in a cell.

DNA probe A substance or technique used to identify a segment of DNA; uses a known sequence of nucleotide bases from a DNA strand to detect a complementary sequence in a sample.

Domestic Entomologically speaking, found in, or immediately around, the home or other area of human habitation; e.g., domestic cockroaches and flies (see *peridomestic*).

Domiciliary Referring to a residence or home.

Doramectine (doramectin) A derivative of ivermectin used as a veterinary drug for treatment of roundworms, grubs, lice and mites in cattle.

Dormant (-cy) A general term for various inactive states of an organism in which growth and development cease and metabolism is reduced; includes aestivation, quiescence, and diapause.

Doxycycline A broad-spectrum antibiotic derived from tetracycline.

Drag cloth A piece of fabric attached to a horizontal pole that is dragged over vegetation and the ground to collect questing (host-seeking) ticks.

Draught animal Any animal, other than a human, that is used for its physical power to pull or operate equipment, transport goods, or otherwise provide work; also, draft animal.

Dromedary A one-humped camel, *Camelus dromedarius*, native to northern Africa and western Asia; also called the Arabian camel.

Dry ice A solid form of carbon dioxide, commonly used as a cooling agent and as a source of carbon dioxide for various insect traps.

Duff A layer of decomposing leaves, needles, and twigs in a woodland setting, between the ground litter and underlying mineral soil.

Dung Animal excrement, usually referring to vertebrates; feces or manure.

Dust bag A sack containing insecticidal dust that is suspended across an opening through which livestock pass; insecticide is applied on contact as an animal walks under it.

Dung flies Dipterous insects that breed in animal excrement, or dung.

Dung pat A discrete deposit of feces, produced by bovine animals when they defecate.

Dysentery Inflammation of the mucous membranes of the lower intestinal tract, particularly the colon; usually caused by bacteria, protozoa, or other parasitic infections; can result in severe diarrhea with passage of blood and mucus.

Dyspnea Difficult or labored breathing.

Ear tag A plastic strip or other device impregnated with a slow-release insecticide or acaricide fastened to the animal's ear; used to control flies and other arthropod pests; also tags with an identification number.

Ecchymosis Seepage of blood from damaged blood vessels into subcutaneous tissue, resulting in purple or black-and-blue discoloration of the skin, e.g., as from a bruise.

Ecdysis The process of molting by arthropods.

Ecdysteroid A general term for insect molting hormones, i.e., ecdysone and its homologues.

Eclose (-ed, -ion) Hatching of the insect larva from an egg; also used to refer to emergence of an adult insect from the pupal stage.

Ecophenotype (-ic) An ecological form of an organism, with slightly distinct morphology; e.g., *Sarcoptes scabiei* on different host species.

Ecotone A transitional zone between two ecological communities or ecosystems.

Ecribellate Referring to spiders that lack a cribellum.

Ectoparasite A parasite that lives on the surface or exterior of another organism.

Eczema An inflammatory skin condition characterized by redness, itching, serous exudates, crusting, and scaling.

Edema An accumulation of an excessive amount of serous or watery fluid in body tissues or cavities.

Edentate Belonging to the Edentata, an order of mammals in Central and South America that have few or no teeth; include armadillos, sloths, and anteaters.

Efficacious Capable of producing a desired effect or result; effective.

Egg breaker (See Egg burster).

Egg burster A cuticular projection, typically on the head of an embryo, which is used to break the egg shell when hatching; often evident in the first-instar larva.

Egg raft A floating mass of insect eggs, in which the eggs are oriented vertically side-by-side with the anterior ends downward; e.g., characteristic of certain mosquito genera such as *Culex* and *Culiseta.*

Ehrlichiosis (-es) Infection caused by rickettsial bacteria of the genus *Ehrlichia*; many species are transmitted by ticks.

EIA Enzyme immunoassay; an assay in which an enzyme is bound to an antigen or antibody that is used as a label for detecting a specific protein; *see* ELISA.

EIA Equine infectious anemia; also known as Coggins of horses; caused by a retrovirus and mechanically transmitted by blood-sucking insects, notably, tabanid flies.

Ekbom syndrome See delusory parasitosis; named after the Swedish neurologist Karl Axtel Ekbom, who described this condition in the 1930s; also called Ekbom's syndrome and Ekbom's disease.

Elaeophorosis Parasitism of deer, elk and moose by filarial nematodes of the genus *Elaeophora*, transmitted by the bite of tabanid flies.

Elateriform A larval form of holometabolous insects resembling that of click beetles (Elateridae), or wireworms; elongate cylindrical body, heavily sclerotized, with short legs.

Elephantiasis A chronic, extreme form of human filariasis characterized by greatly enlarged cutaneous and subcutaneous tissues, especially of the legs and scrotum, caused by lymphatic obstruction due to filarial nematodes.

ELISA See *enzyme-linked immunosorbent assay.*

Elytron (-a) The leathery or hardened forewing of beetles that covers the hindwing.

Emasculate To remove, or render nonfunctional, the testicles of a male animal; castrate.

Embryogenesis The development and growth of an embryo.

Emetic Causing to vomit, or an agent that induces vomiting.

Emphysema A chronic disease of the lungs characterized by abnormal enlargement of the air spaces, or alveoli, and destruction of the alveolar walls.

Empodium (-a) A single, median padlike or bristle-like structure between the pair of tarsal claws in some insects.

Emulsifiable concentrate A formulation of insecticide in which a highly concentrated active ingredient in liquefied form is suspended in another liquid, as an emulsion.

Encapsulate (-ion) To surround or encase something, as if in a capsule; e.g., the body's defensive response to a parasitic organism, or enclosing a drug or slow-release insecticide in a capsule.

Encephalitides diseases that cause encephalitis and encephalomyelitis; some mosquitoes and ticks transmit arboviruses that can cause different encephalitides.

Encephalitis Inflammation of the brain.

Encephalomyelitis Inflammation of the brain and spinal cord.

Encyst (-ed) To enclose or become enclosed in a cyst, a membranous sac filled with fluid, semifluid or solid material.

Endectocide A systemic compound used to kill both internal and external parasites; e.g., in livestock.

Endemic (1) native to a specific geographic region and not naturally occurring elsewhere, as in endemic species; (2) relating to a pathogen or disease that occurs more or less continuously in a particular locality.

Endemicity Confinement of an organism or disease to a particular geographic area; *see* endemic.

Endocarditis Inflammation of the endocardium, or membranous lining of the heart, particularly of the valves.

Endogenous Derived or originating internally; as opposed to exogenous.

Endoparasite A parasite living within the body of its host.

Endophagic Feeding within a host by certain parasitic insects.

Endophilic (-y) Ecologically associated with humans and their domestic environment; used to refer to insects occurring indoors or under overhangs of human and animal dwellings; e.g., certain mosquitoes.

Endothelium (-al) A thin layer of flat epithelial cells lining the blood vessels, lymphatic vessels, heart, and serous cavities.

Endotoxin A toxin produced by certain bacteria that is released only when the bacterial cell breaks down.

Enteric Of or relating to the intestines, or enteron.

Enteritis Inflammation of the intestinal tract, particularly the small intestine.

Enterotoxigenic Producing an enterotoxin, a toxin originating in intestinal contents, usually by bacteria.

Entomology The branch of zoology dealing with the study of insects.

Entomopathogen (-ic) An organism that is pathogenic to insects; e.g., certain bacteria and fungi.

Entomophobia An abnormal or morbid fear of insects.

Envenomation The injection of a poisonous substance by bite, sting, spine, or other means; also envenomization and British envenomisation.

Enzootic Relating to a disease involving nonhuman animals in a specific geographic area, where the disease is constantly present but at low incidence.

Enzootic transmission The transfer of a pathogen or parasite among nonhuman animals in an enzootic disease cycle, typically by arthropods.

Enzootic vector An arthropod that plays a role in transmission of a disease agent in an enzootic cycle.

Enzyme-linked immunosorbent assay (ELISA) An immunoassay that uses an enzyme linked to an antigen or antibody to detect antibodies or antigens of specific pathogens or parasites; often used in diagnostic tests for infectious agents to detect specific antibodies in blood.

Eosinophil A white blood cell with cytoplasmic inclusions or granules that are readily stained by the acid dye eosin; also other cells, microorganisms, or histological elements that are easily stained by eosin.

Eosinophilia An increase in the number of eosinophils in the blood, above normal levels.

Epidemic A widespread outbreak of an infectious or transmissible disease in humans, infecting large numbers of people at the same time; also used as an adjective.

Epidemiology A branch of medical science dealing with the cause, incidence, distribution, ecology, and control of diseases in human populations.

Epidural Located on or outside the dura mater, the outer membrane covering the brain and spinal cord; e.g., epidural fat.

Epigean Living or occurring on or near the surface of the ground.

Epimastigote A developmental stage of flagellate protozoans with an undulating membrane, in which the flagellum arises from the kinetoplast and emerges from the anterior end of the organism; e.g., trypanosomes; replaces former term "crithidial stage."

Epinephrine *See* adrenaline.

Epiornitic Outbreak of a disease in a bird population.

Epipharynx A median structure on the posterior, or ventral, surface of the labrum or clypeus overlying the mouth in some insects; has no relation to the pharynx.

Epiponine Pertaining to tropical social wasps belonging to the subfamily Epinoninae of the family Vespidae.

Epizootic An outbreak of a disease in animals other than humans, involving a large number of individuals within a particular geographic area; also used as an adjective, as in epizootic disease; counterpart of epidemic in human disease.

Epizootic transmission The transfer of a pathogen or parasite from one animal to another in the epizootic cycle of a disease.

Epizootiology The study of diseases in nonhuman animals, involving the cause, incidence, distribution, ecology, and control, especially of epizootic diseases; counterpart of epidemiology in human disease.

Epyginum A hardened plate partially covering the genital opening of female spiders.

Equid *See* equine.

Equine A member of the family Equidae, including horses, zebras, and donkeys; equid; also used as adjective.

Eruciform Caterpillar-like larval form of insects; cylindrical body, with distinct head, and both thoracid legs and abdominal prolegs; characteristic of Lepidoptera, Mecoptera, and some Hymenoptera.

Erucism Urticaria, or other forms of dermatitis, caused by stinging spines of lepidopteran larvae, i.e., caterpillars of moths and butterflies.

Erythema (-atous) Redness of the skin caused by dilatation and congestion of capillaries; a common sign of infection or inflammation.

Erythrocyte (-ic) A red blood cell in vertebrates; transports oxygen and carbon dioxide to and from tissues.

Eschar A dry scab or crust formed on the skin with a necrotic center, as a result of contact with a caustic or corrosive substance or a burn; often used to describe the black scab associated with brown-recluse spider bites or the characteristic entry lesion for some rickettsial pathogens.

Escutcheon On a cow, the shield-shaped area with a distinct hair pattern between the udders and the genital opening.

Esophagitis Inflammation of the esophagus.

Esterase Any enzyme that hydrolyzes an ester into an alcohol and an acid.

Estrogen Any of several female hormones, produced primarily by the ovaries, which promote estrus and the development growth of typical female sexual characteristics.

Ethiopian The zoogeographical region that includes Africa south of the tropic of Cancer, Madagascar, and the southern part of the Arabian Peninsula; also called the Afrotropical region.

Etiologic agent A pathogen or substance that causes a disease; a causative agent.

Etiology (-ic) Study of the causes of disease.

Eurypterid A member of the extinct order Eurypterida, marine chelicerate arthropods that resembled scorpions and lived in the Ordovician period to the end of the Paleozoic era.

Eusocial Behavior in social organisms characterized by cooperation among individuals in rearing young, division of labor, and overlap of generations; e.g., ants, social wasps, and bees.

Euthanasia Intentional painless death by artificial means.

Exarate A type of insect pupa in which the appendages are free, usually not protected by a cocoon; typical of most holometabolous insects other than Diptera and Lepidoptera.

Excoriate (-ed, -ation) To abrade or strip off the epidermis or the coating of any organ of the body by trauma, burns, chemicals, or other causes.

Excrement Waste material, especially feces, passed out of the body following digestion.

Excrescence An abnormal outgrowth or enlargement of a body surface, usually harmless.

Excreta Any waste material excreted from the body; e.g., urine, feces, and sweat.

Exflagellation The formation of microgametes (flagellated bodies) from microgametocytes in sporozoans; e.g., *Plasmodium* spp. in the mosquito midgut.

Exfoliate (-ed) To remove, come off, or separate as scales, flakes, or layers; e.g., exfoliated skin.

Exoerythrocytic Occurring outside red blood cells; e.g., multiplication of *Plasmodium* in liver cells.

Exogenous Derived or originating externally; cf. endogenous.

Exophilic (-y) Ecologically independent of humans and their domestic environment; found only outdoors.

Exotic From another part of the world; foreign; introduced from abroad but not fully naturalized or acclimatized.

Exotoxin A poisonous substance produced by a microorganism and released into the surrounding medium.

Expectorate (-ed) To expel via the mouth body fluids coughed up from the throat and lungs, including saliva, sputum, and phlegm.

Exsanguinate (-tion) To be drained of blood.

Exserted Projecting beyond the body or over a particular point.

Extrafloral nectar (-y) Nectar that is produced outside the flower, in nectaries generally located on the petiole, mid-rib, or margin of the leaf.

Extraoral digestion The digestion of food material outside the mouth; characteristic of spiders and other predaceous arachnids that cannot ingest solid food.

Extrinsic incubation period The time interval between an arthropod vector acquiring a pathogen or parasite and the ability of the vector to transmit the agent to a susceptible vertebrate host.

Exudate (-ive) A fluid or semisolid material that passes slowly out of a body tissue or its capillaries, due to inflammation or injury.

Exuvium (-ae) The cast skin (exoskeleton) of molted insect larvae or nymphs; in the strict sense, used in the plural.

Eyespot A rudimentary ocellus.

Facet A small face or surface; the external surface of each individual unit, or ommatidium, that makes up the compound eye in insects.

Facial lunule The crescent-shaped space at the base of the antennae in members of the Cyclorrhapha (Diptera), bounded by the frontal suture; also called frontal lunule.

Facultative May or may not take place; optional, depending on the conditions.

Fanniid A member of the dipterous family of muscoid flies Fanniidae, closely related to Muscidae.

Fascia A sheet or band of fibrous connective tissue supporting, covering, or separating muscles, organs, and other soft body structures.

Fascial plane A sheet of fibrous connective tissue supporting, covering, or separating muscles, organs, and other soft body structures (i.e., fascia); provides means by which cattle grubs can move within a host animal while minimizing direct injury to organs and other tissues.

Fascicle A bundle or tight cluster of elongate structures; in entomology, used to refer to the stylet-like mouthparts of certain insects that collectively form a food canal and mechanism for piercing plant or animal tissues; e.g., mouthparts of adult mosquitoes.

Febrile Pertaining or characterized by fever; feverish.

Fecund (-ity) Producing or capable of producing offspring, particularly in large numbers.

Feedlot A tract of land where livestock are fattened for market.

Felid A member of the cat family Felidae.

Femorotibial joint The articulation between the femur and tibia.

Femur The leg segment in insects between the coxa and tibia; in arachnids between the coxa and patella (genu).

Fenoxycarb A carbamate insecticide.

Feral Existing in a wild or untamed state.

Fetid Having an offensive odor.

Fibrilla (-ae) A minute filament or fiber.

Fibrinogen A protein in blood plasma that is converted to fibrin when blood clots.

Fibroma A benign tumor consisting primarily of fibrous tissue.

Fibrosis The formation of excessive fibrous tissue.

Filaria (-ae, -al) See *Filarial nematodes.*

Filarial nematodes Members of the superfamily Filarioidea, parasites of vertebrates as adults and as larvae in mosquitoes, black flies, and other insects; characterized by prelarval microfilaria stage; e.g., genera *Wuchereria*, *Brugia*, and *Onchocerca.*

Filariasis A disease caused by infestation of tissues with filarial nematodes.

Filth flies Flies that breed in excrement and other animal wastes, carrion, or garbage; include primarily muscoid flies in the families Muscidae, Calliphoridae, Fanniidae, and Sarcophagidae.

Fistula An abnormal passage from a hollow organ to the surface or from one organ or cavity to another, caused by injury, or disease, or for experimental testing and recording.

Flag cloth A piece of fabric attached to a rod in flaglike fashion, for collecting ticks; can be used to probe animal burrows, under fallen trees, and other sites where a drag cloth is not effective.

Flagellomere The individual units, or pseudosegments, that compose the flagellum of an insect antenna.

Flagellum (-a) The third and apical segment of the basic insect antenna; also a threadlike or whiplike extension of unicellular organisms and other cells that function in locomotion.

Flank The side of an animal between the ribs and hips; also more generally used to refer to the side of livestock animals and other quadrupeds.

Flare A spreading area of redness of the skin surrounding the primary site of an infection or irritation, due to dilation of arterioles.

Flavivirus A genus of single-stranded RNA virus in the family Flaviviridae, also formerly known as group B arboviruses, transmitted by mosquitoes or ticks.

Float hair A specialized dorsal seta on the abdominal segments of *Anopheles* mosquito larvae that helps to hold the abdomen parallel to the water surface; characterized by flattened, movable, usually horizontal branches radiating from a short stem; also called palmate seta.

Fluke A flatworm of the class Trematoda, including internal and external parasites of vertebrates; with thick integument and one or more suckers or hooks for attaching to its host.

Fly speck A small dark spot or stain made by excrement of a fly; also flyspeck.

Fly strike Cutaneous myiasis, especially those caused by blow flies (family Calliphoridae) in sheep (e.g., Australia).

Focus (-i, -al) In epidemiology, a localized area of disease or infection, or the center from which a disease develops and spreads.

Follicle A crypt or narrow-mouthed, cell-lined depression in the skin from which the hair emerges; also a small spherical group of cells with a central cavity, e.g., ovarian follicle.

Follicular relict The remains of a follicle after it has released an egg, usually evidenced by a distention of the ovariole wall.

Fomite Any inanimate object that can be contaminated by an infectious organism and thus is capable of transmitting it from one individual to another; e.g., clothing, bedding, towels, doorknobs, toys, cellphones.

Food poisoning An acute gastrointestinal condition caused by food containing natural toxins or contaminated with a toxic chemical; also by bacteria or their toxins, e.g., *Salmonella*; characterized by fever, chills, headache, abdominal and muscular pain, nausea, and diarrhea.

Forage To wander in search of food or provisions; also, grass or hay browsed or grazed by horses, cattle and other livestock; food for domestic animals, or fodder.

Forensic Pertaining to, or used in, courts of law or public discussion; relating to investigations to establish facts or evidence involving legal procedures; e.g., forensic entomology and forensic medicine.

Forensic entomology The study of insects and their use involving the courts and legal proceedings; the application of such knowledge and data to forensic investigations.

Foretibia Tibia of the first, or front, leg in insects and other arthropods.

Formicophilia A psychosexual disorder of humans in which an individual experiences erotic arousal and orgasm in response to small creatures (e.g., ants, cockroaches, snails, and other invertebrates) crawling, creeping, or nibbling on the body, especially the genitalia, perianal area, or nipples.

Formulation A substance prepared according to a formula; used to refer to commercial products, such as insecticides, indicating the active ingredient and chemical composition, their respective weight or percentage, and other attributes.

Fossa (-ae) A furrow, cavity, or depression; in insects, mites, and other arthropods often for protecting an appendage that can be drawn into it; e.g., antennal fossa and leg fossa; in birds, the nasal fossa containing the nostrils.

Fossula (-ae) a relatively long and narrow depression or groove; in insects, usually on the sides of the head or prothorax.

Foveal pore An opening to a small pit or cuplike depression; e.g., opening from which a sex pheromone produced by some ticks is released.

Frons (-tal) The front of the insect head above the clypeus.

Frontoclypeus (-eal) The front of the insect head composed of the frons and clypeus.

Fulcrum (-a) Any structure that serves as a support to another; e.g., fulcra of pectine in scorpions.

Fulminate (-ing) To occur suddenly or intensely.

Fungivore An organism that feeds on molds and other fungi.

Furunculus (-i, -ar) A boil; a deep inflammation of the skin usually resulting in suppuration and necrosis; also furuncle.

Fusiform Cylindrically rounded and tapered from the middle toward each end; spindle-shaped.

Galea (-ae) Outer lobe of the maxilla; highly modified, especially in Diptera and Hymenoptera; forms coiled tongue of Lepidoptera.

Galliform Referring to ground-nesting birds of the order Galliformes; includes grouse, quail, pheasants, chickens and turkeys; also called gallinaceous.

Gallinaceous *See* galliform.

Gamete A mature sexual cell that unites with another cell to form a zygote, and ultimately, a new organism; e.g., sperm and egg.

Gametocyte A cell from which a gamete develops; e.g., oocyte or spermatocyte.

Gametogenesis The production and development of gametes.

Gametogony A stage in the sexual cycle of sporozoans in which gametes are formed, often by schizogony.

Ganglion cell A neuron, or nerve cell, having its body outside the central nervous system.

Gangrene Death and decay of body tissue caused by insufficient blood supply, often involving an extremity or a limb; necrotic tissue.

Garbage fly A general term for flies that breed in, or are attracted to, discarded food and other solid wastes.

Gaster In ants and some other Hymenoptera, the rounded part of the abdomen posterior to the pedicel or petiole.

Gasterophiline Referring to members of the subfamily Gasterophilinae, or horse bots.

Gastric caecum (-a) A pouch or blind outpocketing of the midgut in insects (and some other organisms).

Gastritis Inflammation of the stomach, especially the mucous lining; associated pain, tenderness, nausea, and vomiting.

Gastroenteritis Inflammation of the mucous membrane lining the stomach and intestines; also called enterogastritis.

Gena (-ae, -al) The side of the insect head below the compound eyes.

Genal ctenidium (-a) A comblike row of strong spines on the anteroventral border of the head in fleas; *see* ctenidium.

Gené's organ In female ticks, a paired extrusible structure located dorsally between the captitulum and scutum, used to apply a coating of wax to eggs as they are deposited.

Geniculate Elbowed, or bent at a joint like a knee; e.g., antennae of ants.

Genital chamber In female insects, an invaginated cavity behind the eighth abdominal sternum into which the gonopore and spermathecal duct opens; often forms a pouchlike or tubular vagina or uterus; in males, a ventral invagination behind he ninth sternum containing the intromittent organ (aedeagus).

Genital pore The external opening of the male or female reproductive tract; also called gonopore.

Genital pouch *See* Genital chamber.

Genitalia Organs of the reproductive system, especially the external genital organs.

Genome(-ic) An organism's genetic material, or full DNA or RNA sequence of a haploid set of an organism's chromosomes.

Genospecies A group of organisms that can interbreed; all are genotypes within a single species.

Genu In arachnids, the segment of the leg between the femur and tibia.

Gingival Of or relating to the gums, the firm fleshy tissue overlying the jaw.

Glanders A disease of horses caused by the bacterium *Pseudomonas mallei* and communicable to humans; characterized by swollen lymph glands beneath the jaw and profuse mucous discharge from the nostrils.

Glaucoma An eye disorder characterized by an increase in intraocular fluid pressure that can lead to atrophy of the optic nerve and blindness.

Glycoprotein A macromolecule made up of protein**(s)** bonded to one or more carbohydrates.

Gnat A nontaxonomic term referring in general to small pesky flies that may or may not bite.

Gnathosoma The anterior-most body region of mites and ticks composed of the mouthparts and palps and their fused bases; also called captitulum.

Gonad A sex gland in animals that produces gametes; e.g., ovary and testis.

Gonoactive A blood-fed female arthropod that is progressing through a gonotrophic cycle.

Gonocoxite Basal segment of the external male or female genital appendages on segment 8 or 9 in insects, surrounding the genital opening.

Gonoinactive A blood-fed female arthropod that is not progressing through a gonotrophic cycle.

Gonopod In arthropods, an appendage modified for a reproductive function, such as copulation, intromission, or oviposition; usually associated with genital segments 8 and 9.

Gonopore See *genital pore.*

Gonostylus (-i) A process on abdominal segment 8 or 9 of insects, in males generally modified to form a clasping organ.

Gonotrophic concordance A situation where a blood-fed female vector follows a predictable gonotrophic cycle.

Gonotrophic cycle The reproductive cycle of blood-feeding, bloodmeal digestion, egg maturation, and oviposition in a vector; most commonly pertaining to mosquitoes and some other Diptera.

Gonotrophic discordance A situation where a blood-fed female vector does not follow a predictable gonotrophic cycle.

Gonotrophic dissociation A situation where a blood-fed female vector does not develop eggs after the bloodmeal.

Grand mal seizure A sudden attack or convulsion characterized by generalized muscle spasms and loss of consciousness; an epileptic attack.

Granulation tissue Small protuberances that form on the surface of a wound during the healing process; consists of outgrowths of new connective tissue and capillaries.

Granulocyte White blood cells containing granules in the cytoplasm; important in phyagocytosis and immunological responses; most numerous of the white cells in humans.

Granuloma (-tous) A mass of granulation tissue resulting from inflammation, infection, or injury; usually associated with skin or lymphoid tissues.

Gravid Carrying eggs or developing young.

Gregarine Sporozoans in the order Gregarinida that are parasitic in the digestive tracts of various invertebrates, including insects, other arthropods, and annelids; produce cysts filled with spores.

Gregarious Tending to form a group with individuals of the same species.

Grub A thick-bodied, typically whitish insect larva with thoracic legs but lacking abdominal prolegs; relatively sluggish or inactive; a term loosely applied to larvae of Coleoptera, Hymenoptera, and certain Diptera (e.g., Oestridae).

Guanine A major excretory product of arachnids; also, one of the constituent purine bases that codes genetic information in DNA and RNA, pairs with cytosine.

Guenon Any of various slender, arboreal African monkeys with long hindlegs, long tail, and long hair around the face; members of the genus *Cercopithecus.*

Guinea fowl An African gallinaceous bird, *Numida meleagris*, introduced to many other parts of the world.

Gullet Esophagus.

Gut barrier (midgut barrier) Something in the gut of a particular blood-feeding arthropod that prevents it from becoming infected with a particular pathogen through that inoculation route.

Gut bolus A soft mass of ingested food in the alimentary canal; in vertebrates, usually referring to the bowel or intestines; in invertebrates, may refer to the midgut or hindgut.

Habronemiasis Infestation of horses by parasitic nematodes of the genus *Habronema* (family Spiruridae), which develop in flies of the genera *Musca* and *Stomoxy*s; if ingested, can cause inflammation of the stomach; can also cause ulcerated cutaneous lesions, or summer sores.

Haematophagia (-y) Feeding on blood.

Haemocoel *See* hemocoel.

Haemogregarine A member of the sporozoan genus *Haemogregarina* (order Coccidia) that is parasitic in the alimentary tract of invertebrates and the circulatory system of vertebrates; also hemogregarine.

Haemoproteid A sporozoan of the family Haemoproteidae parasitic in birds; e.g., *Haemoproteus.*

Haemosporidian A member of the protozoan order Haemosporidia, parasitic at some stage of development in blood cells of vertebrates; includes the families Babesiidae, Haemoproteidae, and Plasmodiidae.

Hair follicle A tubular invagination of the skin from which a hair develops.

Haller's organ A complex sensory apparatus on the dorsal surface of the tarsus of leg 1 in all developmental stages of ticks; includes receptors for taste, odor, temperature, and mechanical stimuli.

Haltere A drumstick-shaped sensory projection that replaces each hindwing of members of the Diptera (and each forewing of members of the Strepsiptera); the sheep ked, *Melophagus ovinus*, has secondarily lost its halteres.

Hammock An elevated tract of forest land surrounded by wetlands, usually applied to areas of freshwater marsh, such as the Everglades, in the southeastern United States.

Hanging groin In human cases of lymphatic filariasis, distention of the scrotum of males due lymphatic blockage and resultant accumulation of fluids.

Haploid An organism or cell having only one set of chromosomes, half the number found in somatic cells.

Harborage A sheltered or protected place; used regarding cockroaches, rodents, and other household pests to indicate where they live and hide.

Haustellum (-late) The anterior part of the insect head or basal portion of the mouthparts modified for sucking; e.g., the conical projection of sucking lice, and the structures of certain flies with sponging or piercing mouthparts, such as tsetse flies and house flies.

Head capsule The fused sclerites of the insect head that together form a hard exoskeletal case.

Hedgehog Small insectivorous mammals of the genus *Erinaceus*, family Erinaceidae, with erect spiny hairs and the ability to roll into a ball for protection; native to Africa, Asia, and Europe.

Helminth Parasitic worms, usually referring to those living in the intestines of vertebrate animals; includes roundworms, tapeworms, and flukes.

Hematocrit The volume of red blood cells in a sample of blood after centrifugation, expressed as a percentage of the total blood in the sample.

Hematologic Relating to the medical study of blood and blood-forming organs.

Hematoma A localized swelling filled with blood resulting from a break in a blood vessel.

Hematophage (-ous) An organism that feeds on blood.

Hematopoiesis (-tic) The formation and development of blood cells; *syn.* Hemopoiesis.

Hematuria Presence of blood in the urine.

Heme A deep red, iron-containing, nonprotein component of hemoglobin in vertebrate blood that binds with oxygen.

Hemelytral pad One of a pair of dorsal caselike projections on the mesothorax of late-instar nymphs of Hemiptera, which give rise to the forewings, or hemelytra, of the adult; also the reduced forewings of adult bed bugs (Cimicidae).

Hemelytron(-a, also hemi-elytron) The forewing of Hemiptera, in which the basal half is thickened and the distal half is membranous.

Hemidesmosome A specialized junction that connects the basal surface of epithelial cells to the underlying basement membrane.

Hemimetabolous Pertaining to metamorphosis in insects represented by three developmental stages: egg, nymph (larva), and adult; e.g., orders Blattaria, Phthiraptera, and Hemiptera.

Hemocoel The body cavity of insects and other artrhopods filled with hemolymph.

Hemocyte A blood cell.

Hemoglobinuria Presence of hemoglobin in urine.

Hemolymph Circulatory fluid filling the body cavity of arthropods and other invertebrates, analogous to blood and lymph of vertebrates; consists of water, inorganic salts, lipids, amino acids, and sugars.

Hemolysin A substance or agent that causes hemolyis; e.g., an antibody or bacterial toxin.

Hemolysis Destruction of red blood cells releasing hemoglobin.

Hemopoiesis *See* hematopoiesis.

Hemorrhage (-ic) Excessive loss of blood from blood vessels; profuse bleeding.

Hemosporine A member of the order Haemosporida (Haemosporidia); includes the genera *Haemoproteus*, *Leucocytozoon,* and *Plasmodium.*

Hemostatic Arresting loss of blood due to hemorrhaging.

Hemotoxic A substance that destroys red blood cells.

Hemotropic An entity (often a pathogen or parasite) that attracts phagocytic cells in the blood; *syn.* hematotropic.

Heparin A natural substance found particularly in lung and liver tissues that prevents clotting of blood; also the commercial form.

Hepatitis Inflammation of the liver, usually caused by a virus or toxin.

Hepatopancreas An organ associated with the digestive tract of arthropods, crustaceans, mollusks, and other invertebrates that serves functions similar to the liver and pancreas of mammals.

Hepatosplenomegaly Enlargement of the liver and spleen.

Hibernaculum (-a) A protective site in which an organism overwinters.

Hippoboscid A member of the dipterous family Hippoboscidae; louse flies and keds.

Histamine A compound released primarily by mast cells in allergic reactions that causes dilation and permeability of capillaries, decreased blood pressure, and constriction of bronchial muscles; also the commercial form.

Histiocyte A macrophage cell found in connective tissue.

Histoblast A cell, or group of cells, capable of forming tissues; in dipteran larvae, epithelial cells that give rise to structures other than appendages; e.g., respiratory histoblasts of black fly larvae.

Histolysis The breakdown and disintegration of tissues.

HIV Human immunodeficiency virus, which causes acquired immune deficiency syndrome (AIDS) by infecting and killing helper T cells.

Hives An eruption of itching wheals, usually of systemic origin; may be due to hypersensitivity to certain foods, drugs, pathogens, or parasites.

Hock A joint in the hindleg of pigs, cows, and horses; above the fetlock and corresponding to the ankle of humans, although it bends in the opposite direction.

Holarctic Region A zoological area in the Northern Hemisphere encompassing the nontropical parts of Europe and Asia, Africa north of the Sahara, and North America south to the deserts of Mexico; includes the Nearctic and Palearctic regions.

Holocyclotoxin A neurotoxin in the saliva of the Australian tick *Ixodes holocyclus* that causes tick paralysis in cattle and other livestock, humans, dogs, and cats; inhibits release of acetylcholine.

Holometabolous Pertaining to metamorphosis in insects represented by four developmental stages: egg, larva, pupa (chrysalis), and adult; e.g., orders Diptera, Siphonaptera, Hymenoptera, Coleoptera, and Lepidoptera.

Holoptic Refers to adult flies in which the pair of eyes are enlarged enough to meet along the dorsal midline of the head; e.g., male horse flies.

Homeostatic The physiological ability of an organism to maintain internal equilibrium.

Homeothermic Refers to an animal, typically a bird or mammal, capable of maintaining a constant body temperature largely independent of the ambient temperature; endothermic.

Honeydew A sugar-rich secretion deposited on vegetation by aphids, scale insects, and other plant-feeding insects; serves as a natural source of sugar for certain hematophagous flies.

Horizontal transmission The transfer of an infectious agent from one individual to another other than from parent to offspring.

Host preference The species or range of species to which an ectoparasitic or other blood-feeding arthropod, given a choice, is typically attracted and on which it feeds.

Host specificity The degree of selectivity exhibited by ectoparasitic or other blood-feeding arthropods in choosing a host.

Humoral immunity The aspect of immune systems mediated by antibodies produced by lymphocytes (B cells) in bone marrow.

Husbandry The application of scientific principles to agricultural practices and farming, particularly livestock and animal breeding.

Hyaline Glassy or transparent.

Hyaluronidase An enzyme that plays a role in breaking down hyaluronic acid, increasing the permeability of tissues to fluids; also called spreading factor.

Hydramethylnon A chemical compound that acts as a metabolic inhibitor, used as an insecticidal bait for cockroaches and ants.

Hydrocele Accumulation of serous fluid in a body cavity; e.g., in scrotum.

Hydroprene An insect growth regulator, used to control cockroaches.

Hymenopteran Referring to members of the insect order Hymenoptera.

Hyperemia Increased blood flow to an organ or other body part.

Hyperendemic Occurrence of a pathogen or parasite at very high incidence in a host population, or in a particular geographical area.

Hypergammaglobulinemia An excess of gamma-globulins in the blood, often associated with chronic infectious diseases.

Hyperimmune (-ity) Exhibiting an unusually high degree of immunity in which the body is extremely reactive against a particular antigen.

Hyperkeratosis Proliferation of cells of the cornea, or thickening of the horny layer of the skin.

Hypermetamorphosis (-phic) A type of holometabolous development in insects in which the larval stage is represented by two or more different larval types; e.g., blister beetles.

Hyperpigmentation Excess pigmentation, or darkening, usually of the skin.

Hypersensitive (-ity) Responding excessively to an allergen or other foreign agent; abnormally sensitive or allergic.

Hypertension Abnormally elevated blood pressure; arterial disease characterized by chronic high blood pressure.

Hypertrophy An abnormal enlargement of an organ or tissues due to an increase in size of the cells but not their numbers.

Hypnozoite A latent or dormant stage of sporozoan parasites; e.g., *Plasmodium* spp. in liver cells, which contribute to relapses in malaria cases.

Hypoendemic Occurrence of a very low incidence of a pathogen in a host population, with little transmission.

Hypognathous In insects, the mouthparts directed downward or ventrally.

Hypopharynx In insects, a median sensory structure anterior to the labium, usually associated with the salivary ducts; in certain sucking insects, an elongate mouthpart containing the salivary channel.

Hypopleuron In insects, the lower part of the of the external mesothoracic wall immediately above the middle and hind coxae.

Hypopode (-es) *See* hypopus.

Hypopus (-i) A highly modified, typically nonfeeding form of the deutonymph in astigmatid mites, adapted for dispersal and enduring adverse environmental conditions.

Hypopygium A modification of the ventral aspect of the last abdominal segment**(s)** in certain insects, notably, Diptera and Coleoptera.

Hyposensitization The process of reducing a person's sensitivity to an allergen or other stimulus, usually by injecting progressively larger doses of the allergen involved; desensitization.

Hypostome The median ventral part of the insect head posterior to the mandibles; in mites and ticks, the ventral, basal part of the gnathosoma, greatly enlarged and modified in ticks for host attachment.

Hypotension Low blood pressure.

Hypothermia Abnormally low body temperature.

Hysterosoma In mites, that portion of the body posterior to the second pair of legs.

Ibex Wild goats of the genus *Capra*, with long, ridged, recurved horns; native to the mountainous regions of Eurasia and northern Africa.

Idiosoma The major body region of mites and ticks, excluding the gnathosoma.

IGR *See* insect growth regulator.

Imidacloprid A chlorinated analog of nicotine used as an insecticide.

Immediate reaction (Type I hypersensitivity) rapid (within minutes) inflammation of the skin, known as wheal and flare, in response to bites by certain arthropods.

Immunity Ability of an organism to resist disease by destroying or inactivating infectious agents or other foreign substances.

Immunocompromised Unable to develop a normal immune response, usually because of malnutrition, disease, or immunosuppressive therapy.

Immunoglobulin (Ig) A large glycoprotein produced by plasma cells in bone marrow and loose connective tissues of vertebrates that functions as antibodies in an immune response; five classes: IgA, IgD, IgE, IgG, and IgM.

Immunoglobulin A (IgA) A class of immunoglobulins comprising 10%−15% of total immunoglobulins; often transferred transplacentally from mother to fetus.

Immunoglobulin D (IgD) A class of immunoglobulins representing less than 0.1% of total immunoglobulins.

Immunoglobulin E (IgE) A class of immunoglobulins produced in the skin, mucous membranes, and lungs that function particularly in allergic reactions, comprising less than 0.01% of total immunoglobulins.

Immunoglobulin G (IgG) A class of immunoglobulins representing the most common antibodies circulating in the blood and lymph (ca. 80% of total immunoglobulins); active against invading microorganisms and other foreign agents.

Immunoglobulin M (IgM) A class of immunoglobulins that includes antibodies released into the blood early in the immune response (typically can be detected within 3 months of an infection), with particular affinity for viruses, comprising 5%−10% of total immunoglobulins.

Immunomodulator A chemical agent that alters the immune response or functioning of the immune system; may strengthen or suppress the response; e.g., stimulation of antibody formation.

Immunosuppression Lowering of the body's normal immune response due to disease, drugs, radiation, or other conditions; e.g., HIV infection or side effects of chemotherapy and radiotherapy.

Immunotherapy Treatment to produce immunity to a disease by inducing, suppressing, or enhancing an immune response.

Impetigo Contagious inflammatory skin disease characterized by pustular eruptions and yellow crusts, commonly on the face; caused by staphylococcal and streptococcal bacteria, especially in children.

in copula Linking of a male and female in the act of pairing or mating; commonly used when referring to insects.

Inapparent infection Presence of infection without symptoms; asymptomatic or subclinical infections.

Inappetence Lack of appetite.

Incidence Frequency or extent of an occurrence: e.g., infection, disease.

Incidental host An unpredictable and very minor host of a parasite or pathogen.

Incompetent Lacking the ability to play a significant role; e.g., incompetent vector, an arthropod that is not susceptible to, and/or lacks the ability to transmit, a given pathogen or parasite.

Incubation period *See* extrinsic *and* intrinsic incubation period.

Indigenous transmission Transfer of a pathogen or parasite from one host to another in its native country or region.

Indigenous Native to and occurring naturally in a given area or environment.

Indolent ulcer Inactive or painless lesion of the skin or mucous membranes caused by superficial loss of tissue, usually with associated necrosis and inflammation.

Indurate (-ed) (-tion) To become firm or hardened.

Infect (-ed) (-tion) To live in or on a host by a pathogenic microorganism or agent; *cf.* infest.

Infection rate The proportion (sometimes expressed as a percentage) of a population or specified group of individuals infected with a pathogenic microorganism or agent.

Infectious disease Any disease caused by growth and multiplication of pathogenic microorganisms in the body; may or may not be contagious.

Infectious dose A specific quantity of a pathogenic microorganism required to establish an infection.

Infective stage The developmental form of a pathogen or parasite that invades a vertebrate host; in a strict sense, a misnomer when applied to organisms other than microbes.

Infective Capable of producing infection; infectious.

Infest (-ed) (-ation) To live as a macroscopic parasite in or on a host, usually implying high enough numbers to be harmful; to parasitize; *cf.* infect.

Inflammation Reaction of tissue to injury, characterized by redness, swelling, tenderness, and pain.

Inguinal Pertaining to, or located in the groin.

Inoculate (-ion) To inject, or otherwise introduce, a microorganism or other disease agent into a host or culture medium; also, to introduce an antigenic substance, serum, or vaccine into an animal to boost immunity to a specific disease agent.

Insect growth regulator (IGR) Any chemical compound or other substance that modifies, disrupts, or otherwise interferes with normal body development and metamorphosis in insects.

Insectivore An animal or plant that feeds on insects; usually refers to members of the mammalian order Insectivora that include moles, shrews, and hedgehogs.

Installment hatching Hatching of groups of insect eggs within the same batch at different time periods, i.e., not simultaneously; often applied to mosquito eggs.

Instar The form of an insect or other arthropod between two successive molts.

Integrated pest management (IPM) The application of a variety of different control techniques against a pest species; for vectors, this may involve, for example, the use of chemical insecticides, insect growth regulators, application of fungal pathogens and/or parasitoids, and management of potential harborage sites.

Integument The outer body surface of an organism, typically applied to arthropods.

Interdigital space The area between fingers and toes, and between the digits of hoofed animals.

Intermediate host An animal in which a multi-host parasite undergoes development but does not become sexually mature.

Interrupted feeding The behavior of arthropods, typically hematophagous insects, in which feeding is disrupted and usually results in subsequent efforts to feed again.

Interstice (-es) A small or narrow space in substrates composed of closely spaced particles such as soil, rocks, sand, and dried mud.

Intranasal Within the nose, or administered via the nose.

Intravascular coagulation Clotting of blood within blood vessels.

Intrinsic incubation period The time period between infection or initial parasitism of a vertebrate host and the onset of symptoms.

Intromittent organ An external, or eversible, genital structure used in copulation; typically refers to the male insect penis or aedeagus (phallosome of some authors).

Inundative release Purposeful release of large numbers of a commercially produced biological control agent into the environment for the purpose of reducing or eliminating a target species; e.g., parasitic wasps to control muscoid flies.

Iritis Inflammation of the iris of the eye.

Isoenzyme Chemically distinct but functionally similar enzymes.

Isolate (-tion) In microbiology, to obtain an organism from a sample or the environment in pure culture.

Isopod Member of the crustacean order Isopoda, including pill bugs and sowbugs.

Ivermectin A semisynthetic product derived from the fungus *Streptomyces avermitillis* used as an anthelmintic, insecticide, and acaricide.

Ixodid Member of the tick family Ixodidae (hard ticks).

Jaundice Yellowish discoloration of the skin, whites of the eyes, and mucous membranes caused by abnormally high levels of bile pigments in the blood.

Johnston's organ An auditory structure in the second antennal segment (pedicel) of most adult insects; perceives movement of the antennal flagellum.

Juvenile hormone A hormone in arthropods that promotes larval development and inhibits molting to the adult stage.

Juvenile hormone mimic A chemical compound that simulates the effects of juvenile hormone; can be used to disrupt normal development of immature insects to the adult stage; juvenile hormone analog.

Karyotype A visual array of chromosomes of an organism, typically arranged by size, shape, number, and other characteristics; to classify the chromosomes or prepare a karyotype.

Keratin A tough structural protein found in hair, nails, claws, horns, hoofs, feathers, and dead outer layers of skin.

Keratitis Inflammation of the cornea; causes watery and painful eyes, blurred vision.

Keratoconjunctivitis Ocular inflammation of the cornea and conjunctiva.

Kinase An enzyme that catalyzes conversion of a proenzyme to an active enzyme; also, an enzyme that catalyzes transfer of a phosphate group from a high-energy phosphate-containing molecule (e.g., ATP, ADP) to a substrate.

Kinetoplast A mass of circular DNA within a large mitochondrion near the base of the flagellum in certain protozoans, e.g., trypanosomes.

Kinin A polypeptide that causes contraction of smooth muscle, vasodilation, and altered permeability of capillaries.

K-strategy (-ist) The production of relatively small, constant numbers of offspring in a stable or predictable environment, maintaining a population near the carrying capacity, K.

Labellum (-a) Sensory structure at the tip of the labium in mosquitoes, muscoid flies and some other dipterans; possess temperature, contact, and chemical receptors.

Labial palp One of a pair of typically multisegmented appendages of the labium; labial palpus (pl., palps or palpi).

Labium Posterior-most or ventral-most mouthpart in insects, depending on the orientation of the mouthparts.

Labral fan A modification of the labrum forming a brushlike structure that can be extended like a fan to trap or filter particles of food from water; e.g., black-fly larvae.

Labrum Anterior-most or dorsal-most mouthpart in insects, depending on the orientation of the mouthparts.

Lacerate (-tion) To cut, tear, or rip, leaving irregular or jagged edges.

Lachrymal Of or relating to tears or tear glands; lacrimal.

Lachryphagy (-ous) Feeding on tears from the eyes of vertebrates.

Lacinia (-ae) The inner or medial lobe of the maxilla of insects.

Lacrimation Excessive secretion of tears; lachrymation.

Lactation Secretion or formation of milk by the mammary glands.

Lactic acid A carboxylic acid produced during anaerobic metabolism of glucose, as in muscle tissue during exercise.

Lactophenol A mixture of lactic acid and phenol used to clear small arthropods, especially mites, prior to slide-mounting.

Lacustrine Of or relating to lakes.

Lagomorph A member of the mammalian order Lagomorpha, including rabbits, hares, and pikas.

Lamella (-ae, ate) A thin plate or leaflike structure; as in lamellate antennae of scarab beetles.

Lancet In insects, the first of three bladelike processes that surround the ovipositor.

Lappet A small flap or projecting, lobelike structure; characteristic of larvae of lappet moths (Lasiocampidae).

Larva (-ae) The immature stage of insects that hatches from the egg and undergoes metamorphosis to the adult; also called nymphs or naiads in certain types of metamorphosis; in mites and ticks, the six-legged stage that hatches from the egg.

Larvicide An insecticide that kills larvae.

Larviparous Bearing or depositing living larvae, rather than eggs.

Larviposit To deposit living larvae, rather than eggs.

Laryngeal Of or relating to the larynx.

Lassitude A feeling or state of weariness, lack of energy; listlessness, languor.

Laterigrade Having a sideways manner of moving, as a crab; used to refer to arthropods that are dorsoventrally flattened with the legs extending laterally in a horizontal plane and move in crablike fashion, e.g., keds.

Latrine A communal toilet, usually in a military area.

LD_{50} The dose of a substance that kills 50% of the treated or targeted organism.

Lechwe An African antelope (*Kobus leche*) that inhabits marshes and wet, grassy plains (pronounced leech' wee).

Leishmaniasis An infection caused by a flagellate protozoan in the genus *Leishmania.*

Lentic Relating to or living in still water, e.g., ponds and lakes.

Lepidopterism An affliction caused by direct or indirect contact with hairs, setae, or wing scales of adult moths and butterflies; as distinguished from similar contact with urticating hairs of caterpillars, or erucism.

Lesion Any abnormal structural change in body tissue, usually caused by trauma or infection.

Lethargy (-ic) A state of sluggish inactivity, listlessness, drowsiness, and apathy.

Leucocytozoonosis A disease of birds caused by infection with a protozoan of the genus *Leucocytozoon.*

Leukemia Cancer of the bone marrow that prevents normal production of red and white blood cells and platelets; results in a proliferation of certain kinds of leukocytes.

Leukocyte A white blood cell.

Leukopenia An abnormally low number of white blood cells circulating in the blood; leucopenia.

Leukosis An abnormal proliferation of leukocyte-forming tissues.

Ligula The terminal lobe(s) of the labium in insects.

Lindane An organochlorine insecticide, gamma benzene hexachloride; used to kill lice and scabies mites.

Listeriosis A bacterial disease of domestic and wild animals and sometimes humans, caused by *Listeria monocytogenes*; characterized by fever, meningitis, and encephalitis.

Litter In livestock or poultry operations, material used as bedding to absorb animals wastes, reduce odors, and facilitate clean-out; e.g., sawdust, wood shavings, straw.

Loiasis An infestation with the filarial nematode *Loa loa*, or African eyeworm.

Loin The area in livestock (or humans) situated ventrally on each side of the hipbone and the false ribs.

Lotic Relating to or living in running water; e.g., streams and rivers.

Lufenuron A benzoylurea compound used in veterinary products to control fleas and filarial nematodes; inhibits chitin production in flea larvae.

Lumen The space within a tubular structure or organ, such as a blood vessel or alimentary tract.

Lunule A small crescent-shaped mark or structure; the frontal suture in certain adult flies (Schizophora); *see* facial lunule.

Lymphadenopathy Pathology of lymph nodes, usually manifesting as chronic node enlargement, often associated with disease.

Lymphedema Swelling, particularly in subcutaneous tissues of the extremities, due to accumulation of lymph resulting from obstruction of lymphatic vessels and lymph nodes.

Lymphocytic choriomeningitis An acute disease caused by an arenavirus and transmitted by rodents; characterized by excessive lymphocytes in cerebrospinal fluid.

Lymphokine A cytokine secreted by helper T cells following stimulation by specific antigens; mediate the immune response by acting on other cells, e.g., activating macrophages.

Lysis The dissolution or destruction of cells.

Macaque Short-tailed monkeys of the genus *Macaca*, native primarily to Southeast Asia and northern Africa.

Macrogametocyte A female gametocyte that produces a macrogamete.

Macrophage Large cells of the reticuloendothelial system that remove cellular debris and particulate substances, including microorganisms, by phagocytosis.

Macropterous Bearing large, well-developed wings in insects.

Macrotrichium (-ia) Relatively large microscopic hairs; e.g., on the wing surface of flies.

Macule (1) a discolored spot or patch of skin not usually elevated, caused by various disease agents; (2) a spot or patch on insect integument, especially on the wings.

Maculopapular Characterized by a skin eruption with both macules and papules.

Maggot Legless, soft-bodied, vermiform fly larva, usually found in decaying matter; typified by larvae of houseflies and blowflies.

Malady Any disease, disorder, or body ailment.

Malaise A vague feeling of physical discomfort, weakness, or uneasiness, often characterizing onset of an illness or disease.

Malathion A dithiophosphorus insecticidal hydrocarbon.

Malignant Dangerous to health, tending toward a progressive, life-threatening condition.

Malodorous Having an unpleasant, offensive odor; foul smelling.

Malphigian tubule Long, slender excretory structures in insects, arachnids, and other terrestrial arthropods, opening into the anterior end of the hindgut.

Mammalophagic Feeding on mammals.

Mammalophily (-ic) Tendency of attraction to mammals by host-seeking arthropods.

Mammilla (-ae) A nipple-like process or protuberance.

Mandible One of a pair of unsegmented mouthparts of insects located between the labrum and maxillae; usually heavily sclerotized for chewing, but highly modified in some insects for piercing-sucking.

Mandibular gland Pheromone-producing gland that opens on the surface of the mandible, found in most Hymenoptera.

Mandibular stylets Insect mandibles that are highly modified as long, slender structures for piercing and/or sucking.

Mangabey Slender, long-tailed monkeys of the genus *Cercocebus* in forests of central Africa.

Mange A persistent skin condition of mammals caused by parasitic mites; characterized by redness, itching, and hair loss.

Mangrove A tropical or subtropical tree or shrub mostly of the genus *Rhizophora*, with stiltlike prop roots that form dense thickets along shallow tidal areas.

Mansonellosis An infestation of humans by filarial nematodes of the genus *Mansonella*, usually used with reference to *M. ozzardi.*

Manure A mixture of animal excrement and bedding or litter, such as hay or straw; used to fertilize land.

Mark-recapture Technique of marking animals by various methods so that they can be recognized when caught again on subsequent occasions; also called mark-release-recapture.

Marsupial A nonplacental mammal of the order Marsupialia, with a pouch and mammary glands for nuturing the young; includes kangaroos, wombats, bandicoots, and opossums.

Masarine Referring to members of the vespid wasp subfamily Masarinae, found in the western United States.

Mast cell A large granular cell found particularly in connective tissue that releases heparin, histamine, and serotonin in response to allergens, inflammation, or injury.

Mastitis Inflammation of the breast or udder.

Mating plug A physical blockage in the reproductive system of female animals, including some arthropods, caused by a seminal mass introduced by a male during copulation.

Matrone In mosquitoes, a substance in male accessory fluid introduced during copulation that causes the female to become unreceptive to other males.

Mausoleum An above-ground burial chamber or building containing tombs.

Maxilla (-ae) One of the second pair of mouthparts in insects, immediately behind the mandibles.

Maxillary palp Typically one of a pair of segmented, sensory appendages on the maxilla of insects (pl., palps or palpi).

Maxillary sinus One of a pair of air cavities in the upper jaw of vertebrates that opens into the middle passage of the nose.

Maxillary stylets Long slender mouthparts formed by the maxillae; usually associated with piercing-sucking insects.

Mebendazole An anthelmintic compound (methyl-5-benzoyl-2-benzimidazolecarbamate) used to treat roundworm infestations; interferes with carbohydrate metabolism.

Mechanical transmission The transfer of a pathogen or parasite via the external surface of the mouthparts, appendages, or other body parts, without involving biological development (cyclodevelopment) of the organism.

Mechanical vector An arthropod that transmits a pathogen or parasite as a contaminant on the external surface of a body part; *see* mechanical transmission.

Mechanoreceptor A sensory structure that responds to mechanical stimuli such as touch, pressure, tension, stretching, sound, and other vibration.

Mecopteroid A member of the insect superorder Mecopteroidea, which includes scorpion flies (order Mecoptera), fleas (Siphonaptera), true flies (Diptera), caddis flies (Trichoptera), and butterflies and moths (Lepidoptera); members of these orders are all endopterygotes that have hypognathus mouthparts.

Mectizan A brand name of ivermectin.

Media (n) Fourth longitudinal vein in the basic insect wing, counting from the anterior margin.

Medicocriminal Pertaining to medically criminal practices, including aspects of forensic entomology.

Medicolegal Pertaining to legal issues in medicine, including aspects of forensic entomology.

Megacolon Abnormal enlargement of the colon, with extreme dilation and hypertrophy.

Megaesophagus Abnormal enlargement and hypertrophy of the lower portion of the esophagus.

Megasyndrome A condition in which various organs in the abdomen are enlarged.

Meibomian gland A sebaceous gland in the eyelid of vertebrates; produces lubricant to prevent eyelids from sticking together; also called tarsal gland (tarsus being the supporting plate of the eyelid, not to be confused with insect tarsus).

Melioidosis An acute infectious bacterial disease caused by *Pseudomonas pseudomallei*, primarily affecting rodents in India and Southeast Asia; can be transmitted to humans causing pneumonia, multiple abscesses, and bacteremia.

Melittin A polypeptide and major active component of honey bee venom that causes localized pain and inflammation; also has antifungal, antibacterial, and antiinflammatory properties.

Meninges The three membranes that envelop the brain and spinal cord; singular meninx.

Meningitis Inflammation of the membranes surrounding the brain or spinal cord.

Meningoencephalitis Inflammation of the brain and its meninges.

Mentum The lower (distal) part of the insect labrum, usually bearing palps.

Mercaptan A class of sulfur-containing compounds with distinctive and often offensive garlic-like odors; also called thiol.

Mermithid A nematode in the family Mermithidae; parasites of insects and other invertebrates.

Merogony A form of asexual schizogony characteristic of sporozoans; involves division of the nucleus several times before the cytoplasm divides, forming a schizont that further divides to produce merozoites.

Meron The base of the coxa in insects located lateral to, and just posterior to, the point of articulation of the coxa and thorax; may be greatly enlarged in some insects.

Meront A stage in the asexual part of the sporozoan life cycle in which schizogony occurs, producing merozoites; occurs in the vertebrate host.

Merozoite A trophozoite, or vegetative form, in the asexual part of the sporozoan life cycle produced by a mature meront; occurs in the vertebrate host.

Mesad Toward the median plane of the body or body part.

Mesal In a middle line or plane; also mesial.

Mesenteric Referring to the mesentery, or peritoneal tissue that surrounds most of the small intestine and connects it to the abdominal wall.

Mesoendemic Referring to a geographic area in which only modest transmission of a disease agent takes place.

Mesonotal suture A groove along the dorsal surface of the second (middle) thoracic segment of some insects.

Mesonotum The dorsal part of the second (middle) thoracic segment of insects.

Mesostigmatid Referring to mites of the suborder Mesostigmata; Gamasida of some authors; Mesostigmatan, of some authors.

Mesothorax The second thoracic segment of insects, bearing the middle pair of legs and first pair of wings.

Metabolic inhibitor A substance or agent that slows or stops chemical reactions involved in an organism's metabolism.

Metacyclic In trypanosomes, referring to the developmental form of the protozoan produced in the arthropod that is the infective stage for the vertebrate host.

Metamorphosis A change in form during development of an organism.

Metanotum The dorsal part of the third (most posterior) thoracic segment of insects.

Metastriate Pertaining to hard ticks (family Ixodidae) in which the anal groove is posterior to the anus; includes all ixodid genera except *Ixodes.*

Metatarsus The tarsus, or last segment, of the second leg in arthropods.

Metathorax The third (most posterior) thoracic segment of insects, bearing the hindlegs and hindwings.

Metazoa (-an) A subdivision of the animal kingdom that includes all multicellular animals, with cells differentiated to form tissues and organs.

Methoprene A synthetic insect juvenile hormone used as a pesticide to disrupt normal larval development as a means of control.

Methoxychlor A synthetic organochlorine insecticide that acts both as a neurotoxin and an endocrine disruptor.

Microfilaremia (-ic) The presence of microfilariae in the blood of a vertebrate host.

Microfilaria (-ae) The minute embryonic larva of filarial nematodes produced in the arthropod host and serving as the infective stage of the vertebrate host.

Microgamete The smaller of the two mature sexual cells, usually the male, that unite to produce a zygote in organisms that produce two types of gametes.

Microgametocyte A gametocyte that gives rise to microgametes.

Microsporidia (-an) Parasitic unicellular fungi, formerly considered to be protozoans, that infect insects, crustaceans, fish, and humans; replicate in host cells by spores.

Microthrombus (-i) A tiny blood clot that can obstruct capillaries and impede blood flow.

Microtine Referring to members of the rodent subfamily Arvicolinae, family Arvicolidae, that includes voles, lemmings, and muskrats; this subfamily sometimes called Microtinae.

Microtrichium (-ia) A minute hairlike structure, typically found on the wings of Diptera.

Microvillus (-i) A microscopic fingerlike or hairlike projection on the surface of an epithelial cell.

Miliary The presence of skin nodules that resemble millet seeds.

Miltogrammine Representatives of the subfamily Miltogramminae within the dipteran family Sarcophagidae.

Minimum infection rate (MIR) a relative measure of infection prevalence based on pools of vectors; No. of positive pools/total specimens tested/unit of time $\times 100$ or $\times 1000$.

Mode of action How a particular drug or compound, such as an insecticide, works; the specific biochemical interactions that produce the resultant effect; also called mechanism of action.

Molossid Members of the bat family Molossidae, called free-tailed bats and mastiffs.

Molt The act or process of forming a new integument and shedding the old one; in arthropods, shedding the old cuticle; in vertebrates shedding hair, skin, horn, or feathers; also used as a verb; *see* moult and ecdysis.

Monocyte A large, circulating white blood cell, produced in bone marrow and the spleen, that engulfs foreign particles and cell debris.

Monograph A treatise or detailed scholarly document on a particular subject, usually in the form of a book.

Monogyny (-ous) In social insects, having only one functioning queen in a colony.

Monophyletic Relating to a taxonomic group of organisms all descended from a single common ancestor.

Monospecific A taxonomic group, such as a family or genus, represented by a single species; in immunology, meaning specific for a single antigen or receptor site on an antigen.

Monotypic Having only one representative; e.g., a monotypic genus with only a single species.

Morbidity A state of being diseased; the incidence of a disease in a specific population or geographical area.

Moribund In a dying state; near death.

Morphogenesis (-tic) The formation or development of structural features of an organism, including tissues and organs.

Mortality The relative frequency of deaths in a population; death rate.

Mortuary A temporary place for keeping dead bodies prior to burial or cremation; e.g., funeral home, morgue.

Morula (-ae) A mass of cells, resembling a bunch of mulberries, resulting from cleavage of a zygote or ovum; characteristic of ehrlichiae inside host cells.

Mosquito coil A flat, spiral device impregnated with an insecticide, usually pyrethrum or a synthetic pyrethroid, which when burned helps to repel or kill mosquitoes and other small flying insects.

Moult Verb meaning to molt; as a noun, a British variant of molt.

Mouth hook One of a pair of clawlike or hooklike sclerites near the oral opening of muscoid fly larvae, which serve the function of mandibles.

Mucocutaneous Of or relating to the skin and mucous membranes.

Mucopurulent Containing mucus and pus.

Multifocal Relating to or arising in many locations.

Multimammate mouse Any of several African species of rodents in the genera *Praomys* or *Mastomys*, family Muridae, so-called because the females possess an unusually large number of teats; also called multimammate rat; *Mastomys natalensis* is often given the name multimammate rat.

Multiplicative transmission (propagative) Transmission of a pathogen or parasite by a vector after asexual reproduction within the vector; the form of the pathogen or parasite transmitted is indistinguishable from the form that was initially ingested by the vector.

Multivoltine Producing three or more broods or generations per year; *cf.* univoltine, bivoltine.

Mummification The formation of a desiccated, leathery, cadaver (or carrion).

Murine Relating to rodents of the subfamily Murinae of the family Muridae, including Old World and peridomestic mice and rats.

Muscid Of or belonging to the fly family Muscidae.

Muscoid Of, or belonging to, the dipteran superfamily Muscoidea.

Muscomorpha (-an) An infraorder within the dipteran suborder Brachycera that includes muscoid flies and most brachycerans; adults with short three-segmented antenna and arista; larvae with reduced head capsule; larvae form a puparium.

Mutualism (-ist, istic) An ecological or behavioral association between two species in which both benefit from the relationship.

Muzzle The forward, projecting part of the head of certain animals, including the nose, mouth, and jaws; the snout.

Myalgia Muscular pain or discomfort.

Mycetome Specialized tissues or structures in insects that harbor symbiotic microorganisms; associated with the alimentary tract, fat body, or gonads.

Mycology The biology or scientific study of fungi.

Myelitis Inflammation of the spinal cord or bone marrow.

Myenteric Of or relating to the muscular coat of the intestinal wall.

Mygalomorph Members of the taxonomic group of spiders called Mygalomorphae, represented by tarantulas and their close relatives.

Myiasis Infestation of tissues, wounds, or body cavities of live vertebrate animals by fly larvae.

Myocarditis Inflammation of the myocardium.

Myocardium (-al) The middle muscular layer of the heart wall.

Myofibroblast Cells that give rise to connective tissue (fibroblasts) and that have some structural and functional characteristics of smooth muscle cells.

Myxomatosis An infectious, usually fatal, viral disease of rabbits; characterized by benign skin tumors composed of connective tissue embedded in mucus, called myxomas.

Nagana Highly fatal disease of domestic animals in tropical Africa caused by flagellate protozoans of the genus *Trypanosoma* and transmitted by tsetse and other biting flies.

Naive In reference to animals, not previously having been exposed to a disease agent, or not previously having been used in a scientific experiment; also naïve.

Nasal fossa (-ae) One of the two halves of the nasal cavity, between the roof of the mouth and floor of the cranium.

Nasopharynx (-geal) The part of the pharynx behind the nose and above the soft palate, continuous with the nasal passages.

Natural immunity The normal antipathogen and antiparasitic activity of all animals, both humoral and cellular.

Nearctic Region The biogeographic area of North America characterized by temperate climate, flora, and fauna (New World); together with the Palearctic Region (Old World) comprises the Holarctic Region.

Necrophagous Feeding on carrion, including corpses and other dead animals.

Necrophilous Drawn to, or feeding on, dead animal or human tissue.

Necropsy Examination of a body, especially of animals, after death, often to determine cause of death.

Necrosis (-otic) Localized death of cells or tissues due to injury or disease; causes include impaired blood supply, infection, and trauma.

Nectary A glandular organ or structure of plants that secretes nectar; can be within a flower (floral) or outside a flower (extrafloral).

Nematoceran A member of the suborder Nematocera (order Diptera); adults with multisegmented antennae, and larvae with a well-developed head capsule and toothed or brushlike mandibles that move laterally.

Neosomy (-ic) Radical intrastadial growth and subsequent gross body changes of certain ectoparasites or subdermal parasites; e.g., female chigoe fleas (*Tunga penetrans)* and females of streblid batflies belonging to the genus *Ascodipteron.*

Neoteny (-ic) The retention of larval or immature structures or traits in adults of a given species.

Neotropical Region The tropical area of the New World extending south from the deserts of Mexico through Central America into South America.

Nettle (-ling) Any of many plants in the genus *Urtica* (family Urticaceae) having stinging hairs that on contact cause skin irritation; also used to refer to other plants that cause similar urticaria.

Neuritis Inflammation of a nerve or group of nerves, characterized by pain, which can lead to loss of function and degeneration of associated muscles.

Neurologic (-cal) Pertaining to the nervous system or associated disorders.

Neuromyopathy (-ic) A disease or disorder affecting nerves and associated muscle tissue.

Neurosis A disorder of the mind and thought processes without evidence of disease or structural change in the nerves or central nervous system.

Neurotoxin (-ic) A toxin that affects nerve cells or tissues.

Neurotropic Having an affinity for, or moving or growing toward, nerve tissue.

Neutrophil (-ic) A phagocytic white blood cell with an abundance of granules in the cytoplasm that are readily stained by neutral dyes.

New World A biogeographical term implying the Americas (North, Central, and South) and the Caribbean islands.

Nidicolous Living in the nest of another animal species; e.g., nidicolous insects or mites.

Nit The egg of a louse.

Nodule A small, rounded, usually firm or hard mass of body tissue; a node or knot.

Noradrenaline A catecholamine precursor of epinephrine secreted by the adrenal medulla and by nerve endings of the sympathetic nervous system; causes vasoconstriction, increased blood pressure and heart rate, and elevated sugar level in the blood; also called norepinephrine.

Notopleura (-al) A more-or-less triangular area of the insect thorax, notably, in Diptera, where the notum and pleuron join above the second pair of legs; may be enlarged as a lobe.

Nuchal (e.g., ligament) pertaining to the neck or nape; e.g., nuchal ligament.

Nullipar (-ous) A female that has not yet produced eggs or young.

Nuptial flight Reproductive behavior among most ants and some bees in which a virgin queen mates with one or more males, before seeking a suitable site to begin a new colony.

Nymph The immature stage of certain insects that resembles the adult except for its smaller size (e.g., lice) and, in the case of pterous species, the lack of functional wings (e.g., cockroaches): also, an immature stage of mites, including ticks, with four pairs of legs.

Obligate Adj. required, necessary, or essential; e.g., obligate parasite, a parasite that requires a host in order to complete its development (as opposed to facultative); as in obligate autogeny, obligate hematophagy, etc.

Obtect Having the antennae, legs, and wings embedded in a secretion that forms a hard cover or protective case, as in obtect pupae characteristic of butterflies and moths.

Occult Concealed, not apparent, obscure; as in an occult infection.

Ocellus (-i) A simple eye in insects, which is sensitive to light and changes in light intensity, but does not form a visual image.

OCP Onchocerciasis Control Programme, in West Africa, under the auspices of the World Health Organization.

Octenol A compound found naturally in ox breath that is attractive to certain host-seeking flies; used to enhance the attractiveness of various traps for collecting biting flies, such as mosquitoes, biting midges, and tsetse flies.

Ocular point A cuticular projection posterior to the antenna in lice lacking eyes, situated where eyes are located in some other species.

Odonate A member of the insect order Odonata, the dragonflies and damselflies.

Odor plume A mass of air moving downwind from an animal, with higher temperature, humidity, and carbon dioxide levels than that of the ambient surroundings; provides indirect clues to the presence of a potential host.

Oedemerid A member of the family Oedemeridae, false blister beetles.

Oestrid A member of the family Oestridae, bot flies and warble flies.

Oiler In pest control, a self-applicating appliance against which livestock rub and become coated with an oil-based pesticide formulation.

Old World A biogeographical term encompassing Europe, Asia and Africa

Olfaction Sense of smell, or act of smelling.

Oligochaete Terrestrial and aquatic annelid worms of the class Oligochaeta, with tiny bristles located singly along their length; e.g., earthworms.

Oligonucleotide A short nucleic-acid chain with less than 20 bases.

Omnivorous Feeding on both plants and animals.

Onchocerciasis An infestation with filarial worms of the genus *Onchocerca.*

Onchocercid Common name for filarial worms of the family Onchocercidae.

Onomatopoetic Formation or use of words that imitate a natural sound associated with the object or action to which it refers.

Oocyst A thick-walled structure that surrounds the developing zygote in the life cycle of certain sporozoan parasites; e.g., *Plasmodium* spp.

Oocyte A cell in the ovary of female animals from which an egg, or ovum, develops by meiosis.

Oogenesis The process by which an ovum is formed and develops to maturity.

Ookinete A motile zygote in the life cycle of certain sporozoan parasites; e.g., *Plasmodium*, which penetrates the stomach wall of a mosquito and forms an oocyst.

Ootheca (-ae) A case or capsule enclosing the eggs of certain insects; e.g., cockroaches.

Operculum (-a) A lidlike part of the insect egg, usually at the anterior end, that opens to allow the insect to emerge, e.g., louse egg; also a similar structure in puparia of certain flies that is pushed open to facilitate adult emergence.

Ophthalmia(-ic) Inflammation of the eye, usually involving the conjunctiva.

Opisthosoma That portion of the arachnid body (idiosoma) posterior to the hindpair of legs.

Orbivirus A reovirus of the genus *Orbivirus*; includes the viruses that cause bluetongue disease and Colorado tick fever.

Orf A contagious pustular dermatitis in sheep and goats caused by the orf virus; primarily affects lambs and can be transmitted to humans, causing a pustular lesion.

Organochlorine A chlorinated hydrocarbon, most commonly used to refer to pesticides such as DDT, aldrin, or dieldrin.

Organophosphate An organic compound containing phosphorus; used as an insecticide that acts as a neurotoxin.

Oriental Region A biogeographical region roughly corresponding to Southeast Asia and some adjacent islands such as Sumatra, Java, and Borneo.

Ornithonosis (es) A disease of birds that is occasionally transmitted to humans.

Ornithophagic Feeding on birds.

Ornithophily (-ic) Attracted to birds.

Orthopteroid An insect superorder including Orthoptera and related insect orders: Phasmatodea, Grylloblattaria, Mantophasmatodea, Mantodea, Blattaria, Isoptera, Dermaptera, and Embiidina (Embioptera).

Ostium (-a) A small opening in a tubular organ or other anatomical structure; e.g., insect heart.

Otitis media Inflammation of the middle ear, behind the eardrum.

Otitis Inflammation of the ear.

Otoacariasis Infestation of the ear by mites; also, otacariasis.

Otoscope (-ic) An instrument for visually examining the external ear canal and eardrum.

Ovarian follicle A cell aggregation in the ovary (or ovaries) in which eggs develop and from which they are released.

Ovariole One of the multiple tubes that form the ovary in insects.

Ovate Egg-shaped or oval in outline.

Overwinter To pass and survive the colder months of the year.

Oviduct A tube through which an egg passes from the ovary.

Ovigerous Bearing eggs; gravid.

Oviparous (-ity) Producing eggs that hatch outside the body.

Oviposit (-tion) To deposit or lay eggs.

Ovipositor The egg-laying structure of female insects formed by modifications of the eighth and ninth abdominal segments; also the egg-laying tube through which an egg passes when deposited.

Ovoviviparous (-ity) Producing live young from eggs that hatch within the female body; common in some insects; e.g., cockroaches.

Pacific region A biogeographical region that includes land masses in, and adjacent to, the Pacific Ocean.

Paederine Referring to beetles of the subfamily Paederinae, particularly members of the genus *Paederus* and closely related species (family Staphylinidae).

Palatal brush A group of hairlike filaments of the palatum (oral surfaces of the labrum and clypeus) in larvae of nematocerous flies; e.g., mouth brushes of mosquito larvae.

Palearctic Region A biogeographical region that includes most of Eurasia and Africa north of the Sahara.

Paleotropics Tropical areas of the Palearctic region.

Palmate Shaped similar to a hand with the fingers extended; e.g., palmate float hairs on abdominal segments of *Anopheles* mosquito larvae.

Palp or Palpus (-i) A segmented appendage associated with the mouthparts of insects, arachnids, and other invertebrates; with sensory receptors for tactile and chemical stimuli.

Palpable Capable of being perceived or felt, especially by touch.

Palpitate (-tion) To beat rapidly, pulsate, or throb; e.g., heart palpitation.

Palpomere A subdivision of a palpal segment; not a true segment.

Pancytopenia An abnormal reduction in the number of red blood cells, white blood cells, and platelets in the blood; usually due to disease of the bone marrow.

Pandemic A human disease or other affliction occurring at above the normal incidence over a wide geographic area, such as a country, continent, or the whole world; a widespread or global epidemic; also an adjective.

Papilla (-ae, -ate) A small nipple-like protuberance or projection; also a term applied to more elongate or leaflike projections of the surface of an organism, as in anal papillae of mosquito and blackfly larvae.

Papilloma A benign epithelial tumor of the skin or mucous membranes.

Papule (-ular) A small, solid, usually inflamed elevation of the skin that does not contain pus.

Papulonodular Referring to a skin condition with both papules and nodules.

Papulovesicle (-ular) A papule that changes to a vesicle or blister.

Paragenital sinus A notch in the posterior margin of the fifth abdominal sclerite of female cimicid bugs that leads via a slit into the sperm-receiving structure.

Paramere A lateral lobe or process at the base of the phallus, or intromittent organ, of male insects.

Parasite An organism that lives in or on another species, the host, and from which it derives nourishment and protection at the expense of the host.

Parasitemia (-ic) Presence of parasites in the blood.

Parasiticide A chemical, other agent, or preparation that kills parasites.

Parasitoid An insect that as a larva develops within the body of another insect, consuming its tissues and eventually killing it; e.g., pteromalid wasps and tachinid flies; used as biological control agents.

Parasitosis (-es) An infestation with parasites, or disease resulting from a parasitic infestation.

Parasympathetic Pertaining to the nerves and ganglia of the autonomic nervous system that arise from the cranial and sacral regions; control involuntary functions such as pupil constriction, heart rate, and dilation of blood vessels.

Parenchyma (-al) The principal tissues of an organ, as distinct from associated connective or supporting tissues.

Parity rate Proportion of parous females per number of females examined.

Parous Having given birth, or in the case of invertebrates having deposited eggs, at least once.

Paroxysm A sudden attack, recurrence, or intensification of disease symptoms.

Parthenogenesis (-tic) A type of reproduction in which the egg develops without fertilization.

Parturition The act or process of giving birth to offspring.

Passeriform Pertaining to birds of the order Passeriformes; passerine.

Passerine (See Passeriform).

Pastern That part of the equine foot from the fetlock to the top of the hoof.

Patas monkey *Erythrocebus patas*, an African ground-dwelling monkey.

Patella The knee cap of vertebrates; in arachnids, the leg segment between the femur and tibia; genu.

Pathogen A microorganism, virus, fungus, or other agent capable of causing disease.

Pathogenesis The origination and development of a disease.

Pathogenic Capable of producing disease.

Pathologic Pertaining to pathology, the nature and cause of disease; pathological.

Paurometabolous A form of development in insects in which the immature stages and adults resemble one another and both live in the same habitat; e.g., cockroaches and lice.

PCR (*See* polymerase chain reaction).

Pecten (-tines) A comblike structure or row of teeth; e.g., on the respiratory tube of mosquito larvae, and pecten of scorpions.

Pectinal tooth The individual toothlike structure that forms a pecten.

Pectinate Having closely parallel, toothlike projections, comblike; e.g., pectinate antennae

Pedal Pertaining to the leg or foot

Pedicel The second basal segment of the insect antenna between the scape and flagellum, bearing Johnston's organ; a slender stalk or stemlike structure serving for support; *see* petiole

Pediculicide A chemical or other agent used to kill lice.

Pediculosis A human or animal infestation with lice.

Pedipalp (-us, -i) One of the second pair of appendages near the mouth of arachnids immediately posterior to the chelicerae; homologous to mandibles of insects.

Pelage Hair, fur, wool, or other soft covering that forms the coat of a mammal.

Peliosis (-es) Any of several blood diseases that cause subcutaneous bleeding marked by purple patches on skin or mucous membranes; purpura.

Penicillin Any of several broad-spectrum antibiotics produced by molds of the genus *Penicillium*; also produced synthetically; most active against gram-positive bacteria.

Pentastomid A member of the Pentastomida, wormlike invertebrates with two pairs of hooks near the mouth; obligate parasites in the respiratory tract of reptiles, birds, and mammals; called tongue worms, due to their resemblance to a vertebrate tongue.

Penultimate Next to the last.

Peptide A compound formed by two or more amino acids linked by the carboxyl group of one amino acid to the amino group of another.

Per oral Via the oral cavity or mouth; e.g., per oral infection: also, per os.

Peracute Very acute or violent.

Perennial Lasting or remaining active on a continual basis from 1 year to the next.

Perianal gland Glands near or around the anus.

Pericardium (-al) The membranous sac that encloses the heart and the origins of the aorta and other large blood vessels in vertebrate animals.

Peridomestic Found around or in proximity to human dwellings or habitation.

Perineum (-al) The general area between the anus and genital organs of vertebrate animals.

Periodic Recurring at regular or irregular intervals of time.

Periodicity A recurrence at regular intervals of time.

Perioral Surrounding the mouth or involving tissues around the mouth.

Periorbital Involving or located around the orbit of the eye.

Peripheral blood Blood circulating in arteries, veins, and capillaries near the general body surface and in the extremities.

Peripylarian Growth or development of a parasite or pathogen in the hindgut of a vector, as in members of the protozoan subgenus *Viannia* of the genus *Leishmania*; as opposed to suprapylarian, which implies growth or development mainly in the midgut.

Perissodactyla An order of nonruminant grazing ungulates with an odd number of toes on each hoof; includes horses, tapirs, and rhinoceroses.

Peritoneum (-al) The transparent serous membrane lining the abdominal cavity and enclosing most of the viscera.

Peritonitis Inflammation of the peritoneum.

Peritreme A sclerotized area of the body wall in insects surrounding a spiracle; in mites, a sclerotized groove in the body wall leading to a spiracle, or stigma.

Peritrophic Pertaining to a delicate matrix that forms a cylindrical envelope surrounding food in the insect midgut; produced by the midgut wall.

Perivascular Surrounding a vessel, especially a blood vessel.

Permethrin A synthetic pyrethroid compound used as an insecticide, acaricide, and insect repellent; acts as a neurotoxin.

Personal protectant A compound or other substance that, when applied to the skin or clothing, serves to reduce the attractiveness of an individual to annoying or biting insects, mites, or ticks; e.g., insect repellents.

Petechia (-ae, -al) Small reddish or purplish spots on the skin or mucous membranes caused by hemorrhaging of capillaries.

Petiole A slender stalk or stem; e.g., the constriction between the thorax and abdomen of wasps and ants; *see* pedicel.

Phagocyte (-ic) A cell, such as a macrophage, that engulfs and destroys microorganisms and other foreign bodies in the blood or other body tissues.

Phagolysosome A vesicle formed within a cell by an ingested particle (phagosome) and a lysosome containing hydrolytic enzymes.

Phagostimulant A compound or other substance in plants or animals that induces an organism to feed.

Pharate A term referring to a fully formed insect or mite while still enclosed within the old separated cuticle of the previous developmental stage; an instar between apolysis and ecdysis.

Pharyngeal pump The sucking mechanism of fluid-feeding invertebrates involving the pharyngeal musculature.

Pharyngitis Inflammation of the pharynx, typically in vertebrates.

Pharynx (-geal) In invertebrates, the anterior part of the foregut extending from the mouth to the esophagus.

Pheromone A chemical substance released by an animal that triggers, or otherwise influences, the behavior or physiology of other members of the same species.

Phlebotomine Referring to members of the psychodid subfamily Phlebotominae, commonly called sand flies.

Phlebovirus A genus of viruses of the family Bunyaviridae, including causative agents of sand-fly fever and Rift Valley fever.

Phlegm Thick mucus secreted in the lungs and respiratory passages, caused by infection.

Phobia A persistent, abnormal, and irrational fear of, or aversion for, a specific object or situation.

Phoresy (-tic) A commensal relationship between two species in which one is carried on the other, typically as a means of dispersal or escaping adverse environmental conditions.

Phospholipase An enzyme that catalyzes the breakdown of phospholipids, by hydrolysis of ester bonds.

Photoperiod The duration of exposure to light by an organism in a daily 24-h cycle; also used to refer to the durations of light and darkness during a 24-h period.

Photophobia An unusual sensitivity to or intolerance of light; an abnormal fear of light.

Phylogeny (-etic) The development of taxonomic groupings and relationships among organisms based on comparative and evolutionary studies; the evolutionary history of groups of organisms.

Physiological age grading Determining the reproductive state of adult organisms, e.g., the number of times a female insect has produced eggs or oviposited; based on examination of the reproductive organs.

Phytophagous Feeding on plants.

Piloerection The raising or bristling of hairs due to reflex contraction of tiny muscles at the base of the hair pulling the hair erect; goose bumps or goose flesh.

Pilus dentilus A short, stout seta at the base of the fixed, or unmovable, digit of the chelicerae in gamasid mites.

Pinkeye Acute, contagious conjunctivitis in humans and other animals usually due to infection by bacteria or viruses; bacterial agents include *Hemophilus aegyptius* in humans and *Moraxella bovis* in cattle.

Pinna (-ae) The outer, visibly projecting, cartilaginous structure of the ear in vertebrates.

Pinworm Any of several small nematodes of the family Oxyuridae parasitic in humans, horses, rabbits, and other mammals; infest the intestines and rectum; e.g., *Enterobius vermicularis* of humans.

Piroplasm Parasitic sporozoans of the family Babesiidae that infect red blood cells of mammals such as humans, cattle, sheep, and dogs; includes *Babesia* and *Theileria* spp.

Piroplasmosis Infection with protozoans of the family Babesiidae, including the tick-borne bovine diseases Texas cattle fever and East Coast fever; e.g., babesiosis, theileriosis.

Pityriasis Any of various skin diseases characterized by the shedding of dry, flaky, or branlike epidermal scales.

Plaque (1) in pathology, any of various small patches or disk-shaped formations on the skin or surface of mucous tissues; (2) a deposit of fatty material on the inner wall of an artery; (3) an area on a cell culture where cells have been killed by a pathogen being screened (plaque assay).

Plasmatocyte A type of phagocytic cell in the hemolymph of arthropods characterized by relatively large size, irregular outline, and basophilic cytoplasm.

Platelet Round or oval disklike cytoplasmic bodies in vertebrate blood, lacking a nucleus and hemoglobin; produced in bone marrow and functioning in blood clotting.

Platyform Platelike in shape.

Pleomorphic Occurrence of two or more structurally distinct forms of a developmental stage in the life cycle of an organism; polymorphism.

Pleural sclerite A small or minor sclerotized plate in the pleural region; pleurite.

Pleuron (-al) The lateral area of a body segment in arthropods, usually referring to the thorax and abdomen; may or may not be hardened, or sclerotized.

Plumose Resembling a plume or feather.

Pneumonia Inflammation of the lung or lungs, usually caused by viruses, bacteria, fungi, or chemical irritants; pneumonitis.

Pneumonic (-ic) Involving the lungs or pneumonia.

Pneumonitis Inflammation of the lung or lungs; pneumonia.

Podosoma The anterior part of the main body region of mites and ticks bearing the legs.

Poikilotherm (-ic) An animal in which the body temperature varies with the temperature of its environment.

Poison Any substance that, when taken into the body, interferes with normal physiological functions; a general term that includes toxins and venoms.

Poliomyelitis Inflammation of the gray matter of the spinal cord and brain stem; a viral disease caused by polioviruses and leading to paralysis, muscular atrophy, and often deformities; polio.

Poll In livestock, the prominent hairy top or back of the head.

Polyamine An organic compound with two or more amino groups.

Polyarthritis Inflammation of multiple joints simultaneously.

Polyctenid A member of the hemipteran family Polyctenidae, ectoparasitic on bats; bat bugs.

Polygenic Pertaining to inheritable characters controlled or caused by interaction of multiple genes.

Polygyny (-ous) In social insects, the presence of more than one functional queen in a colony at the same time; reproductive behavior of animals in which a male mates with more than one female.

Polymerase chain reaction (PCR) A technique for amplifying sequences of DNA; involves separating DNA into two strands and incubating it with oligonucleotide primers and DNA; can be modified for amplifying RNA sequences.

Polymorph (-ic) An organism or structure represented by two or more forms

Polypeptide A molecular chain of 10 to more than 100 amino acids linked together by peptide bonds

Polyphaga (-an) The largest suborder of Coleoptera; includes more than 90% of the described species of beetles

Polytene chromosome A giant chromosome with characteristic dark and light banding patterns, formed by multiple replications of DNA strands that remain tightly together within the cell.

Polytrophic ovariole An ovariole containing a primary oocyte and associated trophocytes, or nutritive cells, all derived from the same female germ cell.

Polyvalent vaccine A vaccine prepared from cultures of two or more strains or species of pathogens, thereby providing protection from more than one pathogenic agent with a single vaccine; multivalent vaccine.

Pool feeder An ectoparasitic or other blood-feeding organism that lacerates the skin of a host with its mouthparts causing blood or other tissue fluids to accumulate at the bite site, which then are drawn into the oral cavity and alimentary tract; telmophagy; *cf.* capillary feeder or solenophagy.

Pool In vector biology, a sample of insects, usually representing a single species, which is prepared for testing to detect evidence of an infectious or other agent.

Porcine Pertaining to or resembling swine or pigs.

Porose area A cluster of tiny pores on the dorsal aspect of the basis capituli of female hard ticks (family Ixodidae), through which are secreted antioxidants that prevent breakdown of waxy compounds protecting the deposited eggs.

Postabdomen The posterior part of the opisthosoma of scorpions that forms the slender, flexible tail terminating in the telson and sting; metasoma.

Posterior-station Referring to the posterior part of the alimentary tract in arthropods; e.g., posterior-station transmission of pathogens, i.e., pathogens passing from an arthropod host via the anus as a source of infection; stercorarian transmission.

Postgena (-al) The portion of the insect head capsule immediately posterior to the gena.

Postgenital Posterior to the genitalia; e.g., postgenital segments.

Postmortem Occurring after death.

Postpartum Occurring after childbirth.

Postscutellum Apparent in some insects as the upper, posteriorly produced, part of the metanotum.

Pour-on A liquid formulation of an insecticide or acaricide that typically is applied by pouring it along the midline of animal's back.

Preabdomen In scorpions, the anterior seven segments of the opisthosma (mesosoma), behind which the opistosoma is modified to form the tail (metasoma); in insects, the relatively unmodified anterior abdominal segments.

Predispose (-ed, -ition) To incline or tend toward something beforehand; to make susceptible, as to a disease.

Prediuresis In hematophagous insects, the separation of liquid components from a bloodmeal in the midgut which are passed directly to the hindgut and excreted as droplets of fluid from the anus, often during feeding.

Prehensile Modified or adapted for seizing, grasping, or holding onto something.

Prelarva(-ae) A nonfeeding developmental stage in certain mites and other arachnids immediately preceding the larval stage.

Prenatal Existing or occurring prior to birth.

Prepupa The last part of the final larval instar in holometabolous insects in which the larva ceases to feed, defecates, and becomes quiescent in preparation for transformation to the pupa.

Preputial gland A sebaceous gland associated with the foreskin of the male penis, the prepuce, and similarly, the fold of skin covering the tip of the female clitoris of mammals.

Prestomal teeth Sclerotized teeth arising along the lateral margins of the prestomum, or on the inner wall of the prestomum, of some adult flies; used for scraping food, or lacerating skin in the case of certain hematophagous flies.

Prestomum (-a) A cleft between the two lobes of the labellum just anterior to the opening to the food canal of the adult stage of certain flies.

Presumptive Based on reasonably convincing, albeit unconfirmed, evidence; e.g., a presumptive case or presumptive diagnosis.

Pretarsus The distal-most part of the tarsus in insects, arachnids, and other arthropods bearing the claws, empodium, or other terminal appendages.

Prevalence The number of cases of something in a population at a given time; also the percentage of a population affected by a particular cause at a specific time; e.g., prevalence of infection or disease.

Priapism A sustained, usually painful erection of the penis not related to sexual arousal.

Primary host A host in which a virus or microorganism most commonly replicates or reproduces; in the case of multicellular parasites, a host in which the organism reaches maturity and reproduces sexually; *syn.* definitive host.

Primary vector An arthropod that plays a major role in transmission of a pathogen or parasite from one vertebrate host to another.

Primer A segment of DNA or RNA that is complementary to a given DNA sequence and that is needed to initiate replication by DNA polymerase; as in PCR.

Privy An outdoor toilet; outhouse.

Proboscidea A mammalian order represented by the single family Elephantidae, elephants, with only three living (extant) species; includes the extinct mastodons and mammoths.

Proboscis In insects and other arthropods, an elongation of the anterior or ventral part of the head composed of the mouthparts and associated structures, typically forming a tube through which food is drawn into mouth and alimentary tract.

Progesterone A female sex hormone that prepares the uterus for implantation of a fertilized ovum, helps maintain pregnancy, and promotes development of mammary glands.

Proglottid A segment of an adult tapeworm containing a completely sexually mature reproductive system, with both male and female reproductive organs; able to survive briefly after breaking away.

Prognathus With the head directed forward, especially in insects.

Prognosis A prediction of the probable course and outcome of a disease, including the likelihood of recovery.

Proleg A process or appendage of insect larvae that serves the purpose of a leg; usually fleshy and unsegmented; *syn.* pseudopod.

Promastigote A flagellate stage of protozoans in the family Trypanosomatidae, e.g., *Leishmania* sp.; characterized by a single anterior flagellum, without an undulating membrane.

Pronghorn *Antilocapra americana*, family Antilocapridae; an antelope-like ruminant with short forked horns found on the western plains of North America.

Pronotum The dorsal surface of the first thoracic segment of insects.

Propagative transmission Transmission of the same form of a pathogen or parasite by a vector in the form as it was originally ingested from an infected host; occurs after asexual reproduction of the pathogen or parasite in the vector; *syn*, multiplicative transmission.

Prophylaxis (-actic) Protective treatment or prevention of disease.

Propodeum The first abdominal segment in hymenopteran adults that has become morphologically part of the thorax.

Propodosoma That part of the body of mites and ticks bearing the first two pairs of legs.

Proprioceptor (-ive) A receptor that responds to stimuli originating within the body, such as pressure, stretch, and position.

Prosoma In mites and ticks, the anterior part of the body including the mouthparts (gnathosoma) and leg-bearing region (podosoma); in other arachnids, the combined head and thorax segments, or cephalothorax.

Prosternum (-al) The anterior-most sternal sclerite of insects, between the forelegs; prothoracic sternum.

Prostigmatid Referring to members of the mite suborder Prostigmata.

Prostrate (-tion) Lying flat or stretched out at full length.

Prostriata (-ate) Hard ticks (family Ixodidae) in which the anal groove curves anteriorly around the anus; includes members of only one genus, *Ixodes.*

Proteolysis (-tic) Breaking down of proteins into simpler compounds.

Prothorax (-ic) The anterior-most thoracic segment of insects, bearing the first pair of legs.

Protist A very diverse group of eukaryote organisms usually referred to as members of the kingdom Protista or Protoctista; includes unicellular or multicellular eukaryotes without highly specialized tissue, i.e., distinct from fungi, plants, or animals.

Protonymph The first nymphal stage in the generalized life history of mites and some other arachnids; often a suppressed developmental stage passed within the egg.

Proventriculus (-ar) A typically muscular part of the posterior foregut of insects and other arthropods located just anterior to the constriction leading into the midgut, or ventriculus; in birds, a glandular part of the stomach in which food is partially digested before passing from the crop to the ventriculus, or gizzard.

Pruritus An intense itching sensation.

Psammophore Setae on the ventral surface of the head and mandibles of certain desert-dwelling ants and wasps, forming a basket in which sand or dry soil is moved or transported.

Pseudopenis The term pseudopenis is typically used for the male intromittent organ of lice (order Phthiraptera) and some related insects; *see* aedeagus.

Pseudopod (-ium, -ia) *See* proleg.

Pseudotrachea (-ae) A structure having the aspect of a trachea, in the labellum of Diptera; pseudotracheae are ringed and ridged grooves used for scraping food.

Psoriasis A chronic noncontagious inflammatory skin disease characterized by discrete patches of pink or dull-red lesions, often covered with silvery scales.

Pteridine An organic yellow crystalline compound composed of a linked pyrimidine and pyrazine ring; includes structural constituents of various animal pigments.

Pteromalid A tiny parasitic wasp of the large chalcidoid family Pteromalidae; includes common parasitoids of the puparia of dung flies.

Ptilinal suture The frontal suture through which the ptilinum is everted during and immediately following emergence of a teneral adult fly from a puparium.

Ptilinum (-al) A saclike, inflatable cuticular structure of the head of some adult flies; used to facilitate emergence from the puparium or to burrow through soil to reach the surface.

Pubescence (-ent) A covering of short, closely spaced, fine hairs or setae.

Pubic Referring to the lowermost part of the abdomen between the thighs, overlying the pubes, or pubic bones.

Pulmonary Relating to or affecting the lungs.

Pulvillus (-i) A padlike structure or lobe of the pretarsus at the base of the tarsal claw.

Pupa (-ae) The quiescent developmental stage between the larva and adult of holometabolous insects; sometimes called a chrysalis or chrysalid for members of the Lepidoptera.

Puparium (-ia) The thickened, hardened integument of third-instar larvae of cyclorraphous flies within which the pupa and adult are formed.

Pupiparous Referring to an insect in which the larva develops within the body of the parent female and is ready to pupate at the time it is deposited; e.g., hippoboscids and tsetse flies.

Purge To evacuate the bowels, or something that causes evacuation of the bowels.

Purpura pulicosa Small, purplish spots on the skin resulting from flea bites and associated capillary leakage.

Purpura Hemorrhaging in the skin, mucous membranes, or serosal tissue resulting in purplish spots or patches.

Purulent Forming, containing, or discharging pus.

Pus A generally viscous yellowish-white fluid resulting from inflammation; consisting of plasma, white blood cells, cellular debris, and necrotic tissue.

Pustule A small inflamed swelling of the skin filled with pus; a pimple or pus-filled blister.

Putative Commonly believed to be true without conclusive evidence; supposed, reputed.

Putrid (putrifaction) Animal or plant material in an advanced state of decomposition, characterized by a foul odor; rotten.

Pygidial gland Any of various glands that open near the pygidium or anus; common in bees, wasps, ants, and beetles.

Pygidium (-al) The tergum, or dorsal part, of the last abdominal segment of insects and other invertebrates.

Pyloric valve A valvelike fold at the juncture of the midgut and hindgut of insects.

Pylorus The anterior part of the hindgut in insects, usually including the pyloric valve; functionally forms the posterior end of the midgut.

Pyoderma gangrenosum A chronic skin disease, characterized by purplish nodules, pustules, and spreading ulcers, usually on the trunk.

Pyoderma An acute destructive inflammation of the skin characterized by pus-filled lesions.

Pyogenic Producing or relating to formation of pus.

Pyrethrin Either of two esters extracted from the seed cases of chrysanthemum flowers, used as an insecticide.

Pyrethroid A synthetic chemical compound structurally related to natural pyrethrin, widely used in commercial products for insect control; e.g., permethrin, resmethrin.

Pyriform Pear-shaped.

Pyriproxyfen A pyridine-based pesticide and juvenile hormone analog that prevents certain insects and other arthropods from developing to adults, thereby precluding reproduction; e.g., methoprene.

Quartan Occurring every fourth day, usually with reference to fever; e.g., quartan malaria.

Queen excluder A barrier inside a beehive that prevents queens from entering or exiting the colony while allowing the smaller workers to come and go; typically a perforated plastic or metal sheet or a wire grid.

Quest (-ing) Host-seeking behavior of ticks, which, in most cases, involves a tick crawling upwards on vegetation and awaiting contact with a potential host when it passes by; the forelegs are held widely extended, allowing the tick to grasp immediately on contact.

Quiescence (-ent) A general term referring to an inactive, dormant, or latent period in the life or development of an organism; many different levels of physiological activity represented.

Quinone Any of a class of naturally occurring aromatic compounds, characterized by two carbonyl groups in an unsaturated benzene ring; *syn.* benzoquinone.

R_1 cell Cell of the insect wing bounded anteriorly by the first radial vein, R_1.

Radioallergosorbent test (RAST) An IgE-mediated allergy test; involves incubating serum from a blood sample with a solid-phase antigen and quantifying the amount of allergen-specific IgE using radiolabeled anti-IgE.

Radius (-al) The third longitudinal vein from the anterior margin of the insect wing, behind the costa and subcosta.

Raptorial Modified or adapted for seizing prey.

Receptor A specialized group of nerve endings or a sensory structure for perceiving stimuli; sense organ; particularly concentrated on the antennae, palps, and tarsi of insects.

Recrudesce (-scent, -scence) To assume renewed activity after a dormant or inactive period; e.g., recurrent symptoms or relapses of a disease.

Recumbent Lying down; resting or inactive.

Red bug A colloquial name for a chigger.

Reflex bleeding The release of hemolymph through the intersegmental membrane of an appendage or other body part of certain insects and other arthropods; usually involves distasteful or toxic chemicals in the hemolymph that serve a defensive function against potential predators.

Refractory Resistant to normal treatment, stimulation, or infection.

Refugium (-a) An area to which an organism can retreat or where it can continue to survive after otherwise being eliminated from the surrounding environment.

Relapse Recurrence of a disease or symptoms after apparent recovery.

Relapsing fever Any of several infectious diseases of humans caused by spirochetes transmitted by lice and ticks, characterized by intermittent attacks of high fever.

Relative abundance A number or index value indicating a comparative estimate of the number of organisms in one area versus another, without knowing the actual or absolute number of individuals present; *cf.* absolute abundance.

Repellent A substance that causes behavioral aversion by an organism; usually a chemical that is distasteful or otherwise offensive, or alters the organism's behavior by blocking or disrupting its sensory receptors.

Replete (-tion) Filled to satiation or satisfaction with food or drink; fully engorged, as in a mosquito or other hematophagous arthropod after taking a bloodmeal.

Reproductive diapause A specific type of diapause in which reproductive development or activity is temporarily suspended.

Reservoir An organism that supports survival and development of a pathogen or parasite typically without experiencing apparent adverse effects; functions in maintaining pathogens or parasites for an extended period of time and as a source of infection for other organisms in the transmission cycle.

Residue (-ual) In an entomological context, components of a chemical formulation that remain for an extended period of time on a surface following a pesticide application.

Resilin A cuticular protein with elastic properties secreted by epidermal cells of the arthropod integument; functions in storage and release of mechanical energy.

Resource partitioning An ecological concept in which a common finite resource such as food or space is shared by two or more species, each adapted to reduce direct competition among them.

Respiratory horn One of a pair of dorsolateral appendages on the cephalothorax of larvae of mosquitoes and some other flies with aquatic immature stages; usually open at the distal end to allow for gas exchange at the water surface; *syn.* respiratory trumpet, air trumpet, trumpet.

Respiratory trumpet *See* respiratory horn.

Resting site A location or microhabitat where a flying insect is typically found between periods of flight activity.

Resurge (-ence) To rise again or rebound after a decline or absence; to make a comeback, as in the case of a resurgent disease.

Reticulocyte An immature red blood cell containing a network of granules or filaments.

Reticuloendothelium (-al) Cells and tissues that make up the reticuloendothelial system, part of the immune system consisting primarily of phagocytic cells such as monocytes and macrophages concentrated in the lymph nodes, spleen, and loose connective tissue.

Retinitis Inflammation of the retina of the eye.

Rhinitis Inflammation of the nasal mucosa.

Rhinoconjunctivitis Inflammation of the conjunctiva and adjacent nasal mucosa.

Ribonucleic acid A linear polymer of nucleotides found primarily in the cytoplasm, which transfers genetic information from DNA to the cytoplasm, plays a key role in protein synthesis, and controls chemical processes in the cell; *abbrev*. RNA; also the genetic material of certain viruses.

Rickettsemia Presence of rickettsial bacteria in the blood.

Rickettsia (-ae) A member of the genus *Rickettsia*, obligate intracellular proteobacteria in the order Rickettsiales, family Rickettsiaceae, transmitted by fleas, lice, chiggers, and ticks; causative agents of various forms of typhus and of rickettsialpox.

Rickettsialpox A generally mild disease caused by *Rickettsia akari* transmitted from mice to humans by the bite of an infected mite.

Rickettsiosis (-es) Infection with a rickettsia.

Ring gland A ringlike structure of endocrine tissue encircling the aorta of muscomorphan fly larvae, representing fusion of the corpora allata, corpora cardiaca, and prothoracic glands; connected to the brain by a pair of nerves.

Ringworm An eruption of the skin caused by fungi, usually belonging to the genera *Microsporum*, *Trichophyton*, or *Epidermophyton*; can affect any part of the body but more commonly the feet, nails, and scalp.

Riparian Occurring or situated on the bank of a stream, river, or other flowing body of water.

RNA *See* Ribonucleic acid

Rock pool A natural depression in the rock bed of a stream or river forming isolated pools of water when the water level drops low enough; a temporary breeding site for certain mosquitoes and other aquatic organisms.

Rosacea A chronic skin condition of the face caused by dilation of capillaries and characterized by red or rose-colored acnelike pustular lesions; *syn.* acne rosacea, acne erythematosus.

Roseola pulicosa A small, slightly swollen, reddish lesion resulting from a flea bite.

Rostrum A general term referring to any snoutlike prolongation of the head in arthropods and other invertebrates.

Roundworm A member of the phylum Nematoda, with a cylindrical, unsegmented body usually tapered at both ends; includes parasitic filarial worms, hookworms and pinworms.

r-strategy (-ist) A reproductive pattern characterized by a high reproductive rate and low survival rate; common in arthropods and other invertebrates.

Ruminant Any even-toed, hoofed, cud-chewing, usually horned mammal of the suborder Ruminantia; with a stomach typically divided into four compartments, the anterior chamber being the rumen; includes cattle, buffalo, sheep, goats, camels, and deer.

s. l. See *sensu latu.*

s. s. See *sensu stricto.*

Saddle A large sclerite usually covering most of the dorsal and lateral surfaces of the anal segment of larvae of mosquitoes and some other nematocerous flies.

Salicylic acid A white crystalline acid derived from phenol, used in making aspirin and as a topical treatment for certain skin conditions.

Salmonellosis Infection with bacteria of the genus *Salmonella*, caused by consuming contaminated or improperly cooked foods; marked by acute onset of abdominal pain, vomiting, diarrhea, and fever; e.g., food poisoning.

Saponify (-ication) To convert into a soap, as when fats or oils chemically react with an alkali; rarely occurs in cadavers and carrion.

Saprophage (-ous, -y) Any organism that feeds on dead plant or animal matter.

Saprophyte Any organism that feeds on dead plant matter.

Satellite lesion A secondary lesion in a medical condition, in addition, to a primary lesion located at the point of injury or infection; e.g., secondary erythema migrans lesions in Lyme disease cases.

Sauria (-an) A parayphyletic group of reptiles in the class Sauropsida, order Squamata, which includes lizards, skinks, and dinosaurs; no longer accepted as a taxonomic term.

Savanna A flat grassland with scattered trees, usually in subtropical or tropical regions.

Scabies An infestation of skin by the mite *Sarcoptes scabiei* characterized by dermatitis and intense itching; occurs especially in cattle, sheep, pigs, dogs, and humans.

Scape The basal-most segment of the insect antenna.

Scarabaeid A member of the beetle family Scarabaeidae, or scarab beetles.

Scarabaeiform Grub-shaped; resembling the larvae of scarab beetles.

Scarabiasis An infestation or invasion of living animals by adult beetles.

Scarify To make scratches or small superficial cuts, as in skin or other tissues.

Scavenger An animal that feeds on dead plant or animal matter.

Schizogony Asexual reproduction by multiple fission to produce merozoites, characteristic of sporozoans, e.g., malarial parasites; *syn.* merogony.

Sciurid A member of the rodent family Sciuridae; includes ground squirrels, susliks, tree squirrels, chipmunks, woodchucks and marmots.

Sclerite A hardened or sclerotized part of the arthropod integument delineated by membranous areas, sutures, or apodemes.

Sclerotin (-ized) Any of several structural proteins that impart hardness, toughness, and usually darkened coloration to the arthropod integument; formed by cross-linkages of molecules of the protein arthropodin in the cuticle by a tanning process called sclerotization.

Scoleciasis Invasion of living animals by lepidopterous larvae.

Scopula (-ae) A brush or dense tuft of setae.

Scotophase The darkness portion of a 24-h period of light and darkness.

Scurf (-y, -iness) Scales or flakes of dry skin; e.g., dandruff.

Scutellum A small shieldlike sclerite; usually referring to a posteromedian projection of the mesonotum of winged insects.

Scutum A shield or shieldlike sclerite; in insects, usually referring to the major dorsal part of the mesothoracic or metathoracic notum; in hard ticks (family Ixodidae) and chiggers (e.g., family Trombiculidae), represented by a dorsal sclerotized plate of the idiosoma.

Scybalum (-a) In a medical context, a hardened mass of feces; in arthropods, fecal pellets, or scybala; e.g., feces produced by scabies mites as they burrow in skin.

Sebaceous gland Oil-secreting glands of the skin, usually opening into hair follicles; produce fatty secretions called sebum.

Seborrhea A disease of the sebaceous glands involving qualitative changes and excessive discharge of sebum, resulting in oily skin and dermal scales or crusts.

Secondary host A host of lesser importance than the primary host for a pathogen or parasite.

Secondary infection Infection by a second pathogen after another pathogen is already present.

Secondary vector An arthropod that transmits a pathogen or parasite from one vertebrate host to another but which cannot sustain the organism in a natural cycle without transmission by a primary vector.

Self-limiting disease A disease that tends to run its course little influenced by treatment or with no treatment at all.

Semiaquatic Living in or near water or wet substrates but not completely aquatic.

Seminal bursa An internal pouch in some female insects (e.g., in some mosquitoes), that receives the sperm during copulation.

Seminal vesicle In male insects, a pouchlike or tubular enlargement of the vas deferens in which seminal fluid and spermatozoa are stored.

Sensilium A highly sensory patch of integument situated on abdominal tergite 9 or 10 of adult fleas; pygidium of older works.

Sensillum (-a) A simple sensory organ or receptor visible externally on the body or appendages of arthropods; highly variable in both structure and types of stimuli perceived.

Sensillum auriformium A sensory organ of arthropods believed to function in proprioception, e.g., on scutum of ticks; *pl.* sensilla auriformia.

Sensillum coeloconicum A sensory organ of arthropods characterized externally by a peglike or conical projection of the cuticle recessed in a pit; functions in olfaction and thermoregulation; *pl.* sensilla coeloconica.

Sensitization To render sensitive; to induce acquired sensitivity; immunization, especially with reference to antigens not associated with infection; the induction of acquired sensitivity or allergy.

Sensu lato (s.l.) In the broad sense, from Latin; in taxonomy, referring to a group of broadly related taxa, e.g., *Borrelia burgdorferi* s.l.; *cf* sensu stricto.

Sensu stricto (s.s.) In the narrow sense, from Latin; in taxonomy, referring to a distinct taxon, e.g., *Borrelia burgdorferi* s.s.; *cf* sensu lato.

Sentinel (Sentinel flock, herd; e.g., chickens, horses); animals (rarely humans) used to screen for vectors or vector-borne diseases; e.g., flocks of sentinel chickens from which weekly or monthly blood samples are screened for antibodies to zoonotic mosquito-borne arboviruses.

Sepsis Presence of pus-forming and other pathogenic organisms, or their toxins, in the blood or tissues.

Septicemia (-ic) A systemic infection with a pathogen or its toxin present in the blood; blood poisoning.

Sequela(-ae) A condition resulting from and following a disease, injury, procedure, or treatment.

Seroconversion Production of antibodies in blood serum in response to an antigen, resulting from infection or immunization.

Serogroup A group of serotypes with one or more antigens in common.

Serological survey (serosurvey) Sampling a population to determine the presence, incidence, prevalence, or distribution of a disease based on serological testing of blood or other body tissues.

Serology (-ic, -ical) The scientific study of the properties and reactions of serum, particularly blood serum.

Seropositive Exhibiting a significant level of antibody or other immunologic marker in serum denoting exposure to a given infectious agent, i.e., having seroconverted.

Seroprevalence The number of cases or percentage of a population exhibiting the presence of an antibody or other element in serum against a specific pathogen or parasite.

Serosanguineous Consisting or having the nature of blood and serous fluid.

Serosurvey *See* serological survey.

Serotonin A neurotransmitter in mammals that acts as a vasoconstrictor, stimulates smooth muscle, and regulates certain cyclic processes; involved in neural mechanisms affecting pain perception, mood, and sleep-wake cycles.

Serotype A group of closely related microorganisms characterized by a shared set of specific antigens determined by serologic testing.

Serpentine Snakelike.

Serrate An edge notched like teeth of a saw; sawlike and used to cut or lacerate.

Serum (-a, -ous) A watery fluid from animal tissues, usually used to refer to blood serum, a clear amber fluid resulting when whole blood coagulates or is otherwise separated into its solid and liquid components; contains proteins and antibodies.

Sessile Attached by the base without a stalk or other projecting support; also, firmly attached and not free-moving.

Seta (-ae) A slender, hairlike cuticular projection of an epidermal cell of the arthropod integument.

Sex pheromone A chemical compound produced by an organism used to attract a member of the opposite sex of the same species.

Sexual dimorphism Differences in size, morphology, behavior, or other characters that distinguish males from females of a given species.

Sheep strike Cutaneous myiasis of sheep, typically involving blow flies (family Calliphoridae); *see* fly strike.

Shigellosis An acute infection of the intestinal tract caused by bacteria of the genus *Shigella*, characterized by diarrhea, abdominal pain, fever, and dehydration.

Shock A potentially fatal physiological condition caused by inadequate oxygen reaching tissues and cells, characterized by pallor, weak pulse, and marked drop in blood pressure and blood flow; collapse of circulatory function caused by blood loss due to injury, hemorrhaging, disease, allergic reactions, and other conditions.

Sibling species Very closely related species that are morphologically very similar or indistinguishable from one another and reproductively incompatible; believed to represent recent speciation.

Sigmodontine A member of the mammalian subfamily Sigmodontinae that includes cotton rats (*Sigmodon*), deer mice (*Peromyscus*), wood rats (*Neotoma*), and related New World species.

Sign − medical interpretation Objective evidence or manifestation of a disease, body dysfunction, or other disorder, excluding impressions or subjective assessments; *cf.* symptom.

Silage Fodder harvested while green and preserved by storing and fermenting in a silo.

Silica gel A gelatinous form of colloidal silica (silicon dioxide) used as a dehumidifying and dehydrating agent.

Silurian Pertaining to the Paleozoic Era of geologic time 425−400 million years ago, characterized by the emergence of air-breathing land invertebrates and terrestrial plants; Silurian Period.

Simuliid A member of the dipterous family Simuliidae, black flies.

Sinuous Characterized by curves or turns; winding or linearly wavy.

Sinusitis Inflammation of a sinus, especially the paranasal sinus.

Siphon A respiratory structure formed by a projection of the body wall of aquatic invertebrates bearing a spiracle, or pair of spiracles, at the tip; e.g., culicid and psychodid larvae.

Site specificity Pertaining especially to ectoparasites and other blood-feeding arthropods whereby they preferentially or exclusively live and/or feed on a particular body region of the host.

Skin biopsy Removal of skin tissue, usually a lesion, for microscopic examination by a pathologist or for diagnostic testing to determine the cause and nature of a lesion or other medical condition.

Skin snip A small sample of skin tissue removed for biopsy.

Sodium phenobarbital A short-acting barbiturate in the form of a sodium salt, used medically to treat seizures and as a preoperative sedative or anesthetic.

Solenophage (-y) An insect or other arthropod with mouthparts modified to pierce the skin and feed on blood directly from a blood vessel.

Solitary Living or existing alone, rather than in a group or colony; e.g., solitary wasps and bees.

Souma A Sudanese name for nagana, a disease of domestic animals in tropical Africa caused by trypanosomes transmitted by tsetse flies.

Source reduction A general term for reducing or eliminating the source of a problem, such as standing water where mosquitoes breed; involves removal or modification of the environment to make the site unsuitable for producing or harboring pest species.

Sowbug A member of the Crustacean class Isopoda in the terrestrial family Oniscidae that feed on decaying vegetable matter; distinguished from the related pillbugs by a pair of tail-like appendages and the inability to roll into a tight ball.

Spermalege A structure of female bed bugs and other cimicids, separate from the normal reproductive organs, that receives sperm during traumatic insemination; sperm passes from there to the oviduct via the hemacoel; also called organ of Berlese and organ of Ribaga.

Spermatheca (-ae) A structure of the female reproductive system of arthropods for receiving and storing spermatozoa from the male.

Spermatogenesis The process of formation and development of spermatozoa.

Spermatogenic arrest The suspension or termination of spermatogenesis as a result of disease, physiological dysfunction, radiation, environmental factors, or other causes.

Spermatophore A capsule or envelope containing spermatozoa and seminal fluid produced by male arthropods and transferred to the female during copulation.

Sphaeromastigote A developmental stage of trypanosome protozoans having a rounded body and an anterior flagellum.

Spine A multicellular, nonarticulated outgrowth of the arthropod integument typically in the shape of a thorn or stiff tapered process with a pointed tip.

Spinneret An external organ via which fluid silk is extruded from glands to produce threads used by insects, spiders, and mites to form webs, cocoons, and other silk structures.

Spiracle A pore or other opening in the arthropod integument connected with a tracheal tube allowing for exchange of gases between the body and surrounding air.

Spiracular plate A sclerotized area of the arthropod integument surrounding a spiracular opening**(s)**.

Spirochete Any of various pathogenic and nonpathogenic spiral-shaped, motile bacteria of the order Spirochaetales; includes *Borrelia* and *Treponema* as pathogens of humans and other animals.

Spirochetosis (-es) An infection caused by spirochetes.

Spirurid A member of the nematode family Spiruridae in which the larva parasitizes insects and the adult parasitizes vertebrates.

Splenomegaly Enlargement of the spleen.

Sporocyst A walled structure within which sporozoites are formed and develop.

Sporogony Asexual reproduction by multiple fission of a spore or zygote to produce sporozoites, characteristic of sporozoans.

Sporozoan A parasitic spore-forming protozoan of the class Sporozoa; e.g., *Babesia*, *Plasmodium.*

Sporozoite A developmental stage of sporozoans usually produced in the arthropod host and that serves as the infective stage for a vertebrate host.

Sporulation Asexual production and release of spores.

Spot treat Application of a pesticide to specific, restricted locations where pest species are most likely to encounter it.

Spot-on A chemical formulation topically applied to the skin of an animal at a localized site where it is absorbed; usually an insecticide or parasiticide.

Spur A spinelike cuticular structure of the arthropod integument that articulates or attaches at its base with the body wall (or with a leg coxa, trochanter, or tibia in hard ticks).

Spurious (Vein) resembling something it is not; not genuine or true; e.g., spurious wing vein.

Sputum Saliva mixed with mucus, phlegm, cellular debris, microorganisms and other substances from the respiratory tract expelled by coughing or clearing the throat.

Squab A young domestic pigeon, typically less than 4 weeks old.

Squama (-ae) A scale or thin platelike structure; e.g., in insects, a scalelike sclerite at the base of the wing or haltere in certain dipterous adults.

Stadium (-ia) The interval of time between two successive molts in the larval or nymphal development of insects and some arachnids.

Stage A specific period of time or step in the development of an organism; in insects, the time between two molts, e.g., third-stage larva, or third instar; also used to refer to a major developmental division in a life cycle, e.g., larval stage.

Stanchion (-ed, ing) A vertical rod or post used as support; a framework of vertical bars for confining cattle at a feed trough or in a stall.

Steppe A vast, semi-arid plain of grassland, especially in parts of Europe and Asia.

Stercorarian Referring to excrement or dung; in vector biology, specifically referring to transmission of a pathogen or parasite via the feces of the vector; posterior-station transmission.

Sternite A sudividion of the sternum or discrete sclerotized plate in the sternal area.

Sternum The ventral sclerotized part of a body segment.

Steroid A class of organic compounds with 17 carbon atoms forming four rings; includes sterols, D vitamins, the sex hormones testosterone and estrogen, adrenal hormones, and plant alkaloids.

Sticky trap A device with adhesive compounds applied to the surface to capture crawling or flying insects (or rodents, etc.).

Stigma (-ata) A spiracle or respiratory opening, usually used when referring to mites; also, a distinctive sclerotized or pigmented spot on the insect wing.

Stillborn Dead at birth.

Sting autotomy Self-amputation of the sting apparatus in certain hymenopterous insects in which barbed lancets anchor the sting in skin, tearing the sting and associated venom gland from the abdomen as the insect pulls away; e.g., honey bees.

Sting A sclerotized apparatus with associated muscles derived from the female reproductive system of some wasps, ants, and bees for delivering venom; any such apparatus associated with venom glands in other insects and arachnids; e.g., sting of scorpions; *syn.* stinger.

Stomatitis Inflammation of the mouth, especially the mucous membranes.

Stool Fecal matter from a single bowel movement.

Stratum corneum The outermost horny layer of the epidermis of vertebrates, consisting chiefly of keratinized dead cells that slough off.

Stratum germinativum The innermost layer of epidermis consisting of a single row of cells that divide to replace other cells of the epidermis as they die and are worn away.

Stratum granulosum A layer of the epidermis between the stratum germinativum and the stratum corneum or stratum lucidum, characterized by deeply staining basophilic granules; functions in strengthening and waterproofing the skin.

Stratum lucidum A translucent layer of epidermal cells underlying the stratum corneum, especially in the thickened skin of the palms and soles; absent in many other epidermal tissues.

Striate (-tion) Marked by long, fine parallel lines, or striae, grooves, or ridges.

Stridulate (-atory) To produce sound by rubbing two body parts together as a means of acoustical communication.

Strike See *Fly strike* and *Sheep strike.*

Stylet A small, rigid, needle-like appendage or other external process in arthropods.

Styletiform Resembling a stylet.

Stylostome A feeding tube produced by chiggers and some other parasitic mites while attached to a host, formed by salivary secretions and through which lysed host tissue is drawn to the mouth.

Stylus (-i) A tapered, nonarticulated process; also, the annulated terminal part of the third antennal segment of some adult flies, e.g., tabanids.

Subacute In relation to disease, between acute and chronic but with some acute features.

Subalar Below the wing.

Subclinical Relating to an early stage of a disease before clinical symptoms are apparent.

Subcosta The longitudinal vein near the anterior margin of the insect wing immediately behind and parallel to the costa.

Subcutaneous Beneath the skin.

Subcylindrical More or less cylindrical in shape.

Subfamily A taxonomic subdivision within a family containing one or more closely related genera, named after the type genus and typically ending in -inae.

Subgenital plate A sternal sclerite or process of the eighth abdominal segment in insects covering or adjacent to the gonopore.

Subgenus A taxonomic subdivision within a genus, the name of which is capitalized, italicized and placed in parentheses following the genus name.

Submucosa A layer of loose connective tissue below a mucous membrane.

Suborder A major taxonomic subdivision within an order, containing a group of related superfamilies and families.

Subperiodic (-ity) Occurring at something less than regular intervals of time.

Subscutellum The ventral surface of a transversely infolded postscutellum of adult insects.

Succession Gradual colonization of a newly formed habitat, usually by a series of species assemblages, as in arthropod succession of cadavers or carrion.

Suid A member of the family Suidae, including domestic and wild pigs, hogs, warthog, and babirusa; swine.

Sulfluramid A fluorinated sulfonamide with insecticidal properties, often used in bait traps to control cockroahes, ants, and other insects.

Supportive therapy Treatment based on clinical signs and symptoms, without necessarily knowing or directly addressing the underlying cause of a medical problem.

Suppurative Producing or discharging pus.

Suprageneric In taxonomic classification, anything above the genus level.

Suprapylarian Growth or development of a parasite or pathogen in the midgut of a vector as in members of the protozoan subgenus *Leishmania* of the genus *Leishmania*; as opposed to peripylarian, which implies growth or development in the hindgut.

Supraspecific In taxonomic classification, anything above the species level.

Surra An infectious disease of domestic animals, especially horses, caused by *Trypanosoma evansi* and transmitted by the bite of tabanid flies; characterized by anemia, fever, and emaciation.

Susceptibility Lack of ability to resist an extraneous agent such as a pathogen, parasite, or drug.

Suslik Any of several Eurasian ground squirrels (Sciuridae), especially the European species *Citellus citellus.*

Suspect(ed) case A medical diagnosis based on reasonable but nonconclusive evidence or laboratory testing.

Suture (-al, -ed) In arthropods, a seam, seamlike line, or boundary between two sclerotized areas of the arthropod integument; also, surgically sewn wounds or incisions.

Sweat flies Nnonbiting muscid flies that are attracted to, and persistently feed on, animal perspiration and other body secretions; used most commonly with reference to members of the genus *Hydrotaea.*

Sylvatic Occurring in, affecting, or transmitted by wild animals.

Symbiont The smaller of two organisms in a symbiotic relationship between different species, which always benefits from the association.

Symbiosis (-tic) Any close and prolonged physical relationship between individuals of two different species; may be mutualistic, parasitic, or commensal.

Sympatry (-ic) Two or more species with the same or overlapping geographical distributions.

Symptom (-atic) Any perceptible change in the body or its functions indicative of a disease or other disorder; in a more restricted sense, referring to a subjective change, as opposed to an objective change, or sign (see *sign*).

Synanthropic Ecologically associated with humans.

Syndrome A group of signs and symptoms that collectively characterize or indicate a particular disease or abnormal condition.

Synergist (-istic) A compound or other agent which, when combined with another substance, interacts to increase the effectiveness of the latter; a nontoxic substance in a pesticide that increases the potency of the active ingredient.

Synonym One of two or more scientific names for the same taxon; generally applied to a previously used name that has been superseded by the currently accepted name; the relegated (nonvalid) name(s) is (are) called the junior synonym**(s)**.

Systematics The study and classification of organisms and their relationships.

Systemic Pertaining to, or affecting, the entire body or a particular body system, e.g., nervous system; translocated throughout the body, typically via the circulatory system.

T cell Any of several lymphocytes that develop in the thymus and play an important role in the body's immune system by recognizing and destroying infected or malignant cells; *syn.* T lymphocyte.

Tabanid A member of the dipterous family Tabanidae, horse flies and deer flies.

Tachinid A member of the dipterous family Tachinidae, in which the larva is parasitic on other insects.

Tachycardia Excessively rapid heartbeat, over 100 beats per minute in human adult.

Tachypnea Abnormally rapid breathing or respiration.

Tagma (-ata) A group of successive segments forming a distinct section of an arthropod (especially insect) trunk, such as along the abdomen.

Tail head In ungulates such as cattle and horses, the base of the tail where it joins the rump.

Tapeworm A member of the class Cestoda, flat ribbon-like or tapelike parasites found as adults in the alimentary tract of vertebrate animals; typically develop as immature stages in other vertebrate or arthropod hosts.

Tarsomere A subsegment of the arthropod tarsus.

Tarsus The distal-most apparent segment of the arthropod limb; actually the penultimate segment, usually bearing the claws and associated structures, or pretarsus.

Taxon (pl., taxa) A unit of classification for animals, plants or microorganisms; e.g., species, genus, family, order, phylum, kingdom, etc.

Taxonomy The practice of describing, naming, identifying, and classifying organisms according to a system based on the rules of zoological nomenclature.

Tegmen (-ina) The sclerotized, leathery forewing of certain orders of insects, including cockroaches and grasshoppers; protects the more delicate hindwing when folded beneath.

Telmophage (-gy) *See* pool feeder.

Telson The terminal-most part of the insect abdomen, or opisthosoma of certain arachnids, bearing the anus; not a true segment; term applied to the terminal segment of the scorpion tail.

Temephos A nonsystemic organophosphorus insecticide; often applied to lakes, ponds, rivers or wetlands to control larvae of aquatic Diptera such as those of mosquitoes and black flies; also used to control fleas and lice on animals.

Temperate region An area having a moderate climate (i.e., not tropical or subtropical); an area that experiences cooler winter months compared to summer months.

Teneral Pertaining to a recently molted immature or adult arthropod in which the integument has not yet hardened and is still pale compared to the final coloration.

Tentorium The internal skeleton of the insect head.

Tergal gland A gland associated with the tergum of the male cockroach that produces pheromones and other secretions upon which the female feeds during courtship and mating.

Tergite A sclerotized subdivision of the tergum of arthopods.

Tergo-trochanteral Referring to the origin and insertion of insect muscle between a tergum and trochanter.

Tergum (-a) The dorsal surface of an arthropod body segment.

Terminalia The posterior-most segments and associated structures of the insect abdomen, including modifications to form the external genitalia.

Terrapin Any of various web-footed turtles of the family Emydidae inhabiting fresh or brackish waters of North America, including Mexico; some turtles from other parts of the world are also sometimes called terrapins.

Territoriality The behavior of an animal in establishing and defending a defined geographic area.

Tertian Recurring at approximately 48-h intervals, or every other day, i.e., every third day; e.g., tertian fever.

Tessellate (-ed) A mosaic composed of little squares, like a checkerboard; e.g., color pattern on dorsum of abdomen of adult sarcophagid flies.

Testosterone The male sex hormone of vertebrates secreted primarily by the testes, which stimulates development of male sex organs, secondary sexual traits, and sperm.

Tetracycline A broad-spectrum antibiotic synthesized or derived from soil microorganisms of the genus *Streptomyces.*

Thallus (-i) A vegetative body not differentiated into true roots, stems, or leaves; characteristic of fungi and lichens.

Theileriosis An infection by a protozoan of the genus *Theileria*; e.g., East Coast fever of cattle; theileriasis.

Theraphosid A member of the spider family Theraphosidae, or tarantulas.

Thermal fog An insecticidal fog produced by heating the product without degrading the active ingredient.

Thermoregulation The control, adjustment, or maintenance of body temperature within defined limits by physiological or behavioral means.

Thigmotaxis (-actic) Movement of an organism toward or away from an object in response to touch or a mechanical stimulus.

Thiocyanate A salt or ester of thiocyanic acid, formed when alkaline cyanides combine with sulfur.

Thorny-headed worm A member of the phylum Acanthocephala, parasitic worms with an eversible proboscis armed with spines used to attach to the gut wall of invertebrate and vertebrate hosts; *syn.* thorn-headed worms.

Thrombocytopenia An abnormal decrease in the number of platelets in circulating blood.

Thrombosis (-es) The formation of a blood clot, or thrombus, in any part of the circulatory system.

Tibia (ae) The fourth segment of the insect leg, between the femur and tarsus.

Tibial spur A spur usually located near the distal end of the insect or hard-tick tibia.

Tibio-tarsal claw The terminal grasping structure of a sucking louse leg formed by the curved tarsal element opposing the tibial spur.

Togavirus A genus of arboviruses belonging to the family Togaviridae.

Tormogen The epidermal cell that forms a cuticular depression at the base of a seta; *syn.* tormogen cell.

Torticollis Spasmodic contractions of the neck muscles twisting the head to one side with the chin pointing to the other.

Toxemia The presence of a toxin in the blood disseminated from a site of bacterial infection or metabolic toxins resulting from disease or organ failure; *syn.* blood poisoning.

Toxicant A general term for any poison or poisonous agent.

Toxicosis (-es) A pathological condition resulting from a poison or toxin; systemic poisoning.

Toxin A poisonous substance produced by plants or animals.

Toxoplasmosis A disease caused by the protozoan *Toxoplasma gondii*; commonly transmitted to humans from infected cats or cat feces.

Trachea (-eae) (1) in insects and some other arthropds, one of a series of large respiratory tubes that connect externally to the spiracles and internally to the tracheoles; (2) in vertebrates, the windpipe or airway extending from the larynx into the thorax.

Tracheitis Inflammation of the trachea (in vertebrates).

Tracheobronchial Pertaining to both the trachea and bronchi or bronchioles.

Tracheole The smaller, finer tubules of the arthropod tracheal system.

Trachoma A contagious eye disease caused by infection of the conjunctiva and cornea by the bacterium *Chlamydia trachomatis*; characterized by inflammation, granulations, and scarring; a major cause of blindness in Africa and Asia.

Transmissible (-ility) Capable of being carried from one individual to another, as an infectious agent.

Transovarial transmission The passage of a pathogen from a female organism to its offspring by infection of eggs while developing or retained in the ovary.

Transovarial Relating to the passage of any agent or substance from a female organism to its offspring via the ovary and egg.

Transplacental infection The passage of a pathogen from mother to fetus via the placenta.

Transstadial transmission The passage of a pathogen in arthropods from one instar or developmental stage to the next.

Traumatic insemination An unusual form of copulation in bed bugs and other cimicids in which the male aedeagus penetrates the female body wall to deposit sperm directly into a specialized structure (spermalege) of the female reproductive system.

Tree hole Any rotted cavity in a tree; often collects rainwater and serves as a temporary breeding site for mosquitoes, biting midges, and other aquatic insects.

Triatomine Referring to members of the subfamily Triatominae, or kissing bugs (family Reduviidae).

Trichinosis A human disease caused by ingestion of larvae of the parasitic nematode *Trichinella spiralis* by eating raw or insufficiently cooked pork; the larvae develop to adults in the intestinal tract.

Trichobothrium (-ia) A specialized sensory seta of arthropods characterized by a cuticular depression or socket at the base; mechanoreceptors for tactile stimuli.

Trichogen An epidermal cell that forms a seta; *syn.* trichogen cell.

Tritonymph The third nymphal stage in the basic life cycle of some mites.

Tritosternum A medioventral, forked structure on the sternite between the first pair of legs of gamasid mites, believed to function in guiding fluids while the mite is feeding.

Trochanter The second, and typically smallest, segment of the insect and arachnid leg.

Troglobite (-tic) A cave-dwelling animal that has become specifically adapted to living in total darkness.

Trombiculid A member of the mite family Trombiculidae, or chiggers.

Trombiculosis An infestation with trombiculid mites, i.e., chiggers.

Trombidiosis A synonym in the older literature for trombiculosis; no longer used.

Trophallaxis The direct transfer of food or other fluids between members of a colony of social insects such as ants and bees; may be mouth-to-mouth or anus-to-mouth.

Trophic Relating to nutrition.

Trophozoite A protozoan in the active feeding stage of its life cycle.

Tropicopolitan A biogeographical term implying distribution of an organism throughout all of the tropical regions of the world; e.g., Tropicopolitan distribution.

Trumpet *See* respiratory horn.

Trypanosome A flagellate protozoan of the genus *Trypanosoma*, family Trypanosomatidae; parasites in blood and tissues of humans and other animals, usually transmitted by insects

Trypanosomiasis An infection or disease caused by a trypanosome

Trypanotolerant Able to endure or resist the adverse effects of infection with trypanosomes

Trypomastigote Any protozoan of the family Trypanosomatidae that has the typical form of a mature trypanosome, i.e., with flagellum and undulating membrane

Tsutsugamushi A disease of humans caused by the rickettsial organism *Orientia tsutusgamushi* transmitted by chiggers; *syn.* tsutsugamushi fever, tsutsugamushi disease, scrub typhus, chigger-borne rickettsiosis.

Tuberculate (-ed) Covered with small rounded projections, or tubercles.

Tungiasis An infestation of the skin by the flea *Tunga penetrans.*

Turbinate A scroll-like spongy bone of the nasal passages of humans and other vertebrates.

Tylenchid A member of the nematode family Tylenchidae.

Typhoid An infectious enteric disease of humans caused by the bacterium *Salmonella typhi*.

Typhus Any of several forms of infectious disease caused by rickettsial organisms of the genus *Rickettsia* transmitted by lice, fleas, ticks, and chiggers; characterized by high fever and eruption of reddish spots on the skin; sometimes, incorrectly used to imply only louse-borne (epidemic) typhus.

Ulcer (-ate, -ed, -ous, -ation) An open sore or lesion of the skin or mucous membranes with necrosis and formation of pus.

Ultra-low volume Referring to highly concentrated formulations of pesticides that are applied in very small quantities (0.6−4.7 L/ha) as microscopic droplets within the size range of 0.1−50 μm in diameter (80% of the droplets must be within the range, 0.1−30 μm).

ULV See *ultra-low volume.*

Umbilicus (-cal) Depressed scar in middle of abdomen denoting attachment of umbilical cord during fetal development; navel.

Ungulate A hoofed mammal; includes members of the two orders Perissodactyla and Artiodactyla.

Univoltine Having one generation per year.

Uric acid A white nitrogenous waste product excreted by insects, most reptiles, and birds.

Urogenital Pertaining to both the urinary and reproductive systems of vertebrates.

Urticaria (-al) A vascular reaction of the skin characterized by eruption of pale wheals and intense itching; caused by, among other things, allergic reaction to insect bites and stinging hairs of caterpillars; *syn.* hives, nettling rash.

Vaccination Inoculation with a vaccine.

Vaccine (-ation) A suspension of a weakened (attenuated) or killed pathogen, or part of a pathogen, used to inoculate an individual to stimulate the production of antibodies as a means of immunological protection against future infection.

Vasoconstriction Narrowing of the lumen of a blood vessel.

Vasodilatation An enlargement or stretching of a blood vessel, especially small arteries and arterioles; *syn.* vasodilation.

Vasodilator A nerve, drug, or other agent that dilates a blood vessel.

Vector An organism, usually an arthropod, which transmits a disease agent from an infected to a noninfected animal or plant.

Vector competence The susceptibility of an arthropod species to infection with a pathogen or other parasite and its ability to transmit the acquired organism.

Vector-borne disease A disease involving a vector, usually an arthropod, as the means by which the causative agent is transmitted from one individual to another.

Vectorial capacity A quantitative summary of the basic ecological attributes of a vector relating to its ability to transmit a pathogen or parasite.

Venation See *wing venation.*

Venereal Pertaining to sexual intercourse or the genitals; e.g., venereal disease, venereal transmission.

Venom (-ous) A poison secreted by an animal and usually transmitted by a bite or sting.

Ventriculus The digestive part of the alimentary tract of insects; *syn.* midgut, stomach.

Venule A small vein; in insects, a branch of a vein in the wing.

Vermiform Resembling a worm; elongate, cylindrical, and tapered or rounded at both ends; e.g., vermiform larva.

Vernal Relating to or occurring in the spring.

Vertex The top of the head of an insect between the eyes.

Vertical transmission The passage of a pathogen from parent to offspring.

Vertigo A sensation of moving around in space, objects whirling around the individual, or falling as a result of disturbance to one's equilibrium.

Vesicant A chemical agent that causes blistering.

Vesicle A small sac or bladder-like elevation of the skin containing serous fluid; blister.

Vesicular dermatitis Inflammation of the skin marked by vesicles or small blisters.

Vesicular stomatitis A viral disease of horses, cows, and swine, characterized by erosive blisters in and around the mouth caused by rhabdoviruses of the genus *Vesiculovirus*; can be transmitted by insects during epizootics.

Wallaby Any of various marsupials related to, but generally smaller than, kangaroos and common to Australia, New Guinea, and adjacent islands; includes the genera *Macropus*, *Lagostrophus*, *Lagorchestes*, *Wallabia*, and *Petrogale.*

Wallow A pool of water or mud where animals slowly and clumsily roll about.

Wapiti A large North American deer, *Cervus canadensis*, with large branched antlers in the male, commonly called American elk.

Warren An area where rabbits live in burrows; a colony of rabbits.

Waste lagoon A natural or artificial shallow body of water in which animal wastes are collected and undergo biodegradation.

Wasting condition A general deterioration of an animal's health characterized by loss of weight and strength, and emaciation; wasting disease.

Wasting disease *See* wasting condition.

Wattle A fleshy, often wrinkled and brightly colored fold of skin hanging from the throat or neck of certain birds such as chickens and turkeys.

Welt An unbroken elevation of the skin caused by an allergic reaction or by a lash or blow, as from as stick or whip.

Wettable powder A dry formulation of a pesticide that is mixed with water to form a suspension prior to making an application.

Wheal A small raised mark or swelling of the skin of short duration, as from an arthropod bite or allergic reaction; usually itches or burns.

Whipworm A slender, whip-shaped nematode of the family Trichuridae, especially *Trichuris trichiura*, which parasitizes the human intestine.

WHO World Health Organization, an agency of the United Nations established in 1948 with headquarters in Geneva, Switzerland.

Wing cell See *cell.*

Wing venation The complete system and pattern of veins of an insect wing.

Wipe-on A chemical formulation that is topically applied to an animal with a cloth or other fabric, usually as a toxicant or repellent to protect the animal from attack by biting insects.

Withers The highest part of the back of a horse, cow, sheep, and other livestock located at the base of the neck between the shoulder blades.

Wool slippage Damage to the fleece of sheep caused by a heavy infestation of biting (chewing) lice resulting in patches of lost hair.

Xanthine A yellow-white, nitrogenous, crystalline compound produced in the breakdown of purines to uric acid.

Xenodiagnosis A method of diagnosing an arthropod-transmitted disease by allowing an uninfected vector species to feed on the patient, then examining the arthropod for the presence of the infective organism following a suitable incubation period.

Yak An ox, *Bos grunniens*, of the Tibetan highlands, with a stout body, short legs, a thick shaggy coat of hair hanging to the ankles, and large upward-curved horns; both wild and domesticated.

Zoogeographic region A large-scale biogeographic division of the earth's surface defined by zoologists on the basis of characteristic fauna, representing long periods of isolation and evolutionary distribution patterns.

Zoonosis (-es, -tic) A disease of wild or domestic animals that can be transmitted to humans.

Zoophagy (-ic) Feeding on live animals.

Zoophily (-ic) Attraction to or preference for feeding on animals.

Zooprophylaxis A precaution or preventive measure taken to protect animals from contracting or transmitting a disease agent; e.g., immunization, quarantine, screened enclosures; also, the diversion of feeding by vectors onto hosts that are not reservoirs for certain vector-borne pathogens or parasites.

Zygote The cell formed by the union of two gametes, typically an ovum and sperm, prior to undergoing cleavage; fertilized egg.

This page intentionally left blank

Taxonomic Index

'*Note*: Page numbers followed by "f" indicate figures and "t" indicate tables.'

A

Abbreviata caucasica, 73t
Acanthocheilonema, 223
Acanthocheilonema dracunculoides, 431t–432t, 432
Acanthocheilonema reconditum, 101, 155t, 165–166
Acanthoscurria, 508
Acarus, 556
Acarus farris, 545, 546f
Acarus siro, 545, 545f, 547, 553, 555–556
Achipteria, 559, 594t–595t
Acinetobacter, 71t–72t
Acinetobacter baumannii, 88, 94–95, 98
Acomatacarus, 559
Acomys, 539
Actinomyces, 364
Actinoptera, 396
Actinopus, 507–508
Actornithophilus, 85
Adoristes, 595t
Aedes, 19f, 36, 262, 264, 272–273, 279–280, 283t, 286t, 298t, 308, 696t–700t
Aedes abserratus, 298t
Aedes aegypti, 3, 37, 228, 263, 264f, 271, 275–276, 278–280, 283t, 287–292, 288t, 289f, 291f, 297, 681t–682t, 688, 696t–700t
Aedes africanus, 263, 283t, 286, 288t, 289, 292, 296
Aedes albifasciatus, 283t, 284–285
Aedes albopictus, 48–49, 263, 275, 278, 287, 288t, 292, 292f, 297, 681t–682t, 696t–700t
Aedes atlanticus, 298t, 299
Aedes bromeliae, 263, 288t, 289
Aedes canadensis, 298t
Aedes cinereus, 265f
Aedes communis, 298t
Aedes cordellieri, 286
Aedes dorsalis, 278–279, 283t, 284–285, 298t, 696t–700t
Aedes fitchii, 266f
Aedes formosus, 263
Aedes fulvus, 298t
Aedes fulvus pallens, 265f
Aedes furcifer, 286, 288t, 292
Aedes infirmatus, 299
Aedes intrudens, 298t
Aedes luteocephalus, 286, 288t, 292
Aedes macintoshi, 696t–700t
Aedes melanimon, 41, 283t, 284–285, 297, 298t, 696t–700t
Aedes metallicus, 288t
Aedes niveus, 288t
Aedes opok, 286, 288t, 292
Aedes pembaensis, 298t
Aedes polynesiensis, 263, 283t, 288t, 292
Aedes provocans, 298t
Aedes pseudoscutellaris, 263, 288t, 292
Aedes rotumae, 288t
Aedes scapularis, 298t
Aedes scutellaris, 288t, 292
Aedes serratus, 221
Aedes sollicitans, 283t, 284
Aedes stimulans, 298t
Aedes taeniorhynchus, 274
Aedes taylori, 286, 288t
Aedes tormentor, 298t, 299
Aedes triseriatus, 38, 298–299, 298t, 696t–700t
Aedes trivittatus, 284–285, 298t, 299
Aedes vexans, 274, 278–279, 294, 298t
Aedes vigilax, 283t, 696t–700t
Aedes vittatus, 288t
Aegyptianella pullorum, 625–626
Aeromonas, 71t–72t
Africacarus, 595t
Agelena labyrinthica, 508
Aglossa, 56t
Alacran tartarus, 491
Alcaligees faecalis, 71t–72t
Alcelaphus buselaphus, 377
Alces alces, 345
Aleochara, 141
Aleuroglyphus ovatus, 552, 555–556
Allodermanyssus sanguineus, 539
Allogalumna, 595t
Allolobophora, 354
Alloxacis, 134
Alouatta, 290, 405–406
Alphitobius diaperinus, 138–141, 139f
Amara, 139
Amblyomma, 20f–21f, 603t, 616, 654
Amblyomma americanum, 611f, 619t–620t, 624f, 632, 648
Amblyomma hebraeum, 624–625, 625f, 654
Amblyomma maculatum, 624f, 636
Amblyomma nuttalli, 611
Amblyomma rotundatum, 609f
Amblyomma variegatum, 624–625, 663
Amblyopinus, 140
Analges chelopus, 569f
Ananteris coineaui, 496–497
Anaphe, 443
Anaplasma, 652
Anaplasma marginale, 337t, 339, 655
Anaplasma phagocytophilum, 637
Anaticola anseris, 81t
Anaticola crassicornis, 81t
Anaxipha gracilis, 209–210
Ancyclostoma duodenale, 72–73
Androctonus, 490, 499–501
Androctonus amoreuxi, 490t
Androctonus australis, 25t–26t, 490t, 496f, 500, 500f
Androctonus crassicauda, 490t
Androctonus mauretanicus, 490t, 500
Androlaelaps, 562
Androlaelaps fahrenholzi, 535f
Anisomorpha buprestoides, 9, 9f
Anisomorpha ferruginea, 9
Anocentor, 605
Anocentor nitens, 604
Anolisimyia, 386t–389t, 402
Anomalohimalaya, 603t, 605
Anopheles, 37, 42, 262, 264, 264f, 267f, 272–273, 272f–273f, 277–280, 283t, 286t, 287, 297, 298t, 301–302, 304–305, 308, 678
Anopheles aconitus, 304t
Anopheles albimanus, 303
Anopheles annularis, 304t
Anopheles annulipes, 304t
Anopheles anthropophagus, 304t
Anopheles arabiensis, 262, 304t
Anopheles atroparvus, 304t
Anopheles aztecus, 304t
Anopheles balabacensis, 304t
Anopheles bancrofti, 304t
Anopheles campestris, 304t
Anopheles christyi, 304t
Anopheles claviger, 304t
Anopheles culicifacies, 304t
Anopheles darlingi, 303
Anopheles dirus, 304t
Anopheles donaldi, 304t
Anopheles earlei, 266f
Anopheles farauti, 304t
Anopheles flavirostris, 304t
Anopheles fluviatilis, 304t
Anopheles freeborni, 304t

Anopheles funestus, 262, 287, 304t, 696t–700t
Anopheles gambiae, 262, 266f, 270f, 271, 276, 279–280, 287, 304f, 304t, 681t–682t
Anopheles hispaniola, 304t
Anopheles karwari, 304t
Anopheles koliensis, 304t
Anopheles labranchiae, 304t
Anopheles letifer, 304t
Anopheles leucosphyrus, 304t
Anopheles ludlowae, 304t
Anopheles maculatus, 304t
Anopheles maculipennis, 265f, 304–305
Anopheles melas, 304t
Anopheles merus, 304t
Anopheles messeae, 304t
Anopheles minimus, 304t
Anopheles moucheti, 304t
Anopheles multicolor, 304t
Anopheles nigerrimus, 304t
Anopheles nili, 304t
Anopheles pattoni, 304t
Anopheles pharoensis, 304t
Anopheles philippinensis, 304t
Anopheles pseudopunctipennis, 304t
Anopheles pulcherrimus, 304t
Anopheles punctimacula, 304t
Anopheles punctipennis, 304t
Anopheles punctulatus, 304t
Anopheles quadrimaculatus, 265f, 304t, 696t–700t
Anopheles sacharovi, 304t
Anopheles sergentii, 304t
Anopheles sinensis, 304t
Anopheles smithii, 312
Anopheles stephensi, 228, 304t
Anopheles subpictus, 304t
Anopheles sundaicus, 304t
Anopheles superpictus, 304t
Anopheles tesselatus, 304t
Anopheles whartoni, 304t
Anoplocephala magna, 594t
Anoplocephala perfoliata, 594t
Anthela nicothoe, 443
Anthocoris musculus, 107
Anthrenus, 56t
Anthrenus flavipes, 137f
Anthrenus scrophulariae, 137f
Anthrenus verbasci, 137f
Antricola, 605
Anuroctonus, 491
Aops, 493
Aops oncodactylus, 493
Apatolestes, 328t
Apatolestes actites, 330–331
Aphodius, 141
Aphonopelma, 516
Aphonopelma hentzi, 515–516
Aphonopelma seemani, 528
Aphonopelma surinamensis, 516f
Apis, 463, 478–479
Apis dorsata, 478
Apis florea, 478
Apis mellifera, 24, 24f, 25t–26t, 27f, 478–479, 478f–479f
Apis mellifera scutellata, 478–479
Apodemus, 544, 559
Apodemus sylvaticus, 165
Aponomma, 605, 616
Aposycorax chilensis, 192–193
Aprostocetus hagenowii, 75
Arcyophora, 451
Arenaviridae, 702t
Arenavirus, 702t
Argas, 603t, 605
Argas brumpti, 648
Argas persicus, 625–626
Argas reflexus, 648
Argiope aurantia, 508–509
Argiope lobata, 508–509
Arilus cristatus, 107
Armigeres, 262t, 270
Arsenophonus, 425
Ascaris, 72–73
Ascaris lumbricoides, 72–73
Ascarops strongylina, 140
Ascoschoengastia, 559
Asfarviridae, 696t–700t
Asfivirus, 696t–700t
Aspergillus, 138–139, 545
Aspergillus penicillioides, 554
Aspidoptera falcata, 430–432
Atelerix albiventris, 585
Atelopus, 192–193
Atherix, 182f
Atrax, 507–508, 517
Atrax robustus, 24, 25t–26t, 28f, 508, 517, 517f
Attagenus, 56t
Atylotus, 328t, 337t
Atylotus agrestis, 339
Atylotus thoracicus, 330–331
Auchmeromyia, 386t–389t
Auchmeromyia luteola, 401
Auchmeromyia senegalensis, 386t–389t, 401, 401f
Augochloropsis metallica, 476f
Austenina, 369–370
Austroconops, 213, 216
Austrosimulium australense, 244t
Austrosimulium pestilens, 231, 240, 244t, 253–254
Austrosimulium ungulatum, 244t, 248t–249t
Autographa gamma, 441f
Autoserica castanea, 137
Avipoxvirus, 311
Avitellina bangaonensis, 594t
Avitellina chalmersi, 594t
Avitellina goughi, 594t
Avitellina lahorea, 594t
Avitellina sudanea, 594t
Avitellina tatia, 594t
Avitellina woodlandi, 594t

B

Babesia, 627–628, 649–650
Babesia bigemina, 2–3
Babesia caballi, 620
Babesia canis, 620
Babesia duncani, 627
Babesia microti, 627
Bacillus, 138–139
Bacillus anthracis, 280, 337t, 338
Bacillus cereus, 71t–72t
Bacillus sphaericus, 209–210, 315
Bacillus subtilis, 70–72, 71t–72t
Bacillus thuringiensis, 209–210, 254
Bacillus thuringiensis israelensis, 315
Badumna insignis, 528
Badumna robusta, 528
Balataea, 443
Balumna insignis, 509
Bandicota, 160
Baraimlia fuscipes, 150
Bartonella, 201, 425, 431t–432t, 433
Bartonella bacilliformis, 199t, 201, 201f
Bartonella clarridgeiae, 161–162
Bartonella elizabethae, 161–162
Bartonella henselae, 155t, 161–162
Bartonella melophagi, 424, 433
Bartonella quintana, 88, 97, 99t
Bartonella schoenbuchensis, 430, 431t–432t
Basilia boardmani, 424f
Basilia hispida, 425
Batrachomyia, 386t–389t, 396
Batrachomyia silvarum, 398
Batrachyla, 192–193
Beauveria bassiana, 150, 166
Belisarius xambeui, 491
Bembix texana, 341
Berlinia, 369–370
Bertiella mucronata, 559
Bertiella studeri, 559
Besnoitia besnoiti, 337t
Bironella, 262t
Blaberus, 63
Blaberus craniifer, 71t–72t
Blaps, 135
Blarinobia simplex, 565–566
Blastocrithidia, 339
Blatta lateralis, 65, 65f, 71t–72t
Blatta orientalis, 62f, 65, 65f, 71t–73t
Blattella, 61
Blattella asahinai, 69
Blattella germanica, 62f, 68–69, 68f, 70f, 71t–73t, 681t–682t
Blawan, 392
Blomia, 555
Blomia tropicalis, 552–553
Bombus, 459–460, 477–478, 477f
Boophilus, 604, 625
Boophilus annulatus, 2–3
Booponus, 386t–389t
Borrelia, 37, 40–41, 40f
Borrelia baltazardii, 644t
Borrelia brasiliensis, 644t
Borrelia burgdorferi, 28, 40–41, 162, 165, 338, 626–627, 641–642, 641f
Borrelia burgdorferi sensu lato, 638–639
Borrelia causasica, 644t
Borrelia crocidurae, 644t

Borrelia dipodilli, 644t
Borrelia duttoni, 162, 626, 644t
Borrelia graingeri, 644t
Borrelia hermsii, 626, 644t
Borrelia hispanica, 644t
Borrelia kalaharica, 644t
Borrelia latyschewii, 644t
Borrelia mazzottii, 644t
Borrelia merionesi, 644t
Borrelia microti, 644t
Borrelia miyamotoi, 646
Borrelia neotropicales, 644t
Borrelia parkeri, 644t
Borrelia persica, 644t
Borrelia recurrentis, 3–4, 96–97, 99t
Borrelia tillae, 644t
Borrelia turicatae, 644t
Borrelia venezuelensis, 644t
Bothriocroton, 603t, 605, 616
Botta, 391–392
Botus, 391–392
Bovicola, 85, 89
Bovicola bovis, 81t, 84, 89, 90f, 100f
Bovicola caprae, 81t
Bovicola crassipes, 81t
Bovicola equi, 81t, 84, 90–91, 91f
Bovicola limbata, 81t
Bovicola ocellata, 81t
Bovicola ovis, 81t, 84, 89, 90f, 92, 100–101, 100f
Brachypelma, 516
Brachystegia, 369–370
Bradysia, 179
Brandbergia, 492
Bratrachomyia, 396f
Breinlia, 314
Brucella abortus, 162, 165
Brugia, 314
Brugia malayi, 37, 305, 309t
Brumptomyia, 194, 198
Brunnetia, 196
Bufolucilia, 386t–389t
Bunyaviridae, 695, 696t–700t, 702t
Burkholderia mallei, 165
Burkholderia pseudomallei, 165
Buteo ridgwayi, 364
Buthus, 490, 499, 501
Buthus occitanus, 490t, 500

C

Caccobius, 137
Calchas, 492
Callipepla californica, 230
Calliphora, 386t–389t, 398
Calliphora vicina, 52, 399f, 681t–682t
Calliphora vomitoria, 56t
Calliteara pundibunda, 443
Caloglyphus berlesei, 555–556
Calomys musculinus, 580
Calyptra, 452
Calyptra bicolor, 452
Calyptra fasciata, 452
Calyptra ophideroides, 452
Calyptra parva, 452
Calyptra pseudobicolor, 452
Calyptra thalictri, 452f
Camponotus, 466–467
Campylobacter jejuni, 71t–72t
Candida albicans, 28
Cannabis sativa, 53
Canthon, 141
Canthon pilularius, 137f
Caparinia, 584
Caparinia erinacei, 584–585
Caparinia tripilis, 584
Capreolus capreolus, 577
Caraboctonus, 492
Carabodes, 594t–595t
Carcinops, 141
Cardiofilaria, 314
Carios, 603t, 605, 613
Carios kelleyi, 608f, 618, 626
Carpoglyphus, 556
Carpoglyphus lactis, 545, 545f, 556
Castor canadensis, 567
Catacola, 449
Cavernicola, 111
Cazierius, 493
Cediopsylla simplex, 146f, 146t, 150, 152
Centruroides, 490, 497, 499–501
Centruroides balsasensis, 501
Centruroides elegans, 501
Centruroides exilicauda, 490t
Centruroides gracilis, 490
Centruroides guanensis, 490
Centruroides hentzi, 490
Centruroides infamatus, 501
Centruroides limpidus, 490t, 500–501
Centruroides noxious, 500–501
Centruroides sculpturatus, 25t–26t, 490, 499–501, 500f
Centruroides suffuses, 501
Centruroides tecomanus, 500–501
Centruroides vittatus, 490, 490f, 496f, 498f
Cephalopina titillator, 386t–389t, 409–410, 410f
Cephenemyia, 386t–389t, 394, 407–409, 416
Cephenemyia apicata, 409
Cephenemyia fontinella, 404
Cephenemyia jellisoni, 409, 409f
Cephenemyia phobifer, 409
Cephenemyia pratti, 409
Cephenemyia trompe, 409, 416
Cepheus, 595t
Ceratitis capitata, 688
Ceratophyllus, 153–156
Ceratophyllus gallinae, 146t, 147f, 153–154, 154f, 163–164
Ceratophyllus niger, 146t, 163–164
Ceratoppia, 595t
Ceratozetes, 594t–595t
Cercophonius, 289, 492
Cercopithecus aethiops, 286
Ceroxys latiusculus, 358
Cervus elaphus, 577
Cervus wapiti, 577
Cetonia, 140
Chactopsis, 491
Chaeopsestis ludovicae, 451f
Chaerilus, 491
Chagasia baffana, 279
Chalcosia, 443
Chalybion, 471–472
Chaoborus astictopus, 180, 180f
Cheiracanthium, 509–510, 518
Cheiracanthium inclusum, 518
Cheiracanthium japonicum, 518
Cheiracanthium mildei, 518
Cheiracanthium mordax, 518
Cheiracanthium punctorium, 518
Chelopistes meleagridis, 81t
Cheyletiella blakei, 564–565, 565f
Cheyletiella parasitivorax, 565, 565f
Cheyletiella yasguri, 564–565, 566f
Cheyletomorpha lepidopterorum, 547
Cheyletus eruditus, 547, 555–556
Cheyletus malaccensis, 547
Chilomastix, 356–357
Chirodiscoides caviae, 567
Chironomus, 181f
Chiroptonyssus, 539
Chiroptonyssus robustipes, 540–541, 560
Chlamydia, 28, 253
Chlamydia ornithophilia, 248t–249t
Chlamydia trachomatis, 357–358
Chlorotabanus, 328t
Chlorotabanus crepuscularis, 333
Choanotaenia infundibulum, 139, 139f, 360
Choresine, 131
Chorioptes, 584
Chorioptes bovis, 583–584, 584f
Chrysomya, 386t–389t, 398
Chrysomya bezziana, 175f, 386t–389t, 398–400
Chrysomya megacephala, 399f, 400
Chrysomya rufifacies, 390, 398, 400
Chrysops, 327–333, 328t, 330f, 330t, 336, 337t, 340
Chrysops atlanticus, 336
Chrysops callidus, 328f, 331
Chrysops cincticornis, 331, 331f
Chrysops dimidiata, 336
Chrysops dimidiatus, 336, 337t
Chrysops discalis, 337–338
Chrysops fuliginosus, 333
Chrysops silaceus, 336, 337t
Cimex, 19f, 119, 434
Cimex brevis, 124
Cimex hemipterus, 119–123, 120f
Cimex lectularius, 114, 119–124, 119f–121f, 123f, 681t–682t
Cimex pilosellus, 123
Cimex pipistrelli, 123
Cimexopsis nyctalis, 123
Cistudinomyia, 402
Cistudinomyia cistudinis, 386t–389t
Citellophilus, 158
Citrobacter, 71t–72t
Clethrionomys, 165
Clogmia, 192, 195, 198, 386t–389t
Clogmia albipunctata, 191f, 192, 198

Clogmia (Telmatoscopus) albipunctata, 394
Clostridium bifermentans, 208
Clostridium novii, 71t–72t
Clostridium perfringens, 71t–72t
Clubiona obesa, 518f
Cnephia, 176f
Cnephia ornithophilia, 244t, 248t–249t, 251–252
Cnephia pecuarum, 244t, 251, 253–254
Cobboldia, 410
Cobboldia elephantis, 386t–389t
Cochliomyia hominivorax, 386t–389t, 391, 398–400, 400f, 411, 413f, 415–416, 681t–682t, 687
Cochliomyia macellaria, 56t, 383, 386t–389t, 391, 400, 400f
Coelomycidium simulii, 254–255
Coelotes obesus, 508
Coloradia pandora, 442f
Colpocephalum, 85
Coltivirus, 630–631
Columbicola columbae, 83f
Comperia merceti, 75
Conicera tibialis, 183–184, 183f
Conispiculum, 314
Connochaetes taurinus, 577
Conorhinopsylla, 149
Copris, 137
Coquillettidia, 262, 262t, 264, 270, 273–274, 278–279
Coquillettidia perturbans, 266f, 284, 294
Coquillettidia venezuelensis, 221
Cordylobia, 386t–389t, 401
Cordylobia anthropophaga, 386t–389t, 401f, 411
Corethrella, 180
Corethrella brakeleyi, 180
Corethrella brunnea, 181f
Corethrella mitra, 181f
Corethrella wirthi, 180
Corvus brachyrhynchos, 294
Corynebacterium, 364
Corynebacterium lipoptenae, 431t–432t
Cosmiomma, 603t, 605
Coxiella burnetii, 155t, 161, 638
Crassacarus, 585
Creophilus maxillosus, 56t
Cricetomys gambianus, 160
Cricetus, 403–404
Cryptoiclus, 493
Cryptosporidium, 356–357
Ctenocephalides, 155t
Ctenocephalides canis, 146t, 151, 151f, 162–163
Ctenocephalides felis, 19f–20f, 146t, 147f–148f, 151, 151f, 154, 155t, 160, 162–163, 166
Ctenophthalmus, 158
Cuclutogaster heterographus, 81t, 93–94
Culex, 35, 37, 262, 264, 264f, 270, 272–273, 273f, 278–280, 288t, 292–294, 304–305, 308, 696t–700t
Culex annulirostris, 283t, 288t, 296, 696t–700t
Culex australicus, 262
Culex cedecei, 286t
Culex fuscocephala, 295
Culex gelidus, 288t, 295
Culex globocoxitus, 262
Culex melanoconion, 284–285, 696t–700t
Culex modestus, 288t
Culex molestus, 308
Culex nigripalpus, 283t, 288t, 295
Culex ocossa, 285, 286t
Culex pallens, 262
Culex panocossa, 285, 286t
Culex pipiens, 48–49, 209–210, 228, 262, 288t, 293–295, 299–300, 696t–700t
Culex pipiens fatigans, 2
Culex portesi, 286t
Culex pseudovishnui, 295
Culex quinquefasciatus, 35–36, 221, 262, 269f, 278–280, 288t, 294–295, 294f, 307–308, 681t–682t
Culex restuans, 264f
Culex (Melanoconion), 283t
Culex taeniopus, 286t
Culex tarsalis, 39–40, 283t, 284–285, 288t, 294, 696t–700t
Culex theileri, 299–300
Culex tritaeniorhynchus, 288t, 294–295, 696t–700t
Culex univittatus, 287, 288t, 293–294
Culex vishnui, 288t, 295
Culicoides, 19f, 213–223, 214f–215f, 219t–221t, 225, 227–229, 231–232, 231f, 250, 252
Culicoides actoni, 225
Culicoides adersi, 230t
Culicoides arakawae, 230, 230t
Culicoides arboricola, 230, 230t
Culicoides austeni, 221t, 223
Culicoides barbosai, 218, 221t, 222
Culicoides baueri, 230
Culicoides bedfordi, 219t–220t
Culicoides biguttatus, 216
Culicoides bolitinos, 219t–220t, 225, 228–229
Culicoides bottimeri, 230
Culicoides brevitarsis, 219t–220t, 224–225, 232
Culicoides chiopterus, 232
Culicoides circumscriptus, 217, 230t
Culicoides cornutus, 219t–220t
Culicoides crepuscularis, 216, 230t
Culicoides debilipalpis, 218, 225
Culicoides denningi, 217
Culicoides diabolicus, 219t–220t
Culicoides downesi, 230t
Culicoides dycei, 219t–220t
Culicoides edeni, 230, 230t
Culicoides floridensis, 218
Culicoides fulvithorax, 223
Culicoides fulvus, 219t–220t, 225
Culicoides furens, 214f, 216, 218, 221t, 222, 232
Culicoides glabrior, 216f
Culicoides grahamii, 221t, 223
Culicoides gulbenkiani, 219t–220t, 225
Culicoides guttifer, 230t
Culicoides guttipennis, 216–217
Culicoides haematopotus, 216, 230, 230t
Culicoides hinmani, 219t–220t, 230, 230t
Culicoides histrio, 219t–220t
Culicoides hollensis, 216, 218
Culicoides hortensis, 223
Culicoides imicola, 214, 219t–220t, 224–225, 228–229, 232, 696t–700t
Culicoides impunctatus, 230, 232
Culicoides inornatipennis, 221t, 223
Culicoides insignis, 219t–220t, 224–225, 232
Culicoides kingi, 219t–220t
Culicoides knowltoni, 230, 230t
Culicoides krameri, 223
Culicoides kumbaensis, 223
Culicoides lahillei, 222
Culicoides larifer, 225
Culicoides magnus, 225
Culicoides marksi, 219t–220t
Culicoides melleus, 216–218
Culicoides midges, 696t–700t
Culicoides milnei, 219t–220t, 223
Culicoides mississippiensis, 216–218
Culicoides nanus, 230
Culicoides niger, 216
Culicoides nivosus, 219t–220t
Culicoides nubeculosus, 216–217, 221t, 230t, 232
Culicoides obsoletus, 214, 219t–221t, 225, 229, 232
Culicoides occidentalis, 213–214
Culicoides oxystoma, 216–217, 219t–220t, 225, 229
Culicoides pallidipennis, 219t–220t
Culicoides pallidothorax, 219t–220t
Culicoides paraensis, 218, 219t–221t, 221–222, 230, 232, 696t–700t
Culicoides peregrinus, 219t–220t, 225
Culicoides phlebotomus, 221t, 222
Culicoides pulicaris, 214, 225, 232
Culicoides punctatus, 219t–220t, 229, 232
Culicoides pungens, 221t
Culicoides pusillus, 225
Culicoides pycnostictus, 223, 225
Culicoides ravus, 223
Culicoides rutshuruensis, 223
Culicoides sanguinipes, 200
Culicoides scoticus, 219t–220t, 225, 229
Culicoides shultzei, 225, 230t
Culicoides sonorensis, 213–214, 216–217, 217f, 219t–220t, 224–225, 227, 229, 232, 681t–682t, 685f, 696t–700t
Culicoides sphagnumensis, 230t
Culicoides spinosus, 216, 232
Culicoides stellifer, 216, 218, 225, 232
Culicoides stilobezzioides, 230t
Culicoides travisi, 216
Culicoides variipennis, 213–214, 216–218, 219t–221t, 228, 231–232, 696t–700t
Culicoides variipennis albertensis, 213–214

Culicoides variipennis australis, 213–214
Culicoides variipennis occidentalis, 213–214
Culicoides variipennis sonorensis, 213–214
Culicoides venustus, 216, 232
Culicoides victoriae, 221t, 231
Culicoides vitshumbiensis, 223
Culicoides wadi, 225
Culicoides wisconsinensis, 216–217
Culicoides zuluensis, 225
Culiseta, 262, 262t, 264, 270, 272–273, 278–279, 297, 696t–700t
Culiseta annulata, 298t
Culiseta inornata, 271, 275, 298t, 299
Culiseta melanura, 36, 283t, 284, 696t–700t
Cunila angustifolia, 141
Cupiennius, 509
Cuterebra, 386t–389t, 394, 403–404, 404f
Cuterebra baeri, 405–406
Cuterebra emasculator, 394
Cuterebra fontinella, 404f
Cyanocitta cristata, 230
Cyanocitta edeni, 230
Cyclocephala borealis, 137
Cylindrothorax, 133
Cylindrothorax bisignatus, 133, 134t
Cylindrothorax dusalti, 133, 134t
Cylindrothorax melanocephalus, 133, 134t
Cylindrothorax picticollis, 133, 134t
Cylindrothorax ruficollis, 134t
Cynomya, 386t–389t, 398
Cynomys, 157
Cyrnea colini, 73t, 74
Cytauxzoon, 627
Cytodites, 586
Cytodites nudus, 586, 591f, 592

D

Dabertia tricalcarata, 570f
Damalina, 84, 101
Dasymutilla occidentalis, 471, 471f
Datana integerrima, 455f
Deinocerites, 262, 262t, 275, 278
Deinocerites cancer, 271
Deinocerites pseudes, 285, 286t
Deltaretrovirus, 696t–700t
Demodex, 547–549, 585–586
Demodex bovis, 574
Demodex brevis, 547–548
Demodex canis, 573–574, 573f–574f
Demodex caprae, 574
Demodex cati, 573f, 574
Demodex cornei, 573–574
Demodex folliculorum, 547–548, 548f–549f
Demodex gatoi, 574
Demodex injai, 573–574
Demodex phylloides, 573f
Demodex tauri, 574
Dendrolimus, 441
Deraiophoronema, 314
Dermacentor, 603t, 604–605, 620
Dermacentor albipictus, 604, 631
Dermacentor andersoni, 3–4, 619t–620t, 620, 622f, 630–631, 696t–700t
Dermacentor marginatus, 604, 618, 620
Dermacentor nitens, 604, 620
Dermacentor occidentalis, 604, 620, 631, 636
Dermacentor parumapertus, 631
Dermacentor reticulatus, 604, 620
Dermacentor variabilis, 604, 609f, 611–612, 615, 617, 620, 622f, 634
Dermanyssus, 19, 20f, 537
Dermanyssus americanus, 538
Dermanyssus gallinae, 537–539, 538f, 560
Dermanyssus hirundinis, 538–539
Dermatobia, 393, 415
Dermatobia busckii, 396
Dermatobia funebris, 396
Dermatobia hominis, 252, 386t–389t, 393, 396–397, 403, 405f–406f
Dermatobia hydei, 396
Dermatobia immigrans, 396
Dermatobia melanogaster, 396
Dermatobia repleta, 396
Dermatobia simulans, 396
Dermatophagoides, 553
Dermatophagoides farinae, 552, 553f, 554–556, 681t–682t
Dermatophagoides pteronyssinus, 552–553, 555–556
Dermatophagoides scheremetewski, 552
Dermatophilus congolensis, 657–658
Dermestes, 56t
Dermestes maculatus, 140
Diachlorus, 328t
Dicrocoelium dendriticum, 483
Didelphis virginiana, 160
Didymocentrus, 493
Dinoponera gigantea, 460–461
Dipetalogaster maxima, 112–113
Dipetalonema, 223
Dipetalonema spirocauda, 99t
Diplocentrus, 493
Diplocentrus diablo, 493f
Diploptera punctata, 63, 71t–72t
Dipylidium caninum, 98, 99t, 101, 155t, 162, 163f, 165
Dirofilaria, 314
Dirofilaria immitis, 313–314
Dirofilaria roemeri, 340
Dirofilaria ursi, 248t–249t
Dolichovespula, 25t–26t, 460, 472
Dolichovespula arenaria, 461t, 473–474, 474t
Dolichovespula maculata, 461t, 472f, 473–475, 474t
Dolomedes, 510
Dolomedes fimbriatus, 510
Dometorina suramerica, 559
Dorcadia, 149
Dorcadia ioffi, 146t, 163
Draschia megastoma, 360–361, 363
Drosophila, 176f, 185, 383, 396, 396f
Drosophila funebris, 185
Drosophila melanogaster, 185, 185f, 386t–389t
Drosophila repleta, 185
Dysdera, 509
Dysdera crocata, 509

E

Eadiea brevihamata, 565–566
Echidnophaga gallinacea, 146t, 152–154, 152f, 163
Echidnophaga larina, 146t, 165
Echidnophaga myrmecobii, 146t, 164
Echinonyssus, 562
Echinonyssus butantanensis, 563
Echinophthirius horridus, 82t, 99t
Eciton, 460–461, 463–464
Ehrlichia, 636, 675–677
Ehrlichia canis, 652
Ehrlichia chaffeensis, 636, 675–677
Ehrlichia ewingii, 637, 653
Ehrlichia muris eauclairensis, 637, 653
Ehrlichia phagocytophilum, 653
Ehrlichia ruminantium, 624, 653f
Eimeria, 138–139
Elaeophora schneideri, 337t, 340, 340f
Elassoctenus harpax, 510
Eleodes, 135
Elephantoloemus indicus, 386t–389t
Entamoeba, 186, 356–357
Enterobacter, 71t–72t
Enterobius vermicularis, 72–73
Enterococcus, 71t–72t
Eobia apicifusca, 134
Epanaphe, 443
Epicauta, 20f, 24, 25t–26t, 27f, 133–134, 133f
Epicauta cinerea, 134t
Epicauta fabricii, 138
Epicauta flavicornis, 134t
Epicauta hirticornis, 134t
Epicauta lemniscata, 138
Epicauta maculata, 133, 133f, 134t
Epicauta occidentalis, 138
Epicauta pennsylvanica, 133, 133f, 134t, 138
Epicauta pestifera, 138
Epicauta sapphirina, 134t
Epicauta temexa, 138
Epicauta tomentosa, 134t
Epicauta vestita, 134t
Epicauta vittata, 133, 133f, 134t, 138
Epicoma, 443
Epidermoptes bilobatus, 568–569, 571f
Epidermoptes odontophori, 568–569
Epilohamminia, 595t
Erasmia, 443
Eratigena, 518–519
Eratigena agrestis, 508, 519, 519f
Eremaeus, 594t–595t
Eretmapodites, 262t
Eretmapodites chrysogaster, 275
Erinaceus europaeus, 584–585
Eristalinus, 184
Eristalis, 184, 386t–389t, 395
Eristalis arbustorum, 395
Eristalis dimidiata, 395
Eristalis tenax, 184, 184f, 395, 395f
Erysipelothrix rhusiopathiae, 162
Escherichia, 138–139
Escherichia coli, 70–72, 71t–72t, 356–357

Eucalliphora, 386t–389t, 398
Euclea delphinii, 446, 446f
Euhoplopsyllus glacialis, 146t, 149
Eulimdana, 99t
Eumacronychia, 402
Eumacronychia nigricornis, 386t–389t
Eumacronychia sternalis, 386t–389t
Eumusca, 362
Euoniticellus, 141
Eupelops, 595t
Euproctis, 441
Euproctis chrysorrhoea, 441, 448
Euroglyphus, 553
Euroglyphus maynei, 553, 554f, 555
Eurotium, 545
Eurycotis floridana, 62–63, 67–68, 67f
Euschoengastia, 559
Euscorpius, 491, 497
Eutrombicula, 559
Eutrombicula alfreddugesi, 543–544
Eutrombicula belkini, 54
Eutrombicula lipovskyi, 544
Eutrombicula splendens, 544
Euzetes, 595t

F

Falco deiroleucus, 364
Fannia, 345, 351–353, 355–356, 359, 365, 390, 396–398, 397f, 404–405
Fannia canicularis, 346t, 350f, 353f, 355–356, 355f, 386t–389t, 396–397
Fannia femoralis, 346t
Fannia incisurata, 396–397
Fannia manicata, 396–397
Fannia scalaris, 56t, 346t, 386t–389t, 396–397
Fannia thelaziae, 359
Felicola subrostrata, 81t, 92, 101–102
Ficalbia, 262t
Filoviridae, 702t
Filovirus, 702t
Flaviviridae, 695, 696t–700t
Flavivirus, 280–282, 281t, 287–288, 696t–700t
Folyella, 314
Forcipomyia, 213, 216, 222
Forcipomyia townsvillensis, 221t, 231
Formica, 459, 461–462, 462f, 467, 483
Francisella tularensis, 99t, 155t, 161, 280, 337–338, 337t, 338f, 620, 646, 656–657
Frankliniella tritici, 10
Fregata magnificens, 432–433
Furcoribula, 595t
Fusca, 369–370, 373, 379

G

Gahrliepia, 559
Galindomyia, 262t
Galumna, 559, 594t–595t
Gasterophilus, 386t–389t, 394, 410
Gasterophilus haemorrhoidalis, 386t–389t
Gasterophilus intestinalis, 386t–389t, 393, 410
Gasterophilus nasalis, 386t–389t, 393
Gasterophilus nigricornis, 386t–389t, 410
Gasterophilus pecorum, 386t–389t, 410
Gastronyssus bakeri, 585
Gazalina, 443
Gazella thompsonii, 577
Gedoelstia, 386t–389t
Gedoelstia cristata, 409–410
Geospiza fortis, 364
Geotrupes, 140
Giardia, 356–357
Glaucomys volans, 96, 161
Gliricola porcelli, 81t, 102
Glossina, 338–339, 369–370, 369f, 371f, 373, 379
Glossina austeni, 369–370, 373, 378t
Glossina brevipalpis, 370, 378t
Glossina fusca, 370, 378t
Glossina fuscipes, 370, 376–377, 378t
Glossina fuscipleuris, 371f, 378t
Glossina longipalpis, 370, 378t
Glossina longipennis, 370
Glossina medicorum, 370, 373
Glossina morsitans, 370, 374f, 377, 378t
Glossina morsitans morsitans, 681t–682t, 686
Glossina pallidipes, 370, 377, 378t, 381
Glossina palpalis, 3, 370, 376–377, 378t
Glossina swynnertoni, 370, 377, 378t
Glossina tabaniformis, 370, 378t
Glossina tachinoides, 370, 376–377, 378t
Glossina vanhoofi, 378t
Glycyphagus domesticus, 553, 553f
Glycyphagus hypudaei, 537f
Gongylonema, 73t, 74–75
Gongylonema neoplasticum, 73t, 74–75
Gongylonema pulchrum, 72–75, 73t, 140
Goniocotes gallinae, 81t
Goniodes dissimilis, 81t
Goniodes gigas, 81t
Goniops, 327–328, 328t
Goniops chrysocoma, 331
Greniera, 241
Gruninomyia mira, 386t–389t, 407
Gymnopais, 239–241
Gyropus ovalis, 81t, 83f, 102
Gyrostigma, 386t–389t, 410

H

Habibiella, 493
Habronema exigua, 355
Habronema irritans, 355
Habronema microstoma, 363
Habronema muscae, 360–361, 363
Habronema scambus, 359
Hadogenes, 492
Hadogenes troglodytes, 492
Hadronyche, 508, 517
Hadronyche cerberea, 517
Hadronyche formidabilis, 517
Hadronyche infensa, 517, 517f
Hadronyche versuta, 517
Hadruroides, 492
Hadrurus, 492
Haemagogus, 262, 262t, 264, 270, 272–273, 279, 288t, 290
Haemaphysalis, 603t, 604–605, 696t–700t
Haemaphysalis leporispalustris, 604–605, 615, 631
Haemaphysalis longicornis, 648
Haemaphysalis punctata, 604–605
Haemaphysalis spinigera, 604–605, 619t–620t
Haematobia, 390
Haematobia atripalpis, 361
Haematobia irritans, 348f, 350f, 353f, 363f, 681t–682t
Haematobosca alcis, 345
Haematomyzus elephantis, 81t, 83f–84f, 84
Haematopinus, 83
Haematopinus asini, 84–85, 90–91, 91f
Haematopinus eurysternus, 82t, 90, 90f
Haematopinus quadripertusus, 82t, 90
Haematopinus suis, 82t, 91–92, 91f, 99t, 100
Haematopinus tuberculatus, 82t, 89
Haematopota, 327–328, 328t, 330, 337t
Haematopota americana, 328
Haematosiphon inodorus, 123
Haemodipsus ventricosus, 82t, 93, 102
Haemogamasus, 562
Haemogamasus liponyssoides, 541, 562
Haemogamasus pontiger, 541
Haemophilus influenzae, 186
Haemoproteus, 229
Haemoproteus brayi, 229
Haemoproteus columbae, 431t–432t, 433
Haemoproteus danilewskyi, 230, 230t
Haemoproteus desseri, 230t
Haemoproteus fringillae, 230t
Haemoproteus kochi, 229
Haemoproteus lophortyx, 230, 431t–432t
Haemoproteus lopohortyx, 431t–432t
Haemoproteus maccallumi, 431t–432t
Haemoproteus mansoni, 230t
Haemoproteus meleagridis, 223, 229–230, 230t
Haemoproteus metchnikovi, 340
Haemoproteus nettionis, 230t
Haemoproteus sacharovi, 431t–432t
Haemoproteus velans, 230t
Hafnia alvei, 71t–72t
Halarachne, 590
Hantavirus, 702t
Harpactirella, 515–516
Harpactirella lightfooti, 515–516
Harpyrhynchoides, 585
Heizmannia, 262t
Helichrysum angustifolium, 547
Heliocopris, 141
Helodon decemarticulatus, 248t–249t
Hemiceratoides heiroglyphica, 451f
Hemiceratoides hieroglyphica, 443–444, 450
Hemileuca maia, 447, 447f
Hemiscorpius, 493
Hemiscorpius lepturus, 493

Hepatocystis, 229
Hepatocystis brayi, 230t
Hepatocystis kochi, 230t
Hepatozoon erhardovae, 165
Hepatozoon muris, 562–563
Hepatozoon pitymysi, 165
Hepatozoon sciuri, 165
Hermanniella, 594t–595t
Hermetia illucens, 56t, 183, 183f, 386t–389t, 394–395, 395f
Herpetomonas, 229
Herpyllus blackwalli, 509
Herpyllus ecclesiasticus, 509
Heterodoxus spiniger, 81t, 84–85, 92, 93f
Heterometrus, 493–494
Heteromys, 285
Heteronebo, 493
Heteroscorpion, 493
Hippelates neoproboscideus, 186
Hippobosca, 425–426
Hippobosca equina, 428, 428f
Hippobosca longipennis, 427–428, 431t–432t
Hippobosca struthionis, 426
Hippobosca variegata, 428
Hirundo, 538–539
Hirundo rustica, 561
Hister, 56t
Hodgesia, 262t
Hoffmannihadrurus, 492
Holotrichius innesi, 108
Homo erectus, 86
Homo sapiens, 86
Hoplopleura captiosa, 82t
Hoplopleura pacifica, 82t, 92–93, 160
Hoplopsyllus anomalus, 146t, 158
Horaiella, 192
Hottentotta, 490
Hottentotta hottentotta, 496–497
Hottentotta tamulus, 500
Hyalomma, 603t, 605, 696t–700t
Hyalomma asiaticum, 605, 696t–700t
Hyalomma detritum, 605
Hyalomma dromedarii, 228, 612f, 617
Hyalomma impeltatum, 611
Hyalomma marginatum, 696t–700t
Hyalomma marginatum marginatum, 605
Hyalomma truncatum, 605, 659
Hybomitra, 327–329, 328t, 331, 333, 334f, 337t, 340
Hybomitra bimaculata, 332
Hybomitra hinei, 333
Hybomitra illota, 333
Hybomitra lasiophthalma, 332
Hydrochoerus hydrochoeris, 577
Hydrotaea, 347, 348f, 349, 356, 364–365, 390, 397
Hydrotaea aenescens, 346t, 356
Hydrotaea ignava, 346t, 348f, 350f, 353f, 356
Hydrotaea leucostoma, 56t
Hydrotaea meteorica, 346t, 356, 359
Hydrotaea rostrata, 386t–389t
Hydrotaea scambus, 346t
Hylemya, 386t–389t, 396
Hylesia, 443, 453
Hymenolepis, 72–73, 131–132
Hymenolepis diminuta, 131–132, 139, 155t, 163, 165
Hymenolepis nana, 131–132, 139, 155t, 163, 165
Hypochrosis hyadaria, 450–451
Hypochrosis irrorata, 450f
Hypochrosis pyrrhophaeata, 451f
Hypodectes propus, 585
Hypoderma, 386t–389t, 406–407, 407f, 413–417
Hypoderma actaeon, 406–407
Hypoderma bovis, 386t–389t, 393, 406, 407f–408f, 413–414
Hypoderma diana, 406–407
Hypoderma lineatum, 386t–389t, 393, 406, 407f–408f
Hypoderma rostrata, 397
Hypoderma sinensis, 406–407
Hypoderma tarandi, 386t–389t, 393, 406–407, 416
Hypozetes, 595t

I

Icosiella neglecta, 192–193
Icosta albipennis, 431t–432t
Icosta americana, 431t–432t, 432
Icosta angustifrons, 431t–432t
Icosta ardeae boutaurinorum, 431t–432t
Icosta hirsuta, 431t–432t
Icosta holoptera holoptera, 431t–432t
Icosta nigra, 431t–432t
Icosta rufiventris, 431t–432t
Idiommata blackwalli, 508
Illiberis, 443
Isoberlinia, 369–370
Isometrus maculatus, 490
Iurus, 492
Ixodes, 603t, 604, 609, 696t–700t
Ixodes affinis, 604
Ixodes cookei, 696t–700t
Ixodes dentatus, 604
Ixodes jellisoni, 641
Ixodes ovatus, 604
Ixodes pacificus, 604, 606f, 637, 641
Ixodes persulcatus, 604, 619t–620t, 641
Ixodes ricinus, 40–41, 604, 611–612, 616, 619t–620t, 641, 681t–682t, 696t–700t
Ixodes scapularis, 604, 607f, 616–617, 619t–620t, 620–623, 641, 681t–682t
Ixodes sculptus, 631
Ixodes spinipalpis, 604, 631, 641

J

Johnbelkinia, 262t

K

Kerteszia bellator, 304t
Kerteszia cruzii, 304t
Kimia, 262t
Kirkioestrus, 386t–389t
Klebsiella, 71t–72t
Knemidokoptes jamaicensis, 569–570
Knemidokoptes mutans, 569–570, 572f
Knemidokoptes pilae, 570
Kobus kob, 588
Kolotl, 493
Kutzerocoptes gruenbergi, 578

L

Lachnosterna, 140
Laelaps, 562
Laelaps echidninus, 160, 542, 562–563
Laelaps jettmari, 541
Laelaps nuttalli, 563
Lagoa crispata, 445
Lagoa pyxidifera, 445
Laminosioptes cysticola, 571, 572f
Lampona, 509
Lasioderma serricorne, 131
Lasiohelea, 213, 218, 221–222
Latoia, 443
Latrodectus, 25t–26t, 134–135, 507, 510–511, 514–515, 523–525, 524f, 527–529
Latrodectus bishopi, 524–527, 526f
Latrodectus curacaviensis, 525, 527
Latrodectus erebus, 528
Latrodectus geometricus, 524–526, 524f, 526f
Latrodectus hasselti, 525, 527, 527f
Latrodectus hesperus, 524–525, 526f
Latrodectus katipo, 525, 527
Latrodectus mactans, 25t–26t, 523–527, 525f
Latrodectus tredecimguttatus, 525, 527–528
Latrodectus variolus, 525
Leeuwenhoekia, 559
Leishmania, 201f, 203–207, 203f–204f, 675
Leishmania (Viannia), 205
Leishmania aethiopica, 199t
Leishmania amazonensis, 199t, 205–207
Leishmania archibaldi, 199t
Leishmania braziliensis, 199t, 206–207
Leishmania (Viannia) braziliensis, 205
Leishmania chagasi, 199t, 203
Leishmania colombiensis, 199t
Leishmania donovani, 199t, 205, 207–208
Leishmania garnhami, 199t
Leishmania guyanensis, 199t, 205–207
Leishmania (Viannia) guyanensis, 205
Leishmania infantum, 199t, 203, 205, 208
Leishmania killicki, 199t, 203
Leishmania lainsoni, 199t
Leishmania major, 199t, 206–207, 206f
Leishmania mexicana, 199t, 206–208
Leishmania naiffi, 199t
Leishmania panamensis, 199t

Leishmania peruviana, 199t, 206–207
Leishmania pifanio, 199t
Leishmania shawi, 199t
Leishmania tropica, 203, 205–207
Leishmania venezuelensis, 199t
Leiurus, 490, 499, 501
Leiurus quinquestratus, 24, 25t–26t, 28f, 490t, 495, 496f, 500–501, 500f
Lentivirus, 696t–700t
Leopoldamys, 559
Lepidoglyphus destructor, 547, 552–553
Leporipoxvirus, 696t–700t
Leptinillus, 140
Leptinus, 140
Leptocimex, 120
Leptocimex boueti, 119–120
Leptoconops, 213–214, 216, 218, 222
Leptoconops becquaerti, 218, 221t, 222
Leptoconops kerteszi, 218
Leptoconops linleyi, 218
Leptomonas ctenocephali, 166
Leptopsylla segnis, 146t, 154, 154f, 160, 163
Leptotrombidium, 543–544, 559
Leptotrombidium akamushi, 543, 544t, 558, 558f
Leptotrombidium arenicola, 544, 544t
Leptotrombidium deliense, 543, 544t
Leptotrombidium fletcheri, 544, 544t
Leptotrombidium pallidum, 544, 544t
Leptotrombidium pavlovskyi, 544, 544t
Leptotrombidium scutellare, 544, 544t
Lepus, 219t–220t, 403–404
Leucocytozoon, 229–230, 251–252
Leucocytozoon cambournaci, 248t–249t
Leucocytozoon caulleryi, 223, 229–230, 230t
Leucocytozoon dubreuili, 248t–249t
Leucocytozoon icteris, 248t–249t
Leucocytozoon lovati, 248t–249t
Leucocytozoon neavei, 248t–249t
Leucocytozoon sakharoffi, 248t–249t
Leucocytozoon schoutedeni, 248t–249t
Leucocytozoon simondi, 248t–249t, 252
Leucocytozoon smithi, 248t–249t, 252
Leucocytozoon tawaki, 248t–249t
Leucocytozoon toddy, 248t–249t
Leucocytozoon ziemanni, 248t–249t
Leucophaea maderae, 73t
Leucotabanus, 328t
Leucotabanus annulatus, 331–333
Liacarus, 594t–595t
Liebstadia, 594t–595t
Limatus, 262t, 274–275
Limenitis arthemis astyanax, 440f
Limnonectes leporinus, 181f
Linognathus, 89
Linognathus africanus, 82t, 84–85, 92, 100f–101f, 101
Linognathus ovillus, 82t
Linognathus pedalis, 82t
Linognathus setosus, 82t, 84–85, 92, 93f, 101
Linognathus stenopsis, 82t
Linognathus vituli, 82t, 89, 90f
Linshcosteus, 109
Liocheles australasiae, 496–497
Liohippelates, 185–186
Liohippelates collusor, 185–186
Liohippelates flavipes, 186
Liohippelates pallipes, 185–186
Liohippelates puruanus, 186
Liohippelates pusio, 185–186, 185f
Lipeurus caponis, 81t, 93–94, 94f
Liponyssoides, 537
Liponyssoides sanguineus, 539, 539f, 556–557
Lipoptena, 423, 426, 428–429
Lipoptena cervi, 428–429, 429f, 431t–432t
Lipoptena depressa, 428, 431t–432t
Lipoptena mazamae, 425f, 429–430, 431t–432t
Lisposoma, 492
Listeria monocytogenes, 162, 165
Listrophoroides cucullatus, 567
Listrophorus synaptomys, 567f
Litomosoides, 433
Loa loa, 336–337, 337t, 338f
Loa loa papionis, 336
Loberopsyllus, 140
Lobocraspis, 451
Lobocraspis griseifusa, 439–440
Loiana, 314
Lonomia achelous, 25t–26t, 447
Lonomia obliqua, 448f
Loxosceles, 25t–26t, 507, 510, 512, 519–520, 520f, 522–523, 528–529
Loxosceles arizonica, 519–520
Loxosceles blanda, 519–520
Loxosceles deserta, 519–520
Loxosceles devia, 519–520
Loxosceles laeta, 25t–26t, 519–520, 522–523, 529
Loxosceles obliqua, 25t–26t
Loxosceles reclusa, 25t–26t, 519–523, 519f–522f, 529
Loxosceles rufescens, 519–520, 529
Lucilia, 386t–389t
Lucilia coeruleiviridis, 56t
Lucilia cuprina, 398, 413, 415, 417, 681t–682t, 689
Lucilia hominivorax, 689
Lucilia illustris, 412
Lucilia sericata, 56t, 57f, 398, 402, 412, 681t–682t
Lutzia, 262t
Lutzomyia, 191–193, 198, 201, 696t–700t
Lutzomyia anthophora, 199t, 207
Lutzomyia antunesi, 199t
Lutzomyia apache, 198–200
Lutzomyia ayacuchensis, 199t
Lutzomyia ayrozai, 199t
Lutzomyia camposi, 197
Lutzomyia carpenteri, 197
Lutzomyia carrerai, 199t
Lutzomyia christopheri, 199t
Lutzomyia columbiana, 199t, 202
Lutzomyia cruzi, 196f, 199t
Lutzomyia dasipodogeton, 201
Lutzomyia davisi, 201
Lutzomyia diabolica, 198, 199t, 207
Lutzomyia dysponeta, 197
Lutzomyia evansi, 197–198, 199t
Lutzomyia flaviscutellata, 199t
Lutzomyia gomezi, 195, 197–198, 199t
Lutzomyia hartmanni, 199t
Lutzomyia intermedia, 193, 199t
Lutzomyia lichyi, 196, 199t
Lutzomyia longipalpis, 193, 195f, 196–197, 199t, 681t–682t
Lutzomyia longipalpus, 195–197
Lutzomyia migonei, 193, 199t
Lutzomyia nuneztovari, 199t
Lutzomyia olmeca, 198, 199t
Lutzomyia ovallesi, 199t
Lutzomyia panamensis, 195, 197–198, 199t
Lutzomyia paraensis, 199t
Lutzomyia peruensis, 202
Lutzomyia pessoai, 199t
Lutzomyia pessoana, 195–198
Lutzomyia reducta, 199t
Lutzomyia rorotaensis, 196
Lutzomyia sanguinaria, 197–198
Lutzomyia serrana, 196
Lutzomyia shannoni, 196, 197f, 198–200, 208
Lutzomyia spinicrassa, 199t
Lutzomyia squamiventris, 199t
Lutzomyia townsendii, 199t
Lutzomyia trapidoi, 195–201, 199t, 208
Lutzomyia trinidadensis, 196, 199t
Lutzomyia triramula, 197
Lutzomyia ubiquitalis, 199t, 201
Lutzomyia umbratilis, 199t, 201
Lutzomyia verrucarum, 193, 196–198, 199t, 202, 209
Lutzomyia vespertilionis, 197
Lutzomyia vexator, 208
Lutzomyia wellcomei, 198
Lutzomyia ylephiletor, 198, 199t, 200–201, 208
Lutzomyia youngi, 199t
Lutzomyia yucumensis, 199t
Lycoriella ingenua, 179
Lycosa, 509–510
Lycosa tarentula, 509–510, 514–515
Lyctocoris campestris, 107
Lymantria dispar, 448, 448f
Lytta phalerata, 134t
Lytta vesicatoria, 133, 133f, 134t

M

Mackiena, 559
Macracanthorhynchus, 131–132
Macracanthorhynchus hirudinaceus, 140
Macracanthorhynchus ingens, 140
Macrodactylus subspinosus, 138
Macropocopris, 140
Macropus major, 638
Macropus minor, 638
Macrothele, 508
Malaya, 262t

Mansonella, 222
Mansonella ozzardi, 218–223, 221t, 248t–249t, 250
Mansonella perstans, 218–221, 221t, 223
Mansonella streptocerca, 218–221, 221t, 223
Mansonia, 262, 262t, 264, 264f, 273–274, 278–279, 283t, 286t, 308
Mansonia annulifera, 308
Maorigoeldia, 262t
Margaropus, 603t, 605
Martes pennanti, 577
Mastigoproctus giganteus, 24
Mastomys natalensis, 157
Mastophora, 508–509
Mastophorus muris, 73t, 74–75
Maxomys, 559
Medicago sativa, 138
Megacormus, 491
Megalopyge, 25t–26t
Megalopyge opercularis, 25t–26t, 444–445, 445f
Megaphobema, 517
Megarthroglossus, 149
Megaselia, 183f, 386t–389t
Megaselia assimilis, 397
Megaselia autumnalis, 397
Megaselia pabulorum, 397
Megaselia scalaris, 183–184, 183f, 386t–389t, 395
Megaselia spiracularis, 386t–389t, 395
Megaselia stabulans, 397
Megistopoda aranea, 425
Melanolestes picipes, 107
Melanoplus sanguinipes, 200
Melolontha, 140
Melophagus, 423–424, 426
Melophagus montanus, 426
Melophagus ovinus, 100–101, 423, 423f, 426–427, 426f, 431t–432t, 434f
Menacanthus, 85
Menacanthus stramineus, 81t, 83f, 93, 94f, 102, 102f
Menopon gallinae, 81t, 93–94, 94f, 102
Mercomyia, 328t
Meromyza americana, 185
Mesobuthus martensii, 28
Mesocestoides, 559
Metacnephia lyra, 241, 248t–249t
Metoposarcophaga, 402
Metoposarcophaga importuna, 386t–389t
Microleon, 443
Microlichus americanus, 568–569
Microlichus avus, 568–569
Microlichus macdonaldi, 568–569
Microlynchia pusilla, 431t–432t
Microtus, 403–404, 539, 544, 559
Mimomyia, 262t, 273
Misumenoides, 511
Molinema, 314
Monema, 443
Moniezia autumnalis, 594t
Moniezia benedeni, 594t
Moniezia denticulata, 594t
Moniezia expansa, 594t
Moniezia neumani, 594t
Moniliformis dubius, 73t
Moniliformis moniliformis, 73t, 140
Monomorium minimum, 460–461
Monomorium pharaonis, 470–471, 470f
Moraxella, 361
Moraxella bovis, 361
Morellia, 390
Morganella morganii, 71t–72t
Morpho achillaena, 450
Musca, 349, 354, 362, 397
Musca autumnalis, 346t, 347f–349f, 361f, 397
Musca biseta, 354
Musca bovis, 361
Musca domestica, 56t, 346t, 347f–348f, 350f, 353f, 360f, 386t–389t, 397, 397f, 681t–682t
Musca levida, 355, 359
Musca sorbens, 354, 357f, 397
Musca vetustissima, 141, 353–354
Muscidifurax, 365
Muscidifurax zaraptor, 365f
Muscina, 347, 351, 353, 358, 365, 386t–389t, 397
Muscina assimilis, 355
Muscina levida, 346t
Muscina stabulans, 346t, 348f, 350f, 353f, 386t–389t, 397f, 398
Mus musculus, 92, 154, 160, 539, 556, 566
Musophagobius cystodorus, 570f
Mycobacterium avium, 139
Mycobacterium leprae, 71t–72t
Mycoplasma, 28
Mycoplasma coccoides, 98, 99t, 102
Mycoplasma muris, 98, 99t, 102
Mycoplasma parvum, 100
Mycoplasma suis, 98
Mydaea, 386t–389t, 397–398
Mylabris bifasciata, 134t
Mylabris cichorii, 134t
Myobia, 565–566
Myobia murismusculi, 566, 566f
Myobia musculi, 566
Myocoptes musculinus, 567–568
Myodopsylla insignis, 146t
Myrmecia, 25t–26t, 465–466
Myrmecia gulosa, 25t–26t
Myrmecia pilosula, 25t–26t
Myrmica, 465–466

N

Nairovirus, 696t–700t
Natada nasoni, 447f
Nauphoeta cinerea, 71t–72t
Nebo, 493
Nebo hierichonticus, 493
Necator americanus, 72–73
Necrobia rufipes, 55–57, 56t
Necrophila americana, 56t, 57f
Nemapalpus nearctica, 192
Nemorhina, 369–370
Neobellieria bullata, 681t–682t
Neobellieria citellivora, 386t–389t, 402
Neocalchas, 492
Neocnemidocoptes gallinae, 570–571
Neocordylobia, 386t–389t
Neocuterebra squamosa, 386t–389t, 410
Neohaematopinus sciuropteri, 96
Neolipoptena, 423, 426, 428–429
Neolipoptena ferrisi, 429, 431t–432t
Neomusca, 397–398
Neomusca (Philornis), 386t–389t
Neomyia, 390, 397
Neopsylla, 158
Neorickettsia sennetsu, 636
Neoschoengastia, 559
Neotoma, 117, 403–404
Neotoma albigula, 207
Neotoma micropus, 207
Neotrombicula, 559
Neotrombicula autumnalis, 543–544, 543f
Neottiophilum, 396
Neottiophilum praeustrum, 386t–389t
Neotunga, 148–149
Nicrophorus, 56t
Nocardia, 71t–72t
Nopoiulus kochii, 6
Norape ovina, 445, 445f
Nosema pulicis, 150
Nosoma, 603t, 605
Nosopsyllus, 155t
Nosopsyllus fasciatus, 146t, 153, 154f, 158, 160, 163, 165
Nothogreniera fergusoni, 239f
Notochaeta bufonovoria, 386t–389t
Notoedres, 578
Notoedres cati, 552, 578–579, 578f–579f
Notoedres centrifera, 579
Notoedres muris, 579–580
Nullibrotheas alleni, 491
Nuttalliella, 603t
Nuttalliella namaqua, 603–604, 606
Nylanderia fulva, 467
Nymphalis antiopa, 450f
Nyssorhynchus albimanus, 304t
Nyssorhynchus albitarsis, 304t
Nyssorhynchus allopha, 304t
Nyssorhynchus aquasalis, 304t
Nyssorhynchus argyritarsis, 304t
Nyssorhynchus braziliensis, 304t
Nyssorhynchus darlingi, 304t
Nyssorhynchus nuneztovari, 304t

O

Ochotonia lindneri, 386t–389t, 406–407
Ochrogaster lunifer, 455
Odocoileus, 101
Odocoileus hemionus, 340
Odontacarus, 559
Odontomachus haematodus, 466–467
Oeciacus, 123
Oeciacus hirundinis, 123
Oeciacus vicarius, 123, 284

Oedemagena tarandi, 406–407
Oestroderma, 386t–389t, 406–407, 408f
Oestroderma potanini, 386t–389t
Oestrus, 407–409
Oestrus ovis, 384, 386t–389t, 389, 393–394, 409, 409f
Oiceoptoma, 56t
Oiclus, 493
Olfersia bisulcata, 431t–432t
Olfersia fumipennis, 431t–432t
Olfersia sordida, 431t–432t
Olfersia spinifera, 431t–432t
Oligella urethralis, 71t–72t
Omosita, 56t
Onchocerca cervicalis, 221t, 223, 230–231, 231f
Onchocerca cervipedis, 248t–249t, 252
Onchocerca dewittei, 248t–249t
Onchocerca dukei, 248t–249t, 252
Onchocerca gibsoni, 221t, 231
Onchocerca gutturosa, 221t, 231, 248t–249t, 252
Onchocerca lienalis, 248t–249t, 252
Onchocerca lupi, 248t–249t, 252
Onchocerca ochengi, 248t–249t, 252
Onchocerca ramachandrini, 248t–249t, 252
Onchocerca reticulata, 221t, 231
Onchocerca skrjabini, 248t–249t
Onchocerca sweetae, 221t
Onchocerca takaokai, 248t–249t, 252
Onchocerca tarsicola, 248t–249t
Onchocerca virginianus, 340
Onchocerca volvulus, 245–247, 248t–249t, 250, 336–337
Onchocerca, 231, 252
Onirion, 262t
Onthophagus, 137, 140–141
Onychocerus albitarsis, 131
Ophionyssus, 539
Ophionyssus natricis, 541, 542f, 562
Ophioptes, 585
Ophyra, 356
Ophyra aenescens, 356
Ophyra leucostoma, 356, 399f
Opifex, 262t, 275
Opisthacanthus lepturus, 492
Opistophthalmus, 493–494
Oppiella, 595t
Orbivirus, 201, 223–227, 696t–700t
Orchopeas howardi, 96, 146t, 154–156, 155t, 161
Oribatella, 595t
Oribatula, 595t
Orientia tsutsugamushi, 544, 544t, 557, 559
Orius insidiosus, 107
Ornithocoris toledoi, 123
Ornithoctona erythrocephala, 431t–432t
Ornithoctona fusciventris, 431t–432t
Ornithodoros, 603t, 608f, 613, 626, 643, 652, 696t–700t
Ornithodoros brasiliensis, 644t, 659
Ornithodoros coriaceus, 648
Ornithodoros costalis, 644t
Ornithodoros erraticus, 626, 644t
Ornithodoros graingeri, 644t
Ornithodoros hermsi, 644t
Ornithodoros kairouanensis, 644t
Ornithodoros lahorensis, 659
Ornithodoros macrocanus, 644t
Ornithodoros merionesi, 644t
Ornithodoros moubata, 114, 626, 644t, 648
Ornithodoros occidentalis, 644t
Ornithodoros parkeri, 644t
Ornithodoros porcinus, 626, 644t
Ornithodoros rudis, 644t
Ornithodoros savignyi, 626, 659
Ornithodoros sonrai, 644t
Ornithodoros talaje, 644t
Ornithodoros tartakowskyi, 644t
Ornithodoros tholozani, 644t
Ornithodoros turicata, 612f, 644t, 648
Ornithodoros verrucosus, 644t
Ornithodoros zumpti, 644t
Ornithoica confluenta, 431t–432t
Ornithoica vicina, 431t–432t
Ornithomya anchineuria, 431t–432t
Ornithomya avicularia, 423f
Ornithomya bequaerti, 431t–432t
Ornithomya fringillina, 421
Ornithonyssus, 18–19, 20f, 539
Ornithonyssus bacoti, 160, 539–540, 539f, 557, 560–561
Ornithonyssus bursa, 540, 540f, 561
Ornithonyssus sylviarum, 540, 541f, 561–562
Oropsylla montana, 146t, 158, 158f
Orthobunyavirus, 280–282, 281t, 297, 696t–700t
Orthoperus, 131
Orthopodomyia, 262, 262t
Orthopodomyia alba, 270
Oryctolagus, 403–404
Oryctolagus cuniculus, 163, 565
Orygia pseudotsugata, 443
Oryzomys, 285
Oscinella frit, 185
Oswaldofilaria, 314
Otobius, 603t, 606
Otobius lagophilus, 606, 631
Otobius megnini, 606, 613, 626
Otodectes cynotis, 587, 588f
Oxacis, 134
Oxycopis, 134
Oxycopis mcdonaldi, 135f
Oxylipeurus polytrapezius, 81t
Oxyspirura mansoni, 73t, 74
Oxyspirura parvorum, 73t, 74
Oxytrigona tataira, 477

P

Pachychoeromyia praegrandis, 386t–389t
Pachycondyla chinensis, 466–467
Paederus, 24, 25t–26t, 134–135, 136f, 136t
Paederus alternans, 136t
Paederus amazonicus, 136t
Paederus australis, 135, 136t
Paederus brasiliensis, 135, 136t
Paederus columbinus, 135, 136t
Paederus cruenticollis, 135, 136t
Paederus eximius, 136t
Paederus ferus, 136t
Paederus fuscipes, 135, 136t, 138
Paederus islae, 136t
Paederus laetus, 136t
Paederus melampus, 136t
Paederus ornaticornis, 136t
Paederus puncticollis, 136t
Paederus riparius, 135f, 136t
Paederus rufocyaneus, 136t
Paederus sabaeus, 135, 136t
Paederus signaticornis, 135, 136t
Paederus tamulus, 136t
Paleoleishmania proterus, 205
Paleomyia burmitis, 205
Pallasiomyia, 406–407
Pallisiomyia antilopum, 386t–389t
Pamphobeteus, 508
Pandinoides, 493–494
Pandinopsis, 493–494
Pandinurus, 493–494
Pandinus, 493–494, 497
Pandinus gambiensis, 497
Pandinus imperator, 493–494, 494f
Panstrongylus chinai, 110t
Panstrongylus geniculatus, 109f
Panstrongylus lignarius, 110t
Panstrongylus megistus, 109f, 110t, 113, 116–117
Panstrongylus rufotuberculatus, 110t
Pantoea, 71t–72t
Parabelminus, 111
Parabuthus, 490
Parachipteria, 594t–595t
Paracnephia strenua, 238f
Parafilaria, 362
Parafilaria bovicola, 361–362
Parafilaria downsi, 364
Parafilaria multipapillosa, 361
Paralucilia, 386t–389t, 398
Paranoplocephala mamillana, 594t
Paraponera, 480
Paraponera clavata, 24, 25t–26t, 27f, 460–461, 480
Parapoxvirus, 165
Parasa, 443
Parasa chloris, 447f
Parasa indetermina, 446f
Paraspinturnix globosus, 585
Parcoblatta, 61–64, 70
Paruroctonus boreus, 489
Paruroctonus luteolus, 498f
Passeromyia, 356, 386t–389t, 397–398
Pavlovskiata, 406–407
Pavlovskiatasubgutturosae, 386t–389t
Pedicinus, 82t
Pediculus humanus, 19f–20f, 99t
Pediculus humanus capitis, 82t, 83f, 88, 88f
Pediculus humanus humanus, 82t, 84f, 87–88, 87f, 158, 160–161, 681t–682t
Pelecitus, 314
Pelecitus fulicaeatrae, 99t

Peloptulus, 595t
Peloribates, 595t
Penicillium, 545
Pepsis, 471, 471f
Pepsis chrysothemis, 24, 29f
Pergalumna, 595t
Pericoma, 192–194, 193f
Pericoma funebris, 194
Periplaneta, 19f, 61, 63–64, 66, 70, 75
Periplaneta americana, 62f, 65–66, 66f, 71t–73t, 74
Periplaneta australasiae, 62f, 66, 66f, 71t–72t
Periplaneta brunnea, 62f–63f, 66–67, 67f
Periplaneta fuliginosa, 62f, 67, 67f
Peromyscus, 40–41, 285, 403–404
Peromyscus gossypinus, 285, 404f
Peromyscus leucopus, 404f
Peromyscus polionotus, 160
Pestivirus, 696t–700t
Peucetia viridans, 510
Phanaeus, 141
Pharyngobolus africanus, 386t–389t
Pharyngomyia, 386t–389t
Pheidole, 483
Pheromermis myopis, 341
Phidippus johnsoni, 510
Philanthus triangulum, 465
Philoliche, 329–330, 330f
Philonthus, 141
Philornis, 356, 364, 364f, 397–398
Philornis downsi, 364
Philornis pici, 364
Phlebotomus, 191–194, 193f, 198, 200–201, 696t–700t
Phlebotomus aculeatus, 199t
Phlebotomus alexandri, 199t
Phlebotomus ansarii, 199t
Phlebotomus argentipes, 195–198, 207, 209
Phlebotomus ariasi, 196–197, 199t
Phlebotomus bergeroti, 193, 196
Phlebotomus caucasicus, 195–197, 199t, 207
Phlebotomus celiae, 199t
Phlebotomus chinensis, 199t
Phlebotomus duboscqui, 193, 197, 199t
Phlebotomus guggisbergi, 199t
Phlebotomus kandelakii, 199t
Phlebotomus kazeruni, 196
Phlebotomus langeroni, 199t
Phlebotomus longicuspis, 199t
Phlebotomus longiductus, 199t
Phlebotomus longipes, 199t
Phlebotomus martini, 197, 199t, 207, 209
Phlebotomus neglectus, 199t
Phlebotomus orientalis, 196–197, 199t
Phlebotomus papatasi, 191f, 193, 195–198, 199t, 200–201, 209, 681t–682t, 696t–700t
Phlebotomus pedifer, 199t
Phlebotomus perfiliewi, 195, 199t, 200–201, 209
Phlebotomus perniciosus, 196–197, 199t, 200–201
Phlebotomus rossi, 199t
Phlebotomus salehi, 199t
Phlebotomus sergenti, 198, 199t
Phlebotomus smirnovi, 199t
Phlebotomus tobbi, 194, 197, 199t
Phlebotomus transcaucasicus, 197, 199t
Phlebotomus vansomerenae, 199t
Phlebovirus, 200, 280–282, 281t, 297, 630f, 632
Phobetron pithecium, 446, 446f
Phoneutria, 507, 509, 518
Phoneutria keyserlingi, 509, 518
Phoneutria nigriventer, 25t–26t, 509, 518, 518f
Phorcotabanus cinereus, 333
Phormia, 398
Phormia regina, 56t, 58, 386t–389t, 398, 399f, 412–413, 681t–682t
Phormictopus, 508
Phortica variegata, 185, 681t–682t
Phyllophaga, 140
Physaloptera caucasica, 140
Physaloptera praeputialis, 73t, 74
Physaloptera rara, 73t, 74
Physocephalus sexalatus, 140
Piagetiella, 85
Pilogalumna, 595t
Piophila, 395f
Piophila casei, 56t, 184–185, 184f, 386t–389t, 395–396
Pireneitega pyrenaea, 508, 510
Pisaurina, 510
Plasmodium, 35, 229, 278, 300–301, 304–305, 313, 433, 627
Plasmodium anomaluri, 312
Plasmodium brasilianum, 300, 313
Plasmodium falciparum, 300, 317
Plasmodium gallinaceum, 312
Plasmodium knowlesi, 302–303
Plasmodium landauae, 312
Plasmodium malariae, 300, 313
Plasmodium ovale, 302
Plasmodium pulmophilum, 312
Plasmodium relictum, 35
Plasmodium vivax, 41, 302, 317
Platydracus, 56t
Platynothrus, 594t–595t
Platypsyllus, 140
Plecia nearctica, 179
Plesiochactas, 491
Ploiaria domestica, 209–210
Pneumocoptes jellisoni, 592
Pneumocoptes penrosei, 592
Pneumonyssoides caninum, 590, 590f–591f
Pneumonyssus simicola, 589–590, 590f
Pogonomyrmex, 25t–26t, 464–467, 469–470, 470f, 480
Pogonomyrmex badius, 469
Pogonomyrmex barbatus, 469
Pogonomyrmex californicus, 469
Pogonomyrmex maricopa, 25t–26t
Pogonomyrmex occidentalis, 469
Polistes, 25t–26t, 460, 472, 473f, 476
Polistes annularis, 461t, 476
Polistes apachus, 461t, 476
Polistes aurifer, 461t, 476
Polistes carolina, 461t
Polistes dominula, 461t, 476
Polistes exclamans, 461t, 476
Polistes fuscatus, 461t, 465f, 476
Polistes metricus, 461t, 476
Polistes rubiginosus, 461t, 476
Polybia occidentalis, 25t–26t
Polyplax serrata, 82t, 85, 89, 92, 99t
Polyplax spinulosa, 82t, 92–93, 93f, 99t, 102, 160
Portschinskia, 386t–389t, 406–407
Poylchromophilus, 433
Poxviridae, 696t–700t
Proctolaelaps pygmaeus, 537
Proctophyllodes glandarinus, 569f
Proechimys, 285
Prosimulium, 240, 243
Prosimulium impostor, 248t–249t
Prosimulium magnum, 240
Prosimulium mixtum, 239f, 244t
Prosimulium tomosvaryi, 248t–249t
Prosimulium ursinum, 242
Prosthenorchis elegans, 73t, 75
Prosthenorchis spirula, 73t, 75
Proteus, 70–72
Proteus mirabilis, 71t–72t
Proteus rettgeri, 71t–72t
Proteus vulgaris, 71t–72t
Prothoracic spiracles, 346
Protocalliphora, 356, 386t–389t, 400–401, 400f
Protoiurus, 492
Protophormia, 386t–389t, 398
Protophormia terraenovae, 399f, 413
Protoribates, 595t
Protospirura bonnei, 73t, 75
Protospirura muricola, 73t, 75
Provespa, 460
Przhevalskiana, 386t–389t, 406–407
Psalydolytta fusca, 134t
Psalydolytta substrigata, 134t
Psammolestes, 111
Psammomys obesus, 210
Pseudodiamesa, 181f
Pseudolynchia brunnea, 431t–432t
Pseudolynchia canariensis, 429, 430f, 431t–432t
Pseudomonas, 71t–72t, 134–135
Pseudomyrmex ejectus, 466–467
Pseudouroctonus, 492
Pseudouroctonus apacheanus, 495f
Psorergates simplex, 575, 575f
Psorobia ovis, 575
Psorophora, 262, 262t, 264, 270, 272–273, 279, 283t, 286t, 298t
Psorophora ferox, 404–405, 405f
Psoroptes, 581
Psoroptes ovis, 581f–582f, 582–583, 586–587
Psychoda, 192–193, 195–196, 198, 386t–389t
Psychoda albipennis, 198, 386t–389t, 394

Psychoda alternata, 195, 198, 208
Psychoda cinerea, 198
Psychoda pacifica, 198
Psychoda phalaenoides, 194
Psychoda severini, 195, 198
Pterostichus, 139
Pthirus pubis, 20f, 82t, 88–89, 89f
Pulex irritans, 146t, 150, 150f, 158, 160, 162–163
Pulex simulans, 146t, 150, 154–156, 163
Punctoribates, 594t–595t
Pycnoscelus surinamensis, 69, 69f, 73t
Pyemotes, 547
Pyemotes herfsi, 547
Pyemotes tritici, 546–547, 547f
Pyogenes, 364
Pyrota insulata, 138
Pyrrharctia isabella, 448–449
Pythium, 179

R

Radfordia, 565–566
Radfordia ensifera, 566f
Radfordiella, 560, 585
Radfordiella oudemansi, 561f
Raillietia, 587–588
Raillietiella hemidactyli, 73t, 75
Raillietina cesticillus, 139
Raillietina echinobothrida, 483
Raillietina tetragona, 483
Rallietia auris, 588f
Rana esculenta, 192–193
Rasahus biguttatus, 107
Rasahus thoracicus, 107
Rattus, 160, 202, 403–404, 539, 544, 557, 559
Rattus norvegicus, 92–93, 156, 160, 567
Rattus rattus, 92–93, 156, 160, 539–540, 567
Reduvius, 115
Reduvius personatus, 107, 124
Reoviridae, 695, 696t–700t
Retroviridae, 696t–700t
Rhabdoviridae, 695, 696t–700t
Rhagio, 182f
Rhinoestrus, 386t–389t, 407–408
Rhinoestrus purpureus, 386t–389t, 409
Rhipicentor, 603t, 605
Rhipicephalus, 603t, 604
Rhipicephalus appendiculatus, 604, 617–618, 659
Rhipicephalus (B.) decoloratus, 625
Rhipicephalus microplus, 681t–682t
Rhipicephalus (Boophilus) microplus, 615, 625
Rhipicephalus sanguineus, 228, 604, 615, 615f, 618, 634, 652
Rhodnius, 112f
Rhodnius ecuadoriensis, 110t
Rhodnius pallescens, 110t
Rhodnius prolixus, 109f, 110t, 111–113, 116–118, 681t–682t
Rhombomys opimus, 207, 210
Rickettsia aeschlimannii, 635
Rickettsia akari, 539, 556–557
Rickettsia conorii, 98, 618, 635
Rickettsia felis, 155t, 161
Rickettsia massiliae, 635
Rickettsia melophagi, 431t–432t, 433
Rickettsia mooseri, 159–160
Rickettsia parkeri, 624, 636
Rickettsia philipii, 620, 636
Rickettsia prowazekii, 88, 95–96, 99t, 155t, 160–161
Rickettsia rickettsii, 3–4, 98, 633–634, 634f
Rickettsia tsutsugamushi, 557
Rickettsia typhi, 147, 155t, 159–161
Rivoltasia bifurcata, 568–569
Ropalidia marginata, 25t–26t
Rubus, 543–544
Runchomyia, 262t, 275
Rupicapra rupicapra, 577
Ruttenia loxodontis, 386t–389t, 410

S

Sabethes, 262t, 275, 278–279, 288t
Sabethes chloropterus, 290
Salmonella, 70–72, 71t–72t, 138–139, 356–357
Salmonella aureus mastitis, 364
Salmonella bareilly, 71t–72t
Salmonella bovis-morbificans, 71t–72t, 72
Salmonella bredeny, 71t–72t
Salmonella chester, 138–139
Salmonella enterica, 183
Salmonella enteritidis, 161
Salmonella newport, 71t–72t
Salmonella oranienburg, 71t–72t
Salmonella panama, 71t–72t
Salmonella paratyphi-B, 71t–72t
Salmonella pyogenes, 71t–72t
Salmonella sitiens, 356
Salmonella stilesi, 363
Salmonella typhi, 70–72, 71t–72t, 98
Salmonella typhimurium, 71t–72t, 72, 138–139
Sancassania berlesei, 555–556
Sancassania mycophaga, 555–556
Saprinus, 56t
Sarconema eurycerca, 99t
Sarcophaga, 386t–389t, 402, 402f
Sarcophaga bullata, 56t
Sarcophaga haemorrhoidalis, 56t, 402f
Sarcopromusca arcuata, 405f
Sarcopromusca pruna, 404–405
Sarcoptes, 549
Sarcoptes scabiei, 549–551, 550f, 575–578, 681t–682t
Saurositus, 314
Scarabaeus, 140
Scathophaga, 56t
Sceliphron, 471–472, 472f
Scheloribates, 559, 594t–595t
Scheloribates atahualpensis, 559
Sciurus carolinensis, 165
Sciurus vulgaris, 165
Scopelodes, 443
Scorpio, 493–494
Scotophaeus blackwalli, 509
Scutovertex, 559, 595t
Seadornavirus, 630–631
Segestria florentina, 510
Sepsis, 56t
Sergeia, 229
Sergentomyia, 193, 198, 200
Sergentomyia antennata, 197–198
Sergentomyia bedfordi, 197–198
Sergentomyia theodori, 197
Sericopelma, 508
Serretia, 71t–72t
Sessinia, 134
Sessinia kanak, 134
Shannoniana, 262t
Shigella, 186, 356–357
Shigella dysenteriae, 71t–72t
Shunsennia, 559
Sibine stimulea, 445–446, 445f
Sicarius, 510
Sigmodon, 285
Sigmodon hispidus, 285
Silvius, 327–328, 328t, 340
Simulium, 200, 237, 240, 243, 247–250, 248t–249t
Simulium adersi, 248t–249t
Simulium albivirgulatum, 247–250, 248t–249t
Simulium amazonicum, 222, 244t, 248t–249t
Simulium anatinum, 248t–249t, 252
Simulium angustitarse, 248t–249t
Simulium annulus, 242, 248t–249t, 251–252, 251f–252f
Simulium arakawae, 244t, 248t–249t
Simulium arcticum, 254
Simulium argentiscutum, 248t–249t
Simulium aureum, 248t–249t
Simulium bidentatum, 248t–249t, 252
Simulium bovis, 248t–249t, 252
Simulium buissoni, 244t
Simulium callidum, 248t–249t
Simulium cholodkovskii, 244t
Simulium chutteri, 244t
Simulium colombaschense, 244t, 253–254
Simulium congareenarum, 248t–249t
Simulium daisense, 248t–249t
Simulium damnosum, 238, 240–243, 247–250, 248t–249t, 252
Simulium damnosum sensu stricto, 250
Simulium decimatum, 244t
Simulium decorum, 242, 248t–249t
Simulium dieguerense, 248t–249t
Simulium dycei, 248t–249t
Simulium equinum, 244t
Simulium erythrocephalum, 244t, 248t–249t, 252–254
Simulium ethiopiense, 248t–249t
Simulium exiguum, 243, 247–250, 248t–249t
Simulium fallisi, 248t–249t
Simulium guianense, 247–250, 248t–249t, 256

Simulium incrustatum, 244t, 248t–249t
Simulium jenningsi, 241, 244t, 245, 248t–249t, 252, 254
Simulium johannseni, 242, 244t, 251–252, 252f
Simulium jujuyense, 244t
Simulium kilibanum, 248t–249t
Simulium konkourense, 248t–249t
Simulium kurense, 244t
Simulium kyushuense, 248t–249t
Simulium latipes, 248t–249t
Simulium leonense, 248t–249t
Simulium limbatum, 248t–249t
Simulium lineatum, 244t
Simulium luggeri, 244t, 251
Simulium maculatum, 244t
Simulium mengense, 248t–249t
Simulium meridionale, 244t, 248t–249t, 251–252
Simulium metallicum, 242, 247–250, 248t–249t
Simulium neavei, 247–250, 248t–249t
Simulium nigrogilvum, 244t
Simulium noelleri, 241
Simulium notatum, 248t–249t, 253
Simulium ochraceum, 242, 244t, 247–250, 248t–249t
Simulium oitanum, 248t–249t
Simulium ornatum, 244t, 248t–249t, 252–254
Simulium oyapockense, 242, 244t, 247–250, 248t–249t
Simulium parnassum, 242, 244t
Simulium penobscotense, 244t
Simulium pertinax, 244t
Simulium pintoi, 222
Simulium posticatum, 243, 244t
Simulium quadrivittatum, 244t, 248t–249t
Simulium rasyani, 248t–249t
Simulium rendalense, 248t–249t
Simulium reptans, 241, 244t, 248t–249t, 253–254
Simulium rostratum, 240–241
Simulium ruficorne, 248t–249t
Simulium rugglesi, 242, 244t, 248t–249t, 252
Simulium sanchezi, 222
Simulium sanctipauli, 248t–249t
Simulium sanguineum, 244t, 248t–249t
Simulium sanguinipes, 200
Simulium sirbanum, 247–250
Simulium slossonae, 244t, 248t–249t, 252
Simulium soubrense, 248t–249t
Simulium squamosum, 248t–249t
Simulium tescorum, 244t
Simulium thyolense, 248t–249t
Simulium truncatum, 241
Simulium turgaicum, 244t
Simulium usovae, 248t–249t
Simulium vampirum, 241–242, 244t, 251f, 253–254
Simulium venustum, 238f, 242–243, 244t, 245f, 248t–249t
Simulium vernum, 248t–249t
Simulium vittatum, 238f–239f, 240, 242, 244t, 248t–249t, 253
Simulium woodi, 248t–249t
Simulium yahense, 248t–249t
Sinea diadema, 107
Siphunculina, 185–186
Sitophilus granarius, 131
Skrjabini, 252
Skrjabinofilaria, 314
Sodalis glossinidius, 425
Solenopotes, 89
Solenopotes capillatus, 82t, 89–90, 90f
Solenopsis, 25t–26t, 27f, 466–469
Solenopsis geminata, 467–469
Solenopsis invicta, 23f, 25t–26t, 467–469, 467f–468f, 480f, 483
Solenopsis richteri, 467–468, 467f, 483
Solenopsis xyloni, 467–469, 483
Spalangia, 365
Spaniopsis, 182
Spatiodamaeus, 595t
Spermophilopsis leptodactylus, 207
Spermophilus, 402
Sphaerobothria hoffmani, 528
Sphecius speciosus, 472, 472f
Sphingobacterium, 71t–72t
Sphingobacterium mizutae, 71t–72t
Spilopsyllus cuniculi, 146t, 150, 155t, 163–164
Spirocerca lupi, 140
Spirura rytipleurites, 73t
Splendidofilaria fallisensis, 248t–249t
Staphylococcus aureus, 71t–72t, 88, 161, 522
Staphylococcus epidermidis, 71t–72t
Stearibia, 396
Steatoda, 510–511
Steatoda andina, 510–511
Steatoda capensis, 527
Steatoda grossa, 510–511
Steatoda nobilis, 510–511
Steatoda paykulliana, 510–511
Stegomyia, 263, 696t–700t
Stegopterna, 241
Steinernema carpocapsae, 75, 150, 166
Stenoponia tripectinata, 146t, 148
Stephanofilaria, 363
Stephanofilaria stilesi, 363
Sternostoma tracheacolum, 589, 589f
Stilbometopa impresssa, 431t–432t
Stilbometopa podopostyla, 431t–432t
Stilesia globipunctata, 594t
Stilesia hepatica, 594t
Stilesia vittata, 594t
Stomoxys, 390
Stomoxys calcitrans, 346t, 348f–350f, 352f–353f, 362f, 404–405, 681t–682t
Stomoxys nigra, 356
Stonemyia, 327–328, 328t
Streptococcus, 138–139
Streptococcus faecalis, 71t–72t
Streptococcus pyogenes, 88
Strictia carolina, 341
Strobiloestrus, 386t–389t, 406–407
Suidasia, 555–556
Suidasia nesbitti, 545–546, 545f
Suidasia pontifica, 556
Suipoxvirus, 696t–700t
Suncus murinus, 160
Supella, 61
Supella longipalpa, 62f, 68, 68f, 73t
Superstitionia, 491
Superstitionia donensis, 491
Suragina, 182
Sycorax silacea, 192–193
Sycorax wampukrum, 192–193
Sylvilagus, 219t–220t, 312, 403–404
Symphoromyia, 182, 182f, 404–405
Syringophilus bipectinalis, 571f

T

Tabanus, 176f, 327–329, 328t, 330f, 330t, 331–333, 334f, 337t, 340
Tabanus abactor, 330–331, 333
Tabanus atratus, 327, 332
Tabanus brucei, 338–339
Tabanus calens, 332
Tabanus congolense, 339
Tabanus conterminous, 341
Tabanus equalis, 333
Tabanus evansi, 338–339
Tabanus fairchildi, 329
Tabanus fuscicostatus, 334
Tabanus imitans, 331f
Tabanus laticornis, 340
Tabanus marginalis, 175f
Tabanus moderator, 333
Tabanus nigrovittatus, 327, 332–333, 335f, 340–341
Tabanus pallidescens, 333
Tabanus punctifer, 331–332, 337–338
Tabanus simulans, 327
Tabanus subsimilis, 330–332
Tabanus sulcifrons, 330–331, 333f
Tabanus trimaculatus, 328f
Tabanus vivax, 339
Tabanus wilsoni, 333
Tadarida brasiliensis, 540–541, 560
Taenia saginata, 139
Tarsonemus, 556
Tarsonemus granarius, 555–556
Tarsonemus hominis, 556
Tarsoporosus, 493
Tegenaria agrestis, 519
Telenomus fariai, 118–119
Tenebrio, 139
Tenebrio molitor, 131, 139, 547
Tenebrio obscurus, 131
Tetrameres americana, 73t, 74
Tetrameres fissipina, 73t, 74
Tetramorium caespitum, 470, 470f
Tetrapetalonema, 223
Thaumetopoea, 25t–26t
Thaumetopoea processionea, 25t–26t, 441
Thaumetopoea wilkinsoni, 25t–26t
Theileria, 627, 649

Theileria annulata, 651
Theileria taurotragi, 659
Thelazia, 358, 361
Thelazia californiensis, 358–359
Thelazia callipaeda, 185, 358
Thelazia gulosa, 358, 361
Thelazia lacrymalis, 361
Thelazia skrjabini, 361
Thelyphassa apicata, 134
Thelyphassa lineata, 134
Theraphosa, 517
Thereva, 386t–389t
Thogotovirus, 630f
Thyreophagus entomophagus, 555
Thyridanthrax, 381
Thysaniezia giardi, 594t
Thysanosoma actinioides, 594t
Tinamus major, 85
Tinea pellionella, 56t
Tineola bisselliella, 56t
Tipula, 176f, 178f
Tityus, 490, 496–497, 499–501
Tityus bahiensis, 501
Tityus cambridgei, 501
Tityus metuendus, 496–497
Tityus serrulatus, 25t–26t, 490t, 496f, 501, 501f
Tityus trinitatis, 501
Tityus trivittatus, 501
Togaviridae, 695, 696t–700t
Topomyia, 262t, 275
Toxoplasma gondii, 70–72
Toxorhynchites, 262, 262t, 264, 267, 270–273, 278–279
Toxorhynchites amboinensis, 269f
Toxorhynchites brevipalpis, 264f, 265f
Trachelas, 511
Trachelas volutus, 511
Tracheomyia macropi, 386t–389t, 407–408, 409f
Tragelaphus scriptus, 377
Trechona, 508
Trechona venosa, 508
Treponema pertenue, 186
Triatoma, 109, 111
Triatoma barberi, 114
Triatoma brasiliensis, 110t
Triatoma carrioni, 110t
Triatoma dimidiata, 110f, 110t
Triatoma gerstaeckeri, 117
Triatoma guasayana, 110t
Triatoma infestans, 109f, 110t, 111–113, 116–118
Triatoma lecticularia, 117
Triatoma maculata, 110t
Triatoman picturata, 114
Triatoma pallidipennis, 109f
Triatoma patagonica, 110t
Triatoma protracta, 113
Triatoma recurva, 117
Triatoma rubida, 113, 117
Triatoma rubrofasciata, 108f, 109, 118
Triatoma sanguisuga, 117
Triatoma sordida, 110t, 111–112
Tribolium, 131, 135, 139
Tribolium castaneum, 135, 136f
Tribolium confusum, 135, 136f
Trichillum, 140
Trichinella spiralis, 163, 165–166
Trichobius intermedius, 433
Trichodectes canis, 81t, 84–85, 92, 93f, 98, 99t, 101, 162
Trichoecius, 21f
Trichomyia, 192
Trichophaga tapetzella, 56t
Trichophyton erinacei, 584–585
Trichoprosopon, 262t, 270
Trichoprosopon digitatum, 278–279
Trichoribates, 594t–595t
Trichospirura leptostoma, 75
Trichuris trichuria, 72–73
Trinoton anserinum, 81t, 99t
Trinoton querquedulae, 81t
Tripteroides, 262t
Trixacarus, 580
Trixacarus caviae, 567, 580, 580f
Trixacarus diversus, 580
Trochilocoetes, 82
Trochoderma ornatum, 131
Troglocormus, 491
Troglodytes aedon, 538–539
Troglotayosicus, 491
Trogoderma, 137f
Trogoderma angustum, 131
Trogoderma glabrum, 131
Trox, 56t
Trypanosoma, 338–339, 431t–432t
Trypanosoma avium, 248t–249t, 431t–432t, 432
Trypanosoma brucei, 375–379, 378t
Trypanosoma brucei gambiense, 375, 375f, 378t
Trypanosoma brucei rhodesiense, 375, 375f, 377, 378t
Trypanosoma confusum, 248t–249t, 253
Trypanosoma congolense, 377–379, 378t
Trypanosoma conorhini, 118
Trypanosoma corvi, 248t–249t, 253
Trypanosoma cruzi, 109, 110t, 112–119, 113f, 115f, 124
Trypanosoma duttoni, 165
Trypanosoma equinum, 339
Trypanosoma evansi, 337t, 338–339
Trypanosoma gambiense, 374–375
Trypanosoma grayi, 379
Trypanosoma grosi, 165
Trypanosoma hannai, 431t–432t
Trypanosoma hedricki, 124
Trypanosoma lewisi, 155t, 165
Trypanosoma malophagium, 431t–432t, 432
Trypanosoma musculi, 165
Trypanosoma myoti, 124
Trypanosoma nabiasi, 155t, 165
Trypanosoma neotomae, 165
Trypanosoma numidae, 248t–249t
Trypanosoma rabinowitschi, 165
Trypanosoma rangeli, 117–118
Trypanosoma rhodesiense, 374–375
Trypanosoma simiae, 377–379, 378t
Trypanosoma suis, 377–379, 378t
Trypanosoma theileri, 339
Trypanosoma uniforme, 377–379, 378t
Trypanosoma vespertilionis, 433
Trypanosoma vivax, 337t, 377–379, 378t
Trypanosoma wirthi, 180
Trypocalliphora linderi, 386t–389t
Tunga, 148–149, 162, 164
Tunga monositus, 146t, 148–149
Tunga penetrans, 146t, 148–149, 152–153, 152f–153f, 162, 164, 164f
Tunga trimamillata, 146t, 153, 162, 164
Twinnia, 241
Typhaea stercorea, 139
Typhlochactas mitchelli, 491
Tyrolichus casei, 546
Tyrophagus, 556
Tyrophagus longior, 545, 546f, 556
Tyrophagus putrescentiae, 545–546, 552–553, 555–556

U

Udaya, 262t
Unguizetes, 595t
Uraba lugens, 449
Uranotaenia, 262, 262t, 270
Uroctonus, 492
Urocyon cinereoargenteus, 577
Urodacus, 493
Uropsylla tasmanica, 146t, 149
Ursicoptes, 580–581
Ursicoptes americanus, 580–581
Urubambates, 594t–595t

V

Vaejovis carolinianus, 492, 497–499
Vermipsylla, 149
Vermipsylla alakurt, 146t, 163
Verrallina, 262t
Vesiculovirus, 198–200, 696t–700t
Vespa, 25t–26t, 460, 466, 475
Vespa crabro, 461t, 472, 475
Vespula, 25t–26t, 460, 472, 474, 481f, 484
Vespula acadica, 474t
Vespula alascensis, 461t, 473–475, 474t
Vespula atropilosa, 474, 474t
Vespula consobrina, 474t
Vespula flavopilosa, 461t, 474–475, 474t
Vespula germanica, 461t, 473–475, 474t
Vespula maculifrons, 461t, 464, 473–475, 473f, 474t
Vespula pensylvanica, 461t, 473–475, 474t, 484
Vespula rufa, 474
Vespula squamosa, 461t, 473, 474t, 475, 481f
Vespula sulphurea, 461t, 474t, 475
Vespula vidua, 474
Vespula vulgaris, 475
Viannia, 205
Vibrio, 71t–72t
Vibrio cholerae, 181
Vitalius sorocabae, 515f

W

Walchia, 559
Waltonella, 314
Warileya, 198
Wasmannia, 466–467
Wasmannia auropunctata, 467–469
Wenzella, 149
Wohlbachia, 209–210
Wohlfahrtia, 386t–389t, 402
Wohlfahrtia magnifica, 402
Wohlfahrtia nuba, 383, 402
Wohlfahrtia opaca, 402
Wohlfahrtia vigil, 402
Wolbachia, 247, 250
Wuchereria bancrofti, 2, 28–29, 35–36, 39, 305–306, 309t
Wyeomyia, 262, 262t, 264, 270–271, 275, 278–279
Wyeomyia smithii, 270–271

X

Xenillus, 595t
Xenopsylla, 151, 154–156, 155t, 160
Xenopsylla astia, 146t, 160
Xenopsylla bantorum, 146t, 160
Xenopsylla brasiliensis, 146t, 160
Xenopsylla cheopis, 146t, 151, 151f, 156, 158, 158f, 160, 163, 165
Xylobates, 595t
Xylocopa californica, 477
Xylocopa varipuncta, 477
Xylocopa virginica, 477, 477f
Xyloryctes, 140

Y

Yersinia pestis, 71t–72t, 150, 155t, 156–159, 158f
Yersinia pseudotuberculosis, 162
Yunkeracarus, 592

Z

Zelonia australiensis, 248t–249t
Zeugnomyia, 262t
Zygodontomys, 285
Zygodontomys brevicauda, 157
Zygoribatula, 594t–595t
Zythos, 450–451

This page intentionally left blank

Subject Index

'*Note*: Page numbers followed by "f" indicate figures, "t" indicate tables.'

A

Aardvark, tsetse flies, 373
Absconding, bees, 479
Acalyptratae, 175
Acanthocephala, 131–132, 140
Acari. *See* Mites
Acariasis
  definition, 533
  enteric acariasis, 556
  internal acariasis, 555–556
  pulmonary acariasis, 555–556
  urinary acariasis, 556
Acaricides, 660–661
Acarinism, 533
Acarophobia, 7, 533
Accidental myiasis, 383
Accumulated degree hours, 58
Actinopodidae, 507–508
Adenolymphangitis, 307
Adenotrophic viviparity, 424
Adephaga, 129
Adipocere formation, 54
AFLP-PCR, 675
African blue louse, 92, 100f, 101
African hedgehog mange mite, 585
African horse sickness (AHS), 227–228, 227f
Africanized honey bee, 478–479, 479f
African sleeping sickness, 374–377, 375f
African swine fever, 626, 652
African tampan, 605–606, 626
Agas, 213
Agelenidae, 508
Aggregation pheromones, 63–64
Agramonte, Aristides, 290
Ague, 300
AHS. *See* African horse sickness (AHS)
Air-sac mite, 591f
Air trumpet, 266
Alakurts, 146–147
Allergic reactions
  cockroach, 73–74
  equine allergic dermatitis, 231–232, 231f
  flea, 164
  hymenopterous sting reactions, 481t, 482
  mite-induced allergy, 552–555
  silk, 453
  stages, 5–6
  sudden death, 53
  tick bite, 648
Alphavirus, mosquito-borne viruses, 283–287, 283f, 283t, 286t
Amastigote, 115f
Amblycera, 79
American bird mite, 538
American cockroach, 65–66, 66f
American dog tick, 622f
American trypanosomiasis. *See* Chagas disease
Ammotrechidae, 505
Amphibians
  biting midges, 217–218
  chiggers, 542
  flesh flies, 402
  mansonellosis, 250
  mites, 590–593
  mosquitoes, 276
  psychodidae, 192–193
  ticks, 608f
  toad blow flies, 398
  triatomines, 110–111
Amplicons, 673–675
Amplification, parasites, 35
Amplifying host, 35
Ampulla, 272
Analgesics, 8
Anaphylactic shock, 481–482
Anaphylaxis, 481–482
Anaplasmosis, 652–653
  bovine, 655
  canine, 654
  public health importance, 637
  veterinary importance, 655
Anautogenous, 177, 242, 352, 391
Annealing, DNA, 674f
Anophelinae, 261–262, 262t
Anoplura, 79
Anteater, trypanosomiasis, 117
Antelope
  bots, 406–407, 409
  deer ked, 429
  ear mite, 586–587
  skin-piercing moths, 452
  ticks, 626, 631, 658
  trypanosomiasis, 377
Antennal fossae, 146
Anterior-station transmission, 38
Anthocoridae, 119
Anthomyiidae, myiasis, 385, 396
Anthophoridae, 477
Anthrax, tabanid transmission, 338
Anthropogenic changes, 48
Anthropophagic vector, 36–37
Anticoagulants, 8
Antigenic complex, 224
Antimicrobial peptides (AMPs), 28
Antivenins, 499
Ants. *See also* Hymenoptera
  behavior and ecology, 464–465
  fire ants, 467–469, 467f, 469f
  harvester ant, 466–467, 469–470, 470f
  life history, 463–464
  morphology, 460–463
  pavement ant, 470, 470f
  pharoah ant, 470–471, 470f
  prevention and control, 483–484
  public health importance, 479–482
  taxonomy, 459–460, 460t
  venoms, 465–466
  veterinary importance, 483
Apamin, 466
Apidae, 459–460, 477
Apinae, 477
Apocrita, 459
Apoidea, 459
Aposematic coloration, 134–135
Apotele, 20–21
Apyrase, 8
Arachnids, 23, 25t–26t
Arachnophobia, 7
Arañ̃a del trigo, 527
Araneae. *See* Spiders
Araneidae, 508–509
Araneomorph spiders, 508–511
Arbovirus, 123, 695
Arbovirus reaction, 6
Archostemata, 129
Arctiinae, 448–449
Argasidae
  life cycles, 612–613, 612f
  morphology, 608f, 609
  taxonomy, 603–606
Aristate, 175
Arthropod-borne viruses, 695
Arthropod-transmitted animal viruses, 695
Arthropod-transmitted plant viruses, 695
Asian cockroach, 69
Asian needle ant, 466–467, 480f
Astigmata, 534
Athericidae, 182, 182f

Atopomelidae, 567
Atracotoxin, 517
Atracidae, 508
Australian cockroach, 66, 66f
Australian frog fly, 396, 396f
Australian funnel-web spiders, 517
Australian paralysis tick, 623
Australian redback spider, 527, 527f
Autogenous, 391
Autogeny, 177, 242
Autolysis, 55

B

Babesiosis
  bovine, 650
  canine, 650
  human, 627–628
Baboon spiders, 515–516
Bald-faced hornet, 472f
Ballooning, 513
Bangkok hemorrhagic disease, 230
Barbeiro, 109, 113
Bark lice, 9
Bark scorpion, 500
Barmah Forest virus, 287
Barrel-spider, 505
Bartonellosis, 201–202, 201f
Barychelidae, 508
Bat, 605, 608f, 613, 617–618
  Cimicidae, 119–124
  mites, 539–541, 560, 565–566, 578, 585, 591–592
  myiasis, 385, 411
  Psychodidae, 197
  sarcoptic mange, 578
  surra, 338–339
  triatomines, 108, 112
  trypanosomes, 124
  Venezuelan equine encephalomyelitis, 285
  vesicular stomatitis, 200
Bat fly, 424f
Bat tick, 626
Bazaar fly, 354, 357–358, 357f
Bed bug
  behavior and ecology, 121–122
  common names, 119
  life history, 120–121
  morphology, 119–120, 119f
  prevention and control, 124
  public health importance, 122–123
  taxonomy, 119
  veterinary importance, 123–124
Bees. *See also* Hymenoptera
  behavior and ecology, 464–465
  life history, 463–464
  morphology, 460
  prevention and control, 483–484
  public health importance, 479–482
  social bees, 477–479
  solitary bees, 476
  venoms, 465–466
  veterinary importance, 483
Beetles, 129–141
  behavior and ecology, 130–131
  blister beetles, 129, 133–134, 133f, 134t
  darkling beetles, 129, 135, 136f
  dung beetles, 140–141
  false blister beetles, 129, 134, 135f
  ingestion, 138
  intermediate hosts, 139–140, 139f
  lady beetles, 137–138
  larder beetles, 129, 137, 137f
  life history, 130
  morphology, 129–130, 130f
  nest associates and ectoparasites, 140
  pathogens, 138–139
  prevention and control, 141
  public health importatance, 131–138, 132t
  rove beetles, 129, 134–135, 135f, 136t
  scarab beetles, 129, 137, 137f
  taxonomy, 129
  veterinary importance, 138–141
Behavioral defenses, 8
Bembicinae, 341
Bibionidae, 179, 179f
Bicho colorado, 543–544
Bicudo, 109
Bioinformatics, 683–684
Biological transmission, 6
Birth basket, 498
Birds. *See also* Chicken
  black fly attacks, 251–252, 251f
  feather mites, 568
  fleas, 163–164
  lice, 93–94, 102
  malaria, 312
Bison
  epizootic hemorrhagic disease, 226
  flies, 346t, 355
Biting midges. *See* Ceratopogonidae
Biting rate, 43
Blaberidae, 61
Black death, 156–157
Black flies, 237–256
  behavior and ecology, 240–243
  life history, 240
  morphology, 238–240, 238f–239f
  nuisance problems, 243–245, 245f
  prevention and control, 254–256
  public health importance, 243–250, 244t, 245f–246f, 248t–249t
  taxonomy, 237–238
  veterinary importance, 251–254, 251f
Black fly fever, 243–245
Black imported fire ant, 467f, 468
Blacklegged tick, 620–623, 622f, 628
Black widow spiders
  European black widow spider, 527
  northern black widow spider, 525
  southern black widow spider, 525, 525f
  western black widow spider, 526
Blattaria, 61–76
Blattellidae, 61
Blattidae, 61
Blister beetles, 129, 133–134, 133f, 134t
Blow flies. *See also* Calliphoridae, myiasis
  carrion-associaed flies, 398–401
  myiasis, 398, 399f
  name origins, 392
  nest blow flies, 400–401, 400f
  postmortem succession, 55, 57f–58f
  screwworms, 398–400, 400f
  toad blow flies, 398
  tumbu fly, 401, 401f
Bluetongue disease, 223–225, 224f
Bombycoidea, 439
Bont tick, 624–625, 625f
Boopiidae, 80
Borrelioses, 656
Bot flies. *See* Oestroidea
Bothriuridae, 492
Boutonneuse fever, 635
Bovine onchocerciasis, 252
Bovine spongiform encephalopathy (BSE), 592
Bovine tropical theileriosis, 623
Brachycera, 171–172
Breakbone fever, 291
Bridge vector, 40
Brill-Zinsser disease, 96
Brownbanded cockroach, 68, 68f
Brown cockroach, 66–67, 67f
Brown dog tick, 615f
Brown recluse spider, 519f–521f, 520–522
Brown-tail moth, 447–448
Brown widow spider, 526, 526f
Bruce, David, 3
BSE. *See* Bovine spongiform encephalopathy (BSE)
Buboes, 158
Bubonic plague, 158, 159f
Buck moth, 447, 447f
Buffalo fly, 355, 363–364
Bulge-eye, 223
Bulldozer-spider, 505
Bumble bee, 477, 477f
Bung-eye, 223
Bunyaviridae, 221, 280–282, 281t, 297–300, 298t
Bush chinch. *See* Kissing bugs
Bush fly, 354, 358, 360–361
Buthidae, 490
Butterflies. *See* Lepidoptera

C

Cacodminae, 119
Calabar swellings, 336
Calamistrum, 513
California encephalitis virus, 297, 298t
Calliphoridae, myiasis, 173t–174t, 175f
Calypter, 175, 176f
Calyptratae, 171–172, 175, 176f
Camel
  filariasis, 432
  hippoboscids, 428
  mange, 551
  nagana, 377
  spider bites, 528
  stable fly, 528
  trypanosomiasis, 338–339
Camel nose bot, 409–410, 410f

Camel-spider, 505–506
Camel tick, 228, 617
Campestral plague, 157
Canberra eye, 131
Canine ehrlichiosis, 652
Canine trypanosomiasis, 118
Cantheriasis, 131
Cantharidin, 28–29
Carapace, 495
Carbohydrate feeding, 332–333
Card Agglutination Trypanosomiasis Test (CATT), 379
Carpenter bee, 477, 477f
Carroll, James, 290
Castor bean tick, 623
Cat
  bots, 393
  cytauxzoonosis, 651
  dog fly, 427
  ear mites, 587, 588f
  fleas, 150, 152
  leishmaniasis, 208
  lice, 92, 101–102
  Lyme disease, 656
  mites and mange, 539–540, 544, 551, 560–561, 564, 567, 572–574, 578, 587, 588f
  murine typhus, 160
  notoedric cat mite, 578, 578f
  plague, 157
  Q fever, 638
  spider bites, 528
  tapeworms, 165
  theilerioses, 650
  trypanosomiasis, 117
Caterpillar-induced equine abortion, 454–455
Cat flea, 151, 163
Cat follicle mite, 573f, 574
Cat scratch disease, 161–162
CATT. *See* Card Agglutination Trypanosomiasis Test (CATT)
Cattle
  babesiosis, 649–650
  bluetongue disease, 224f
  bovine onchocerciasis, 252
  chorioptic scab mites, 583–584, 584f
  dermatophilosis, 657f, 658
  ear mites, 587–588
  heartwater, 653
  lice, 89–90, 90f, 94f, 99, 100f
  pinkeye, 360–361, 361f
  psoroptic mange, 582f
  tabanid feeding sites, 334, 334f
  theilerioses, 650–651
Cattle fever tick, 625
Cattle follicle mite, 574
Cattle grub, 406–407, 407f, 411–412, 416–417
Cattle tail louse, 90
CCD. *See* Colony collapse disorder (CCD)
cDNA. *See* Complementary DNA (cDNA)
Centipede, 10
Central-European encephalitis, 628
Ceratophyllidae, 145
Ceratopogonidae
  behavior and ecology, 216–218, 216f–217f
  life history, 215–216
  morphology, 214–215, 214f
  prevention and control, 232
  public health importance, 218–223, 219t–220t
  taxonomy, 213–214
  veterinary importance, 223–232, 224f–227f, 231f
Ceratopogoninae, 213
Cercopidae, 107
Cestoda, 131–132
Chactidae, 491
Chaerilidae, 491
Chagas, Carlos, 3–4
Chagas disease, 116f
  acute disease, 114–115
  chronic disease, 115–117
  control, 118–119
  diagnosis, 115
  kissing-bug vectors, 110t, 113–114
  trypanosome life cycle, 113, 113f
Chagoma, 114–115
Chamois, sarcoptic mange, 577
Chandipura virus disease, 200
Changuinola virus disease, 201
Chaoboridae, 180, 180f, 261
Cheyletidae, 547, 564–565, 566f
Chicken
  air-sac mite, 591f
  poultry leucocytozoonosis, 230, 251–252, 253f
Chicken body louse, 93, 94f, 102
Chicken head louse, 93–94
Chicken mite, 537–538, 538f, 560
Chiggers, 542
Chigoe, 152–153, 152f
Chikungunya virus, 286
Chinchorro, 109
Chinese needle ant, 466–467
Ch'ing yao ch'ung, 135
Chipmunk
  Colorado tick fever, 631
  La Crosse virus, 38
  plague, 157
Chipo, 109
Chirimacha, 109
Chironomidae, 173, 180–181, 181f, 261
Chironomoidea, 261
Chloropidae, 185–186, 185f, 385, 396, 396f
Chorion, 264, 271
Choriothete, 372
Christmas eye, 131
Chrysomyinae, 385, 390
Chrysopinae, 327–328
Chupão, 109
Chyluria, 307–308
Cicada killer, 471–472, 472f
Cicadellidae, 107
Cicadidae, 107
Cimicidae, 119–124
Cimicinae, 119
Clavus, 107
Cleg, 327
Climate change, 48
Clinical case, 47
Coarctate, 177
Coccinellidae, 137–138
Cockle, 100–101, 434, 434f
Cockroach
  allergy, 73–74
  American cockroach, 65–66, 66f
  Asian cockroach, 69
  Australian cockroach, 66, 66f
  behavior and ecology, 64
  brownbanded cockroach, 68, 68f
  brown cockroach, 66–67, 67f
  Florida woods cockroach, 67–68, 67f
  German cockroach, 68–69, 68f
  intermediate hosts, 72–73, 73t
  life history, 63–64, 63f, 63t
  morphology, 61–63
  Oriental cockoach, 65, 65f
  pathogens, 70–72, 71t–72t
  prevention and control, 75–76
  public health importance, 70
  smokybrown cockroach, 67, 67f
  Surinam cockroach, 69, 69f
  taxonomy, 61
  Turkestan cockroach, 65, 65f
  veterinary importance, 74–75
Coffin fly, 183–184, 183f
Coleoptera, 129–141
Colony collapse disorder (CCD), 478
Colorado tick fever (CTF), 630–631
Comparative transcriptomics, 684
Complementary DNA (cDNA), libraries, 684
Conenoses, 109
Congo floor maggot, 401, 401f
Connexivum, 109–110
Copra itch, 545–546
Corethrellidae, 261
Corium, 107
Corolla, 264
Coxae, 19–20, 608
Coyote, sarcoptic mange, 577
Crab louse. *See* Human crab louse
Crambidae, 439–440, 451
Crane flies, 171–172, 178–179, 178f
Cribellum, 513
Crimean tick typhus. *See* Boutonneuse fever
CRISPR-Cas9, 687
Crochet, 440
Cryptic species, 507–508
Ctenidae, 509
Ctenophthalmidae, 145–146
CTF. *See* Colorado tick fever (CTF)
Culicidae. *See also* Mosquitoes
  taxonomy, 261–264, 262t
Culicinae, 261–262
Cutaneous leishmaniasis, 205–207, 206f
Cuterebrinae, myiasis, 389, 403–406
Cyclodevelopmental transmission, 39
Cyclorrhapha, 171–172
Cytauxzoonosis, 651
Cytoditidae, 591–592

D

Darkling beetles, 129, 135, 136f
Dark-winged fungus gnats, 179–180, 179f
Darwin, Charles, 115
Dasyheleinae, 213
DDT. *See* Dichloro-diphenyl-trichloroethane (DDT)
Decomposition
  insect species, 56t
  stages, 54–55
  succession, 55–59
Deer
  ear mites, 587
  epizootic hemorrhagic disease, 225–227, 226f
  lice, 101
  psoroptic mange, 583
  sarcoptic mange, 577
Deer flies. *See* Tabanidae
Deer keds, 425f, 428–430, 428f
Deer nose bot, 394, 409, 409f
Deer skin maggot, 401
DEET. *See* N,N-Diethyl-*meta*-toluamide (DEET)
Definitive host, 35
Delayed hypersensitivity reaction, 5–6
Delusory acariosis, 533
Delusory parasitosis, 7–8
Demodecidae, 547–549, 548f, 572–574
Demodex granuloma, 549
Denaturation, DNA, 677
Dengue
  geographic distribution, 291f
  hemorrhagic fever, 291, 291f
  shock syndrome, 291
  transmission, 291, 292f
De novo sequencing, 690–691
Depluming itch mite, 570–571
Dermanyssidae
  public health importance, 537–539
  veterinary importance, 560
Dermationidae, 568–569
Dermatitis
  flea-allergy, 164
  mites, 537, 544–547, 560
Dermatitis linearis, 135
Dermatophilosis, 657f, 658
Dermestidae, 129, 137, 137f
Desensitization reaction, 5–6
Diapause, 272, 279, 353
Dichloro-diphenyl-trichloroethane (DDT)
  flea control, 167
  mosquito control, 315
Dideoxynucleotides (ddNTPs), 683
N,N-Diethyl-*meta*-toluamide (DEET)
  biting midge control, 232
  black fly control, 255
  fly control, 187
  mosquito control, 315
  sand fly control, 209
  tick control, 660
Dioptic, 176–177
Diplocentridae, 493
Dipluridae, 508
Diptera
  behavior and ecology, 177–178
  families of minor interest, 178–186
  life history, 177
  morphology, 173–177, 175f–176f
  public health importance, 172t, 186
  taxonomy, 171–172, 174t
  veterinary importance, 173t, 186–187
Disease agent, 33–34
Dixidae, 261
DNA polymorphism, 680–683
DNA sequencing, 673–675
Dog
  African horse sickness, 228
  anaplasmosis, 654
  babesiosis, 649–650
  borreliosis, 656
  bots, 393
  ear mites, 587, 588f
  eastern equine encephalomyelitis, 310–311
  fleas, 149, 151–152, 164
  leishmaniasis, 208
  lice, 92, 101–102
  mites amd mange, 544, 549, 551, 560, 573–574, 578, 587
  Q fever, 638
  sarcoptic mange, 576f
  spider bites, 528
  stable fly, 354–355, 362–363
  tapeworms, 162, 165
  ticks, 618, 659
  tórsalo, 404–405
  trypanosomiasis, 114, 118, 338–339
  tumbu fly, 401
  tungiasis, 164
  walkingstick interactions, 9, 9f
Dog flea, 151
Dog fly, 427–428, 432
Dog follicle mite, 573–574, 573f
Dog heartworm, 313–314, 313f–314f
Dog nasal mite, 590, 590f
Dolichoderinae, 459
Drepanidae, 439–440
Drone fly, 184, 184f
Drosophilidae, 185, 185f, 396, 396f
Dumdum fever, 207
Dung beetles, 140–141
Dung flies, 345, 346t
Dysderidae, 509

E

Ear mites, 586–590
Earwig, 10
East African trypanosomiasis, 375
East Coast fever (ECF), 650
Eastern equine encephalitis (EEE), 36, 283–284, 283f, 283t, 310f, 317
Eastern tent caterpillar, 454f
Eastern yellowjacket, 475
ECF. *See* East Coast fever (ECF)
Ectoparasite, 33–34
EEE. *See* Eastern equine encephalitis (EEE)
Eggs
  cockroach, 61, 62f
  fleas, 147, 147f
  lice, 80–81, 83f
  mosquito, 264, 264f, 272f, 279
  muscid flies, 347f, 358
  Tabanidae, 330–331, 331f
EHD. *See* Epizootic hemorrhagic disease (EHD)
Ehrlichiosis
  public health importance, 636–637, 636f
  veteriary importance, 652–653
EIA. *See* Equine infectious anemia (EIA)
Elaeophorosis, tabanid transmission, 340, 340f
Elephant
  African horse sickness, 228
  lachryphagous moths, 450–452
  nose bot flies, 407–410
  stomach bot flies, 410
  trypanosomiasis, 338–339
  tsetse flies, 373
Elephantiasis. *See* Filariasis
Elephant louse, 101
Elephant skin maggot, 401
ELISA. *See* Enzyme-linked immunosorbent assay (ELISA)
Elk
  deer keds, 428–429
  elaeophorosis, 340
  epizootic hemorrahgic disease, 226
  mites, 583
  ticks, 617, 631
Elytra, 129
Embryogenesis, 63
Emerging disease, 47–49
Emerging infectious diseases, 33
Emperor scorpion, 494f
Empodium, 268
Emu
  eastern equine encephalomyeltis, 251
  lice, 102
Endemic pemphigus foliaceus, 250
Endemic typhus. *See* Murine typhus
Endoparasite, 33–34
Endophilic vector, 36–37
Enteric acariasis, 556
Entomological inoculation rate, 42, 45
Entomophobia, 7
Entonyssidae, 588–589
Envenomation, 5, 23
Enzootic transmission, 40
Enzyme-linked immunosorbent assay (ELISA), 678
Epidemic polyarthritis, 287
Epidemic typhus
  diagnosis, 95
  historical perspective, 96
  reservoirs, 96
  transmission, 95
  vector, 95
Epidemiology, vector-borne diseases, 33–49

Epidermoptidae, 568–569
Epigynum, 513
Epimastigote, 113
Episystem, 224
Epizootic hemorrhagic disease (EHD), 225–227, 226f
Equine allergic dermatitis, 231–232, 231f
Equine infectious anemia (EIA), 339, 339f
Equine onchocerciasis, 230–231, 231f
Equne amnionitis and fetal loss, 455
Eremobatidae, 505
Ereynetidae, 590–591
Eruciform, 440
Erucism, 439, 453
Erythema migrans, 640
Erythrocytic anaplasmosis, 655
Eschar, 635
Eurasian hedgehog mange mite, 584–585
European black widow spider, 527
European chicken flea, 153–154
European mouse flea, 154
European rabbit flea, 152
Euscorpiidae, 491
Eusocial species, 464
Everglades virus, 285
Exarate, 147–148
Exflagellation, 301–302
Exophilic vector, 36–37
Exophily, 243
Extension, DNA, 677
Extrinsic incubation, 39
Eye gnats, 185–186, 185f

F

Facultative egg diapause, 279
Facultative myiasis, 383
Facultative parasite, 33–34
False blister beetles, 129, 134, 135f
False stable fly, 355, 359
Fanniidae, myiasis, 385, 396–397, 397f
Fanniinae, 345
Fear, arthropods, 7
Feather mites, 568
Feeding frequency, 43
Femur, 19–20
Feral species, 64
Ferret, sarcoptic mange, 577
Filariasis. *See also* Dog heartworm; Elaeophorosis; Mansonellosis; Onchocerciasis
  clinical disease, 307–308
  elephantiasis, 307f, 308
  geographic distribution, 305–306, 306f
  historical perspective, 309–310
  life cycle, 306–307
  mosquito vectors and epidemiology, 308–309, 309t
Filth flies, 345, 346t
Finlay, Carlos, 3, 290
Fipronil, tick control, 660
Fire ants, 467–469, 467f–468f
Five-o, 213
Flaviviridae, 280–282, 281t, 287–297, 619t–620t, 620–623, 628–629
Flea dirt, 147f, 148
Flea-borne typhus. *See* Murine typhus
Fleas, 145–167
  allergies, 156
  behavior and ecology, 149–150
  cat flea, 151
  chigoe, 152–153, 152f
  dermatitis, 164
  dog flea, 151
  European chicken flea, 153–154
  European mouse flea, 154
  European rabbit flea, 152
  hen flea, 153–154
  human flea, 150
  intermediate hosts, 162–163, 165–166
  life history, 148–149
  morphology, 146–148, 146f–148f
  murine trypanosomiasis, 165
  murine typhus, 159–161
  myxomatosis, 164–165
  Northern rat flea, 153
  Oriental rat flea, 151
  pathogens, 155t, 161
  prevention and control, 166–167
  plague, 156–159
  sticktight flea, 152, 152f
  sylvatic epidemic typhus, 161
  taxonomy, 145–146, 146t
  tungiasis, 152f, 162, 164
  veterinary importance, 163–166
Flesh flies. *See* Sarcophagidae
Float, egg, 264
Florida woods cockroach, 67–68, 67f
Fly blown, 180–181
Fly strike, 187
  Flying squirrelmalaria, 312
  typhus reservoir, 96
Fogo selvagem, 250
Food contamination, arthropods, 6–7
Forcipomyiinae, 213
Forensic entomology, 51–59
  decomposition stages, 54–55
  flies, 171
  historical perspective, 51–52
  homicide and accidental death cases, 53–54
  liability cases, 52
Formicinae, 459
Formicoidea, 459
Formicophilia, 8
Fort Morgan virus, 284
Fossula, 110
Fowl cyst mite, 571, 572f
Fowl tick, 625–626
Fowlpox virus, 311
Fox, sarcoptic mange, 577
Free-tailed bat mite, 540–541
Fruit flies, 185, 185f
Fulgoroidea, 107
Fur mites, 564

G

Gadding behavior, 393, 407f
Gametocyte, 229
Gametogony, 301
Garbage flies, 356, 359
Gaster, 460
Gasterophilinae, myiasis, 389, 410
Gastronyssidae, 410
Genal ctenidium, 146
Gene expression analysis, 684–687
Genital operculum, 495, 495f
Genitalia
  fleas, 147, 147f
  lice, 80–81, 83f
  mosquito, 268–269, 269f
  scorpion, 495
Genome, 678
  projects of important arthropods, 678
  sequencing, 679–684
  size, 679–680
  source material, 680–683
Genomics
  bioinformatics, 683–684
  library construction and genome assembly, 680f, 683
  sequencing of vectors and pathogens, 678
Genu, 20–21
Geometridae, 450–451
Gerbil
  leishmaniasis, 208
  plague, 156–157
  sand fly fever, 201
German cockroach, 68–69, 68f
German yellowjacket, 475
Glossinidae. *See* Tsetse flies
Gnaphosidae, 509
Goat
  ear mites, 586–588
  heartwater, 653–654, 653f
Goat follicle mite, 574
Gonoinactive female, 271
Gonotrophic concordance, 271
Gonotrophic cycle, 34, 271, 278
Gonotrophic discordance, 279
Gorgas, William, 290
Graber's organ, 329
Granulocytic anaplasmosis, 620–623
Grassi, Govanni Batista, 3, 304–305
Ground squirrel. *See* Squirrel
Grub, 391–392
Guanine test, mites, 555
Guinea pig, lice, 111
Gulf Coast tick, 624, 624f
Gypsy moth, 448, 448f
Gyropidae, 80

H

Haematopinidae, 80
Haemosporidian, 229
Hag moth, 446, 446f
Halarachnidae, 587–588, 588f
Halictidae, 459–460, 476
Haller's organ, 22, 608, 609f
Haltere, 176, 268
Hanging groin, 247
Hantaan virus, 540–541
Harpirhynchidae, 585
Harvester ant, 469–470, 470f

Harvester's keratitis, 131
Harvest mite, 543f, 544
Haustellum, 18
HBV. *See* Hepatitis B virus (HBV)
Heartwater, 653–654
Heartworm. *See* Dog heartworm
Heavy dragoon, 119
Hedgehog
  Chandipura virus disease, 191–192
  mites and mange, 568–571, 576
  ticks, 623
Heel fly, 407f
Hemelytra, 107, 110
Hemiptera, 107–124
Hemiscorpiidae, 493
Hen flea, 153–154
Hepatitis B virus (HBV), 117
Heteroscorpionidae, 493
Hibernaculum, 279
Highlands J virus, 284
High-throughput sequencing platforms, 683
Hippoboscidae, 421–424, 423f
Hippoboscoidea, 421–435
  behavior and ecology, 425–426
  deer keds, 428–429, 429f
  dog fly, 427–428
  horse ked, 428, 428f
  life history, 424–425
  morphology, 422–424
  pigeon fly, 429, 430f
  prevention and control, 434–435
  public health importance, 429–430
  sheep ked, 426–427, 426f
  taxonomy, 421–422
  veterinary importance, 430–434, 431t–432t
HIV. *See* Human immunodeficiency virus (HIV)
Hobo's disease, 87–88
Hobo spider, 518–519, 519f
Hog cholera virus, 337t, 340
Hog louse, 84–85
Holometabolous, 177
Holometabolous development, 130
Holoptic, 176–177
Homoptera, 107
Honey bee, 460, 463–464, 478–479, 478f
Hoplopleuridae, 80
Horizontal transmission, 37–40, 38f
Horn fly, 353, 353f, 355
Hornets, 475
Horse bot fly, 411f
Horse
  African horse sickness, 227–228, 227f
  caterpillar-induced abortion, 454–455
  chorioptic mange, 583
  eastern equine encephalitis, 250, 253, 592
  equine allergic dermatitis, 231–232, 231f
  equine infectious anemia, 339, 339f
  equine onchocerciasis, 230–231, 231f
  lice, 91
  mare reproductive loss syndrome, 454
  theilerioses, 650–651
  Venezuelan equine encephalitis, 221–222, 250, 285, 286t
  Western equine encephalitis, 285, 538, 592
Horse flies. *See* Tabanidae
Horse ked, 428, 428f
Horse nose bot, 409
Horse stomach bot, 410, 410f
Host accessibility, 36
Host defenses, 8
Host immunity, 35
Host selection, 43
Host susceptibility, 36
House-dust mite, 553f
  allergy, 552–555
  American house dust mite, 553, 553f
  European house dust mite, 553
House fly, 4, 6–7, 171, 186, 345, 347f
House mouse mite, 539, 539f
Human body louse, 79, 81, 87–88, 87f
Human bot fly, 403–405, 405f
Human crab louse, 88–89
Human flea, 150, 150f
Human follicle mite, 548, 548f
Human head louse, 88
Human immunodeficiency virus (HIV), 94–95, 117
Human notoedric mange, 552
Human scabies, 549–550, 550f
Human scabies mite, 549–550, 550f
Hydrocele, 306–308
Hymenoptera, 459–484. *See also* Ants; Bees; Wasps
  behavior and ecology, 464–465
  life history, 463–464
  morphology, 460–463
  prevention and control, 483–484
  public health importance, 479–482
  sting reactions, 460t, 465
  taxonomy, 459–460, 460t
  venoms, 465–466
  veterinary importance, 483
Hyperendemic, 44
Hypersensitivity reaction, 5–6
Hypnozoite, 302
Hypoderatidae, 585
Hypoendemic, 44
Hypopus, 535
Hypopode, 535
Hypostoma, 214–215
Hypostome, 18–19, 605–607

I

Ibex, epizootic hemorrhagic disease, 226
Idiosoma, 534
Igbo Ora, 287
IgG. *See* Immunoglobulin G (IgG)
IgM. *See* Immunoglobulin M (IgM)
IGRs. *See* Insect growth regulators (IGRs)
Immediate-sensitivity reaction, 5–6
Immunoglobulin G (IgG), 35
Immunoglobulin M (IgM), 35
Immunohistochemistry, 686–687
Immunological techniques, 678
Immunostaining, 686–687
Inbred line, 680–683
Incidental myiasis, 383–384
Indian red scorpion, 500
Indian tick typhus. *See* Boutonneuse fever
Infection prevalence, 42
Infectious bovine keratoconjunctivitis, 361
Initiation phase, 271
Insect growth regulators (IGRs)
  cockroach control, 76
  flea control, 166
  hymenopteran control, 484
  tick control, 662
Insect population suppression, 687–689
In situ hybridization, 686
Installment hatching, 273
Intermediate host, 9
International Catalogue of Arboviruses, 701
Interseasonal maintenance, parasites, 41–42
Intrinsic incubation, 39
Io moth, 442f
Ira, 558
Ischnocera, 79
Ischnopsyllidae, 145–146
Isoamyl acetate, 464, 483
Iuridae, 492
Ivermectin, 222, 231
Ixodida. *See* Ticks
Ixodidae
  life cycles, 611–612, 611f
  morphology, 606–611
  taxonomy, 603–606

J

Jackal
  canine filariasis, 155t
  control, 210
Jamestown Canyon virus, 299
Japanese encephalitis virus complex
  geographic distribution, 293f
  Japanese encephalitis virus, 294–295
  Murray Valley encephalitis virus, 296
  St. Louis encephalitis virus, 295
  West Nile virus, 293–294
Jejenes, 261
Jerrymander, 505
Jerrymunglum, 505
Jigger, 152–153
Johnston's organ, 21, 215
Junin virus, 542
Justinian's plague, 156–157

K

Kangaroo
  bot flies, 407–408, 409f
  chiggers, 535, 564
  filarial nematodes, 314
  myiasis, 340
  Q fever, 638
  tabanid disease transmission, 338
  tear-drinking moths, 450, 451f
Kangaroo rat, Lyme disease, 297
Kangaroo throat bot, 409f

Karakurt, 524
Katipo spider, 527
Keds. *See* Hippoboscoidea
Keel, 62
Kenya tick typhus. *See* Boutonneuse fever
Keystone virus, 299
Kilbourne, F. L., 2–3
Kissing bugs
  behavior and ecology, 111–112
  Chagas disease vectors, 110t, 113–114
  life history, 111
  morphology, 109–111
  overview, 109
  prevention and control, 118–119
  public health importance, 112–117
  taxonomy, 109
  veterinary importance, 118
Knemidokoptidae, 569
Koch's postulates, 673
K-strategist, 178
Kuiki, 213

L

Lachryphagous moths, 444, 450–452
La Crosse virus, 38, 38f
Lady beetles, 137–138
Laelapidae, 541–542
Lagomorph. *See* Rabbit
La malmignatte, 527
Laminosioptidae, 571
Lamponidae, 509
Lancet fluke, 483
Larder beetles, 137, 137f
Larval diapause, 279
Larviparous, 177
Lasiocampidae, 449
Latrodectism, 523
Laveran, Charles, 304–305
Lazear, Jesse, 290
Leishmaniasis
  clinical features, 202f
  cutaneous leishmaniasis, 205–207
  geographic distribution, 202f
  transmission, 205, 207
  veterinary importance, 207–208
  visceral leishmaniasis, 207
Lepidopterism, 439
Lepidoptera, 439–455
  behavior and ecology, 443–450
  hairs, 441–443
  lachryphagous moths, 450–452
  life history, 443
  morphology, 440–443
  prevention and control, 455
  public health importance, 452–454
  taxonomy, 439–440
  urticating caterpillars, 444–450
  veterinary importance, 454–455
  wound-feeding and skin-piercing moths, 452
Leptoconopinae, 213
Leptopsyllidae, 145–146
Leucocytozoonosis
  black fly transmission, 252–253
  poultry, 253
Lice
  behavior and ecology, 84–86
  bird lice, 93–94, 102
  cat lice, 92, 101–102
  cattle lice, 89–90, 90f
  deer lice, 92, 101
  dog lice, 92, 101–102
  hog lice, 91–92, 100
  horse lice, 91, 100, 104
  human body louse, 87–88, 87f
  human crab louse, 88–89
  human head louse, 88
  laboratory animals, 92–93, 102
  life history, 83–84
  livestock losses, 98–101
  morphology, 80–83
  pathogens, 98, 99t
  pig lice, 98, 100, 102
  prevention and control, 102–104
  public health importance, 94–98
  sheep and goat lice, 92, 100
  taxonomy, 79–80, 81t–82t
  veterinary importance, 98–102
Limacodidae, 439, 442f, 445–446
Lindane, tick control, 660
Liochelidae, 492
Listrophoridae, 567
Little blue cattle louse, 89–90, 90f
Little fire ant, 469
Little house fly, 355–356, 355f
Lizards
  mites, 562, 592–593
  scorpions, 497
  spider bites, 515
  ticks, 39, 616, 620–623, 642
  triatomines, 111
  tsetse flies, 373–374
Loiasis
  clinical features, 336–337
  control, 336–337
  tabanid transmission, 336
Lone star tick, 605, 611f, 623–624
Longnosed cattle louse, 89, 90f
Loop-mediated isothermal amplification (LAMP), 677
Louping ill, 623, 628, 651–652
Louse-borne relapsing fever, 96–97
Louse flies. *See* Hippoboscoidea
Love-bug, 179, 179f
Loxoscelism, 519, 522
Lunule, 175
Lycosidae, 509–510
Lymantriinae, 443
Lyme disease
  public health importance, 626–627
  tabanid transmission, 338
  veterinary importance, 620–623
Lymphatic filariasis, 305

M

Mackie, F. P, 3–4
Macronyssidae, 560–562
Macrophage, 35
Mad cow disease. *See* Bovine spongiform encephalopathy
Maggot
  clinical use, 412
  myiasis, 391–392
Makunagi, 213
Malaria
  avian, 312
  clinical disease, 302–303
  control, 314–315
  geographic distribution, 306f
  historical perspective, 304–305
  life cycle of parasite, 301–302
  monkeys, 314
  mosquito vectors and epidemiology, 303–304
  reptiles, 312
  rodent, 312
Malignant tertian malaria, 300, 302
Mammalophagic vector, 36–37
Mammalophily, 242
Mange
  cheyletid mange, 564
  human notoedric mange, 552
  mites, 568–571
  psoroptic mange, 581, 583
  sarcoptic mange, 575
Mango fly, 327
Manson, Patrick, 2, 304–305
Mansonellosis
  black fly transmission, 222, 237
  human, 222–223
March fly, 327
Mare reproductive loss syndrome, 454
Marmoset, wasting disease, 287
Marmots, Powassan encephalitis, 630
Marseilles fever. *See* Boutonneuse fever
Maruin, 213
Mastoparans, 466
Mata venado, 505
Mating plug, 276
Matrone, 276
May fly, 327
Mayaro virus, 287
Mechanical transmission, 6, 38–39
Median infectious dose, 42
Medical entomology, 1
Medical-veterinary acarology, 1
Medical-veterinary arachnology, 1
Medical-veterinary entomology
  definition, 1
  historical perspective, 2–4
  literature, 2
Medicocriminal entomology, 51
Megalopygidae, 444–445, 445f
Melicharidae, 537
Melittin, 28–29, 466
Meloidae, 129, 133–134, 133f, 134t
Membracidae, 107
Menoponidae, 80
Merogony, 301
Meront, 628
Merozoite, 302
Merutu, 213

Mesembrinellinae, myiasis, 385
Mesoendemic, 44
Mesosoma, 494–495
Mesostigmata, 494–495
Mesothelae, 507
Metacyclic forms, 113
Metasoma, 494–495, 495f
Metastriata, 603–604
Methoprene, 166
Mexican chicken bug, 123
Mexican typhus. *See* Murine typhus
Microfilaremia, 306
Microfilariae, 306
Micropyle, 238
Microsatellite analysis, 675
Midges. *See* Ceratopogonidae; Chaoboridae; Chironomidae
Millipede, 6
Miltogramminae, myiasis, 385–389
Minors, ants, 460–461
Mites, 533–596
  allergies, 552–555
  behavior and ecology, 536
  delusory acariosis, 533
  dermatitis, 537, 560
  intermediate hosts, 559
  internal acariasis, 555–556
  life history, 535, 537f
  morphology, 534–535, 535f
  rickettsialpox, 556–557
  skin-invading mites, 547–549
  stored-products mites, 544–547
  tapeworm intermediate hosts, 593–596, 594t
  taxonomy, 533–534, 533t
  Tsutsugamushi disease, 544t, 557–559, 558f
  veterinary importance, 559–596
Miturgidae, 510
Mahogony-flat. *See* Bed bug
Mold mite, 545
Mongoose, dog fly, 427
Monkey lung mite, 589–590, 590f
Monkeys
  beetle infestation, 52
  blood protozoans, 213
  bot flies, 406–407
  cat fleas, 163
  Chikungunya virus, 286
  Dengue, 291–292
  filarial nematodes, 306
  malaria, 313
  mites and mange, 568–571
  Oropouche fever, 221
  sarcoptic mange, 575
  spider bites, 527
  tórsalo, 404–405
  triatomines, 109
  vesicular stomatitis, 186
  wasting disease, 75
  yellow fever, 288–290
Monkey slug, 446
Moose fly, 345
Morbidity, 43
Morgellons disease, 8
Mortality, 43
Morulae, 636
Mosquitoes, 261–317
  behavior and ecology, 272–279, 273f
  bites, 280
  filariasis, 305–310
  life history, 269–272
  malaria, 300–305
  morphology, 264–269
  prevention and control, 314–317
  taxonomy, 261–264, 262t
  viruses, 280–287
Moth fly. *See* Psychodinae
Moths. *See* Lepidoptera
Mouse
  bot, 404f
  lice, 89
  trypanosomiasis, 113–114
Mouse fur mite, 566
Mouthparts
  biting midges, 17–18
  black fly, 243
  cockroaches, 61
  Diptera, 175
  fleas, 146–147
  lepidopterans, 440f
  lice, 82
  mosquitoes, 267, 268f
  muscid flies, 345, 348, 349f
  parasitic arthropods, 17–19
  spiders, 511–512
  Tabanidae, 330
  true bugs, 107
  tsetse fly, 370
Mud dauber, 471–472
Multiple gonotrophic cycles, 612
Multiplex polymerase chain reaction, 675
Multiplicative transmission, 39
Mummification, 54
Murine trypanosomiasis, 165
Murine typhus, 159–161
Murray Valley encephalitis virus, 296
Muscidae, 345–366
  bazaar fly, 354, 357–358
  behavior and ecology, 351–353
  buffalo fly, 355
  bush fly, 354, 358
  cluster fly, 354, 358
  face fly, 354, 358, 361–362
  false stable fly, 355, 359
  garbage flies, 356, 359
  horn fly, 355, 363–364
  house fly, 353–354, 357, 360
  life history, 350–351
  little house fly, 359
  morphology, 346–350
  myiasis, 396–397
  prevention and control, 364–366
  public health importance, 356–359
  stable fly, 354–355, 358, 362–363
  sweat flies, 356, 359, 364
  taxonomy, 345, 346t
  veterinary importance, 359–364
Muscinae, 345
Muscoidea, 383–417
  ecology and behavior, 392–394
  life history, 390–392
  morphology, 389–390
  myiasis types, 383–384
  myiasis myths, 394
  prevention and control, 414–417
  public health importance, 411–412
  taxonomy, 384–389, 386t–389t
  veterinary importance, 413–414
Mutillidae, 471, 471f
Mygalomorphae, 507
Myiasis, 383–417
  ecology and behavior of flies, 392–394
  fly families, 394–411
  forms, 383–384
  life history of flies, 390–392
  morphology of flies, 389–390
  myths, 394
  prevention and control, 414–417
  public health importance, 411–412
  taxonomy of flies, 384–389
  veterinary importance, 413–414
Myobiidae, 565–566
Myocoptidae, 567–568
Mystacinobiidae, 411
Myxoma virus, 164–165, 312
Myxomatosis, 164–165, 253
Myxophaga, 129

N

Nason's slug moth, 446, 447f
Nematocera, 171–172, 174t
Nematoda, 131–132
Nematodes, biting midge transmission, 171
Neottiphilidae, 385
Nest blow flies, 385, 400–401
Nest skipper fly, 385
Nested polymerase chain reaction, 675–677
Next-generation sequencing (NGS) techniques, 690–691
Nicolle, Charles, 3–4
No-no, 213
Notodontidae, 439–440, 450–451
Noctuidae, 439–440, 444, 449
Noctuoidea, 439
Nolidae, 439–440, 444, 449, 451
Nonviremic transmission, 39, 626–627
North Coast funnel-web spider, 517
Northern black widow spider, 525
Northern fowl mite, 540, 541f
Northern rat flea, 153, 561–562
Nose bot flies, 407–410
Notoedric cat mite, 578–579
Notoedric rat mite, 579–580
Notoedric squirrel mite, 579
Nott, Josiah, 3
Nukaka, 213
Nulliparity, 34
Nurse worker, ant, 460–461
Nuttalliellidae, 603–604, 606
Nycteribiidae, 424

Nymphalidae, 450
Nyung noi, 213

O

Obligate egg diapause, 279
Obligate parasite, 383
Obligatory myiasis, 383–384
Obtect, 177
Occupational hazards, arthropods, 52
Ockelbo, 287
OCP. *See* Onchocerciasis Control Programme
Ocular points, 83
Oedemeridae, 129, 134, 135f
Oestridae, myiasis, 402–403
Oestrinae, myiasis, 407–410
Oestroidea, 383–417
  Cuterebrinae, 403–406
  ecology and behavior, 351–353
  Gasterophilinae, 410
  Hypodermatinae, 406–407
  life history, 350–351
  morphology, 346–350
  prevention and control, 364–366
  public health importance, 356–359
  taxonomy, 345, 346t
  veterinary importance, 359–364
Onchocerciasis
  bovine onchocerciasis, 252
  control, 255–256
  equine onchocerciasis, 230–231, 231f
  river blindness, 245–247
Onchocerciasis Control Programme, 255
O'nyong-nyong virus, 287
Oocyst, 229
Ookinete, 229
Oothecae, 62
Opisthosoma, 494–495, 534
Opisthothelae, 507
Oribatida, 534
Organ of Berlese, 120–121
Organ of Ribaga, 120–121
Oriental cockoach, 65, 65f
Oriental rat flea, 151
Ornithophagic vector, 36–37
Ornithophily, 242
Oropouche fever, 221
Oviparity, 63, 177
Oviposition, 86, 111, 241, 243, 267, 278, 391, 614
Ovoviviparity, 391
Ovoviviparous, 177
Oxyopidae, 510

P

Palatal brushes, 264–265
Pandemic, 44
Pangoniinae, 327–329
Paper wasp, 473f, 476
Papilionoidea, 439
Papulonodular demodicosis, 573
Paragenital sinus, 120
Parakeet
  lice, 102
  mites, 537–538, 569–570, 589
Parasimuliinae, 237
Parasitoid, 365
Parous female, 34
Pasteur, Louis, 304–305
Pathogen, 33–34
Pathogen evolution, 48–49
Pavement ant, 470, 470f
PCR. *See* Polymerase chain reaction (PCR)
Pectinal teeth, 495
Pederin, 134–135
Pedicinidae, 80
Pediculidae, 80
Pediculicide, 102
Peribunyaviridae, 219t–220t
Peridomestic species, 48, 111
Peritrophic membrane, 268–269
Permethrin
  sand-fly control, 209
  tick control, 659
Petiole, 460
Phantom midges, 180, 180f
Pharaoh's ant, 470–471, 470f
Pheromones, 63–64, 661
Philopteridae, 80
Phlebotominae
  behavior and ecology, 196–198
  life history, 195–196
  morphology, 194–195
  prevention and control, 209–210
  public health importance, 198, 199t
  sand fly, 191f
  taxonomy, 193
Phoridae, 183–184, 183f
Phospholipase, 465
Photoperiod entrainment, ticks, 615
Phthiraptera, 79–104
Phylogenomics, 678
Pig
  African swine fever, 652
  beetles, 140
  Chandipura virus, 200
  fleas, 164
  Japanese encephalitis, 294–295
  lancet fluke, 483
  lice, 90–92
  louping ill, 651–652
  mites and mange, 551, 557, 572, 576, 577f
  muscid flies, 359–360
  myiasis, 397, 401
  sand flies, 200
  sarcoptic mange, 577f
  simuliotoxicosis, 253
  skin-piercing moths, 452
  Tahyna virus, 299
  tick-borne relapsing fever, 645
  vesicular stomatitis, 200
Pigeon
  beetle infestation, 140
  chicken mite, 537–538
  chiggers, 559
  fowl cyst mite, 543f, 571
  hypoderatid mites, 585
  louse flies, 433
  northern fowl mite, 561
  ticks, 605
  tropical fowl mite, 540
  trypanosomiasis, 113
Pigeon fly, 429, 430f
Pigeon mite, 538
Pigeonpox virus, 311
Pinkeye, 38–39, 186, 361f
Piophilidae, 55–57, 184f, 395–396
Piperidine, 465
Piroplasmoses, tick transmission, 649–652
Piroplasms, 627–628
Pisauridae, 510
Pito, 109
Plague
  ecological forms, 156–157
  flea vectors, 158
  outbreak control, 167
  pandemic history, 156–157
  pathogen, 156
Pneumocoptidae, 592
Pneumonic plague, 159
Podosoma, 534
Poison, 5
Polistinae, 460
Polleniinae, myiasis, 385
Polyctenidae, 119
Polymerase chain reaction (PCR), 673–675, 674f
  advantages of, 673–675, 676t
  amplified fragment length polymorphism, 675
  disadvantages of, 673–675, 676t
  multiplex polymerase chain reaction, 675
  nested polymerase chain reaction, 675–677
  real-time polymerase chain reaction, 677
  restriction fragment length polymorphism (RFLP), 675
Polyphaga, 129
Polyplacidae, 80
Pompilidae, 471
Population genomics, 678
Porcupine, malaria, 314, 579
Posterior-station transmission, 118
Postgenal cleft, 239
Postinfestation interval, 52, 57–58
Postmortem interval, 52, 55–59
Post-trophic phase, 271
Poultry. *See* Chicken; Turkey
Prepupa, 177
Pretarsus, 495–496
Previtellogenic phase, 271
Primary myiasis, 383
Primates, nonhuman. *See* Monkeys
Primers, 673–675
Pronotal ctenidium, 146
Propagative transmission, 39
Propodeum, 460
Prosimuliini, 237
Prosoma, 494–495
Prostigmata, 534
Prostriata, 604
Pseudochactidae, 491

Psocoptera, 79
Psorergatidae, 574–575
Psoroptic mange, 581
Psoroptidae, 581
Psychodidae, 191–210, 394
  behavior and ecology, 196–198
  life history, 195–196
  morphology, 193–195
  moth fly. *See* Psychodinae
  prevention and control, 208–210
  public health importance, 198–207
  sand fly. *See* Phlebotominae
  Syrcoracinae, 192–193
  taxonomy, 192–193
  veterinary importance, 207–208
Psychodinae
  behavior and ecology, 196
  life history, 195
  morphology, 193–194
  moth fly, 191f
  prevention and control, 208–209
  public health importance, 198
  taxonomy, 192
Pthiridae, 89
Ptilinum, 177
Pulicidae, 145
Pulmonary acariasis, 555–556
Pulvilli, 62–63
Punkie, 213
Pupariation, 347
Puparium, 177, 347
Purpura pulicosa, 154
Puss caterpillar, 445f
Pyemotidae, 546–547
Pygidium, 146
Pygiopsyllidae, 145–146
Pyrethrins, 166
Pyriproxyfen, 166

Q

Q fever, tick transmission, 638, 657
qRT-PCR, 685–686
Quantitative polymerase chain reaction, 677
Queensland itch, 231
Queensland tick typhus, 636
Quill mites, 568

R

Rabbit
  California encephalitis, 297
  Colorado tick fever, 630–631
  demodectic mange, 572–573
  filarial nematodes, 314
  fleas, 148
  Keystone virus, 299
  lice, 93, 98, 102
  mites, 544, 560, 564–567, 572–573
  muscid flies, 358
  myiasis, 391–392, 394, 403–404
  myxoma virus, 164, 253, 312
  sarcoptic mange, 577
  Snowshoe hare virus, 253
  ticks, 604–605, 616, 647
  trypanosomiasis, 165
  tularemia, 102, 337, 646
  vesicular stomatitis, 200
  Western equine encephalomyelitis, 284–285
Rabbit ear mite, 586, 586f
Rabbit fever. *See* Tularemia
Rabbit flea, 146f
Rabbit fur mite, 565, 565f
Rabbit tick, 618
Raccoon
  fleas, 151, 156, 166
  trypanosomiasis, 117
  vesicular stomatitis, 208
Racquet organ, 505–506
Radioallergosorbent test (RAST), 555
RAPD. *See* Rapid amplification of polymorphic DNA (RAPD)
RAPD-PCR, 675
Rapid amplification of polymorphic DNA (RAPD), 675
RAST. *See* Radioallergosorbent test (RAST)
Rat
  lice, 92–93, 102
  murine typhus, 159–160
  notoedric rat mite, 579–580
Rat typhus. *See* Murine typhus
Rat-tailed maggot, 395, 395f
Real-time polymerase chain reaction, 677
Receptor-mediated endocytosis, 613
Recombinant DNA, 417
Recrudescent typhus, 96
Red bug, 543–544
Red coat, 119
Red imported fire ant, 23f, 27f
Reduviidae, 109–119
Red widow spider, 526–527, 526f
Reed, Walter, 3, 290
Relapsing fever
  louse-borne, 96–97
  tick-borne, 643–646
Reoviridae, 201, 223–224, 630–631
Reproductive diapause, 279
Reptiles. *See also* Lizards; Snakes
  biting midges, 217–218, 229
  chiggers, 542
  flesh flies, 402
  malaria, 312
  mites, 533–534, 539, 541, 543–544
  mosquitoes, 277, 284
  Q fever, 638
  tabanids, 333
  ticks, 604, 609f, 616
  triatomines, 111
  tsetse flies, 373
Reservoir host, 35
Resilin, 19–20, 149–150
Respiratory mites, 588
Respiratory siphon, 265–266
Respiratory spiracles, 347, 348f
Restriction enzymes (REs), 675
Reverse genetics, 687
Reverse transcription, 684
Reverse transciptase-polymerase chain reaction, 673–675
Rhabdoviridae, 198, 223
Rhagionidae, 182, 182f
Rhiniinae, myiasis, 385
Rhinoceros
  myiasis, 410
  skin-piercing moths, 452
  tear-drinking moths, 450
  tsetse flies, 373
Rhinonyssinae, 589
Rhinophoridae, myiasis, 411
Rhopalopsyllidae, 145–146
Rhynchophthirina, 79
Rhyncoptidae, 580–581
Rib cockle, 100–101, 434, 434f
Ricketts, Howard Taylor, 3–4
Rickettsialpox, 556–557
Rift Valley fever, 200
Rift Valley fever virus, 253, 299
River blindness, 245–250, 246f–247f
RMSF. *See* Rocky Mountain spotted fever (RMSF)
RNA analysis, 684–686
  in situ hybridization, 686
  qRT-PCR, 685–686
  transcriptomics, 684, 685f
RNAi, 689
RNA interference, 662
Rocio virus, 297
Rocky Mountain spotted fever (RMSF), 632–635, 634f
Rodents. *See* Mouse; Rat
Roman (solpugid), 505
Romaña's sign, 114–115, 115f
Root maggot, 385
Roseola pulicosa, 154
Ross, Ronald, 3
Ross River virus, 287
Rostrum, 120
Rove beetles, 24, 130, 134–135
RSSE. *See* Russian spring-summer encephalitis (RSSE)
r-strategist, 178
Rural plague, 157
Russian spring-summer encephalitis (RSSE), 628
Ruxton, Buck, 52

S

Sac spider, 518f
Saddleback caterpillar, 445f
St. Louis encephalitis virus, 293f, 295
Salmonellosis, 161
Salticidae, 510
Sand flea, 152–153
Sand fly fever, 200–201
Sand fly. *See* Phlebotominae
Sand-puppy, 505
Sandwich ELISA, 678
Sanger sequencing, 683

Sarcophagidae, myiasis, 384, 401–402, 402f
Sarcoptidae, 549–550, 575–578
Saturniidae, 446–447, 447f
Scabies
  human, 550–552
  sheep, 582
Scabies mite, 575–578
Scaly-face mite, 570
Scaly-leg mite, 569–570
Scarab beetles, 137, 137f, 140
Scarabaeidae, 129, 137, 137f
Scarabiasis, 131
Scatter cockle, 100–101
Schizogony, 229, 301
Schizophora, 175
Sciaridae, 179–180, 179f
Scoleciasis, 439
Scorpionidae, 493–494
Scorpions
  behavior and ecology, 497–498
  life history, 496–497
  medical importance, 500–501
  morphology, 494–496
  prevention and control, 501–502
  public health importance, 498–500
  taxonomy, 489–494, 494f
  toxicity ranking of species, 490t
  veterinary importance, 501
α-Scorpion toxin, 499
β-Scorpion toxin, 499
Scorpion venoms, 29
Screwworm, 385, 391–392, 398
Scrub typhus, 537, 557
Scuttle flies, 183–184, 183f
Secondary myiasis, 383
Segestriidae, 510
Sensilium, 22, 146
Sentinel, 46–47
Septicemic plague, 158–159
Seroconversion, 44–45
Seropositive, 44–45
Shaft louse, 93–94, 102
Sheep
  African horsesickness, 228
  anaplasmosis, 654
  blow flies, 413
  bluetongue disease, 223–224, 224f
  borreliosis, 656
  bot, 409, 409f
  cat fleas, 163
  Chandipura virus, 200
  chiggers, 563–564
  chorioptic mange, 583–584
  cockle, 433
  demodectic mange, 572–573
  elaeophorosis, 340
  epizootic hemorrhagic disease, 226
  harvest mite, 544
  heartwater, 653
  lice, 90–92
  louping ill, 651
  muscid flies, 355
  myiasis, 384, 394, 397, 403–404, 409–410
  nagana, 377
  nematodes, 140
  psoroptic mites, 583–584
  Q fever, 620
  Rift valley fever, 311
  sandfly fever, 201
  scabies, 582
  simuliotoxicosis, 253
  spider bites, 528
  tapeworms, 593–596
  theileriosis, 651
  ticks, 604, 620, 623
  tórsalo, 404
  trypanosomiasis, 339
  tularemia, 646
  vesicular stomatitis, 208
  Wesselsbron virus, 311
Sheep biting louse, 86, 92, 434
Sheep bot fly, 409f
Sheep face louse, 85
Sheep head fly, 356, 364
Sheep itch mite, 575
Sheep ked, 17, 100–101, 423f, 425–427
Sheep nose bot, 187, 409
Sheep scab mite, 582
Sheep strike, 187
Sheep tick, 426
Ship typhus. *See* Murine typhus
Shortnosed cattle louse, 90, 90f
Sibling species, 238
Sicariidae, 510
Silk allergy, 453
Simond, Paul Louis, 3
Simuliidae. *See* Black flies
Simuliinae, 237
Simuliini, 237
Simuliotoxicosis, 253–254
Sindbis virus, 287
Single-base differences, 678
Single nucleotide polymorphisms (SNPs), 675
Siphonaptera, 145–167
Skipper flies, 55–57, 385
Slender guinea-pig louse, 84–85, 102
Smith, Theobald, 2–3
Smokybrown cockroach, 67, 67f
Snake mite, 541, 542f, 562
Snakes
  chiggers, 544
  mites, 541, 562
Snipe flies, 182, 182f
Snowshoe hare virus, 253
Soldier, ants, 460–461
Soldier fly, 171–172, 183, 394–395
Solenopsin, 27f, 28
Solifugae, 505–506, 505f
Solpugids, 505–506, 505f
Soremuzzle, 224
South American violin spider, 523
Southern black widow spider, 525
Southern cattle fever tick, 625
Southern yellowjacket, 475, 481f
Spanish jack, 476
Spermalege, 120–121
Sphecidae, 460, 471–472
Sphingidae, 451
Spicule hairs, lepidopterans, 441–443
Spider wasp, 471f
Spiders, 507–529
  araneomorph spiders, 508–511
  behavior and ecology, 513–514
  black widow spiders, 510–511, 524
  brown recluse spider, 519–523, 519f
  cheiracanthism, 518
  funnelweb spiders, 517
  latrodectism, 523
  life history, 513
  morphology, 511–513, 511f
  mygalomorph spiders, 507–508
  phoneutriism, 507
  prevention and control, 528–529
  public health importance, 514–527
  South American wandering spider, 518
  tarantulas, 515–517, 515f
  taxonomy, 507
  veterinary importance, 527–528
Spine hairs, lepidopterans, 443
Spinose ear tick, 626
Spiny oak-slug caterpillar, 446f
Spiny rat mite, 542, 562–563
Sporadic epidemic typhus, 96
Sporogony, 301
Sporozoite, 301–302
Springtail, 9–10
Squamous demodicosis, 573
Squirrel. *See also* Flying squirrels
  blood protozoans, 229
  borreliosis, 642
  bot, 405f
  California encephalitis, 297
  Colorado tick fever, 630–631
  fleas, 165
  Keystone virus, 299
  La Crosse encephalitis, 38, 298
  leishmaniasis, 207
  lice, 85
  malaria, 312
  mites and mange, 544, 568, 579, 592
  myiasis, 394
  notoedric squirrel mite, 579
  plague, 157
  Powassan encephalitis, 630
  ticks, 615–616, 630–631
  tularemia, 646
  Western equine encephalitis, 284–285
Squirrel flea, 156
Stable fly, 352f, 358, 362–363, 362f
Staphylinidae, 129, 132–135, 136t
Staphylococcal infection, 161
Stenogastrinae, 460
Stercorarian transmission, 38
Sterile-insect technique (SIT), 687
Sticktight flea, 152, 152f
Stilting, 498
Stinging arthropods. *See* Hymenoptera; Scorpions
Stinging rose caterpillar, 446f
Stomach bot flies. *See* Gasterophilinae
Storage mites, 552

Stratiomyidae, 182–183, 183f
  myiasis, 394–395, 395f
Stratiomyomorpha, 171–172
Straw itch mite, 547f
Streblidae, 424
Strike, 392
Stylostome, 558
Sun-spider, 505
Superstitioniidae, 491
Surinam cockroach, 69, 69f
Surra, 338–339
Surveillance, 43–47
Swallow bugs, 119
Swarming, bees, 463
Sweat bee, 476, 476f
Sweat flies, 345, 351, 356, 359, 364
Sweet itch, 231
Swinepox, 100
Swinepox virus, 99t
Sycoracinae, 192–193
Sydney funnelweb spider, 517, 517f
Sylvatic epidemic typhus, 161
Sylvatic plague, 45–46
Symphyta, 459
Synanthropic flies, 345
Synganglion, 610–611
Syrphidae, 184, 184f
  myiasis, 395, 395f

T

Tabanidae, 327–341
  behavior and ecology, 332–336
  horse flies versus deer flies, 327
  life history, 330–332, 331f
  morphology, 329–330, 329f
  prevention and control, 340–341
  public health importance, 336–338, 337t
  taxonomy, 327–329, 328t, 330f, 330t
  traps, 341
  veterinary importance, 338–340
Tabaninae, 328
Tabanomorpha, 171–173
Tache noir, 635f
Tachinidae, myiasis, 411
Tahyna virus, 299
Tangential transmisson, 40
Tapeworm
  beetles as intemediate hosts, 139–140, 139f
  fleas as intermediate hosts, 162, 165
  lice as intermediate hosts, 98
  mites as intermediate hosts, 559, 593–596
Tarantism, 514–515
Tarantula hawk, 28, 29f
Tarantulas, 515f
  hairs, 516, 516f
  tarantism, 514
  toxicity, 515–517
Targeted-sequencing platforms, 690–691
Tarsomere, 19–20
Tarsus, 19–20
TBE. *See* Tick-borne encephalitis complex (TBE)
TBRF. *See* Tick-borne relapsing fever (TBRF)
Tegmen, 62–63
Telmophage, 17, 217
Tenebrionidae, 129, 135, 136f
Terebrantia, 459
Terminalia, 63, 268
Thaumaleidae, 261
Thaumetopoeidae, 449–450
Theileriosis, tick transmission, 651
Theraphosidae, 507–508
Theridiidae, 510–511
Thomisidae, 511
Thrips, 10
Tick-borne encephalitis complex (TBE), 628–630
Tick-borne relapsing fever (TBRF), 643–646
Tick paralysis, 647–648, 658–659
Ticks, 603–663
  African swine fever, 652
  allergy, 648
  anaplasmosis, 603, 637, 654–655
  babesiosis, 627–628
  behavior and ecology, 613–618
  borrelioses, 656
  Boutonneuse fever, 635
  Colorado tick fever, 620
  dermatophilosis, 657–658, 657f
  ehrlichiosis, 637–638
  hard ticks, 604–605
  heartwater, 653–654
  life history, 611–613
  louping ill, 651–652
  Lyme disease, 623, 638–643, 641f
  morphology, 606–611
  piroplasmoses, 649–652
  prevention and control, 659–661
  Q fever, 638
  Rocky Mountain spotted fever, 632–635, 633f
  soft ticks, 605–606
  taxonomy, 603–606, 603t
  theilerioses, 650–651
  tick paralysis, 647–648, 658–659
  tick toxicosis, 659
  tick-borne encephalitis complex, 628–630
  tick-borne relapsing fever, 643–646
  tularemia, 646–647, 656–657
  veterinary importance, 621t, 648–659
Tick toxicosis, 659
Tipulidae, 178–179, 178f
Toad blow flies, 398
Togaviridae, 280–287, 283f
Tórsalo, 404–405
Townsend, C. H. T, 202
Toxicosis, 5
Toxins, 23, 27f
  arthropods, 5
  effects, 24–28
Tracheal skein, 272
Transcriptome, 684
Transfusion, babesiosis risks, 522
Transgenerational transmission, 37–38
Transmission rate, 37
Transstadial transmission, 37
Trematode, 139
Trench fever, 97–98
Triatominae. *See* Kissing bugs
Trichobothria, 22
Trichodectidae, 80
Trivittatus virus, 299
Troglotayosicidae, 491
Trombiculidae, 542–544, 563–564, 591
Trophic phase, 271
Tropical bont tick, 605
Tropical fowl mite, 540, 540f, 561
Tropical rat mite, 539–540, 560–561
Tropical theileriosis, 651
True bugs, 107–124
True flies. *See* Diptera
Trypanosomiasis. *See* African sleeping sickness; Chagas disease; East African trypanosomiasis; Murine trypanosomiasis; Nagana; Surra; West African trypanosomiasis
Tsetse flies, 369–381
  African sleeping sickness, 374–377, 375f
  behavior and ecology, 372–374, 374f
  East African trypanosomiasis, 376
  geographic distribution, 374–375, 375f
  life history, 371–372, 372f
  morphology, 370–371, 371f
  nagana, 377–379, 378f
  pathogens and vectors, 377
  prevention and control, 379–381
  taxonomy, 369–370
  trypanosome life cycle, 376–377
  West African trypanosomiasis, 375–376
Tsutsugamushi disease, 537, 557–559
Tularemia
  clinical features, 337–338, 338f
  flea transmssion, 161
  tabanid transmission, 338–339
  tick transmission, 646–647, 656–657
Tumbler, mosquito, 266
Tumbu fly, 401, 401f
Tungiasis, 162
Turbinoptidae, 591
Turkestan cockroach, 65, 65f
Turkey
  beetle infestation, 138
  biting midges, 224, 229
  blood protozoans, 229
  borreliosis, 656
  fleas, 152
  leucocytozoonosis, 252
  malaria, 312
  mites, 571
  nematodes, 74
  tapeworms, 139, 139f
Turtle
  blood protozoans, 340
  chiggers, 544
  tabanid disease transmission, 340
Two-dimensional gel electrophoresis, 686
Typhus. *See* Epidemic typhus; Murine typhus; Recrudescent typhus

U

Urban plague, 157
Urban typhus. *See* Murine typhus
Urinary acariasis, 556
Urodacidae, 493
Urticaria, 439
  caterpillars, 444–450

V

Vaccines
  African horsesickness, 228
  babesiosis, 629
  bluetongue disease, 225
  epidemic typhus, 95
  fleas, 166–167
  human scabies, 550–551
  leishmaniasis, 210
  malaria, 312
  mosquito borne diseases, 317
  plague, 159
  polio, 186, 294
  tick-borne encephalitis, 629
  tick control, 659, 661–662
  Western equine encephalomyelitis, 35
  yellow fever, 290
Vaejovidae, 492
Vagabond's disease, 87–88
Vector competence, 37
Vectorial capacity, 43
Velvet ant, 28, 460, 471, 471f
Venereal transmission, 39
Venezuelan equine encephalitis, 221–222, 250, 285–286
Venom, 23, 23f
  ants, 465–466
  arthropods, 28–29
  bees, 466
  caterpillars, 440
  effects, 24–28
  loxoscelism, 522–523
  peptides, 28–29
  scorpions, 499
  tarantulas, 515
  wasps, 465
Vermipsyllidae, 145
Vertical transmission, 37–38
Vesicular stomatitis, 186, 198–200
Vespidae, 460, 472–473
Vespinae, 460
Vespoidea, 459
Vinchuca, 109
Vinegaroons, 27f
Visceral leishmaniasis, 207
Viscerocutaneous loxoscelism, 523
Vitellogenin, 271

W

Walkingstick, 9
Wall louse, 119
Walnut caterpillar, 455f
Warble, 364f, 391–392
Wasps. *See also* Hymenoptera
  behavior and ecology, 464–465
  life history, 463–464
  morphology, 460–463
  prevention and control, 483–484
  public health importance, 479–482
  social wasps, 472–473
  solitary wasps, 471
  taxonomy, 459–460, 460t
  venoms, 465–466
  veterinary importance, 483
Water buffalo
  hippoboscids, 428
  horn fly, 355
  lachryphagous moths, 444
  skin-piercing moths, 452
Water buffalo skin maggot, 401
Wesselsbron virus, 311
West African trypanosomiasis, 375–376
Western analysis, 686
Western black widow spider, 526
Western equine encephalitis, 123, 285
Western yellowjacket, 475
West Nile virus, 293–294, 293f
Whamefly, 327
Wheel bug, 107
Whiplash dermatitis, 135
White-flannel moth, 445f
Whole genome sequencing, 678
Whole-genome shotgun sequencing, 683
Wind-scorpion, 505
Wind-spider, 505
Wing louse, 93–94
Wings
  beetles, 129–130, 130f
  black fly, 240
  cockroaches, 62
  Diptera, 176
  kissing bugs, 110
  mosquito, 266–267, 267f
  muscid flies, 347–348
  parasitic arthropods, 17
  true bugs, 107, 108f
  tsetse fly, 370–371, 371f
Wolf. *See also* Dog
  fleas, 151
  lice, 84
  myiasis, 391–392
  sarcoptic mange, 578
Woodchuck. *See* Marmots
Wriggler, mosquito, 264–265

X

Xenodiagnosis, 116

Y

Yellow fever
  geographic distribution, 288–289, 288f
  transmission, 289
  virus, 289
Yellowjacket, 460, 463, 471, 473–475, 483–484
Yellow scorpion, 500

Z

Zebu, 450f, 452, 650
Zinsser, Hans, 96
Zoonosis, 40–41
Zoophagic vector, 36–37
Zoophilous, 444
Zoridae, 510
Zygaenoidea, 439

This page intentionally left blank

MEDICAL AND VETERINARY ENTOMOLOGY
THIRD EDITION

Edited by

Gary R. Mullen
Professor Emeritus
Department of Entomology and Plant Pathology
Auburn University, Auburn, AL, USA

Lance A. Durden
Professor and Curator
Department of Biology
Georgia Southern University, Statesboro, GA, USA

The first and second editions of *Medical and Veterinary Entomology*, edited by Gary R. Mullen and Lance A. Durden, published in 2002 and 2009, respectively, have been highly praised and become widely used as a textbook for classroom instruction. This fully revised third edition continues the focus on the diversity of arthropods affecting human and animal health, with separate chapters devoted to each of the taxonomic groups of insects and arachnids of medical or veterinary concern, including spiders, scorpions, mites, and ticks. Each chapter includes sections on taxonomy, morphology, life history, and behavior and ecology, with separate sections on those species of public-health and veterinary importance. Each concludes with approaches to management of pest species and prevention of arthropod-borne diseases. The third edition provides a comprehensive source for teaching medical and/or veterinary entomology at the college and university level, targeted particularly at upper-level undergraduate and graduate/postgraduate programs. In addition to its value as a student textbook, the volume has appeal to a much broader audience, specialists and non-specialists alike. It provides a key reference for biologists in general, entomologists, zoologists, parasitologists, physicians, public-health personnel, veterinarians, wildlife biologists, vector biologists, military entomologists, the general public and others seeking a readable, authoritative account on this important topic.

Key Features

- Completely revised and updated edition
- A distinguished group of 40 nationally and internationally recognized contributors
- Sixteen new authors, in addition to 25 continuing contributors to the first and second editions
- A new chapter on *Arthropod Toxins and Venoms*
- Illustrated with 560, mostly color, figures and updated maps depicting the distribution of important arthropod taxa and arthropod-borne diseases
- A significantly expanded and well-illustrated chapter on *Molecular Tools Used in Medical and Veterinary Entomology*
- Coverage of emerging and newly recognized arthropod concerns, including mosquito-borne Zika and Chikungunya viruses; tick-borne Bourbon and Heartland viruses; tick-borne rickettsioses and anaplasmosis; and red meat allergy associated with tick bites
- A 1700-word Glossary
- An Appendix of *Arthropod-Related Viruses of Medical and Veterinary Importance*

Cover Images: **Main image:** Asian tiger mosquito *(Aedes albopictus)*. Copyright photo by Jena Johnson. Jena Johnson Photography, Athens, GA. **Vertical panel images** (top to bottom): American dog tick *(Dermacentor variabilis)*, female. Photo by James Gathany, Centers for Disease Control and Prevention, Atlanta, GA; Oriental rat flea *(Xeopsylla cheopis)*, male. Photo by Lorenza Beati and Lance Durden, Georgia Southern University, Statesboro, GA; Saddleback caterpillar *(Sibine stimulea)*. Photo by Sturgis McKeever, Georgia Southern University, Statesboro, GA; Scorpion *(Tityus stigmurus)*, Brazil. Photo by W. David Sissom. West Texas A&M University, Canyon, TX; Greenbottle fly *(Calliphoridae)*. Copyright photo by Jena Johnson. Jena Johnson Photograpy, Athens, GA.

ELSEVIER

ACADEMIC PRESS
An imprint of Elsevier
elsevier.com/books-and-journals

ISBN 978-0-12-814043-7
9 780128 140437