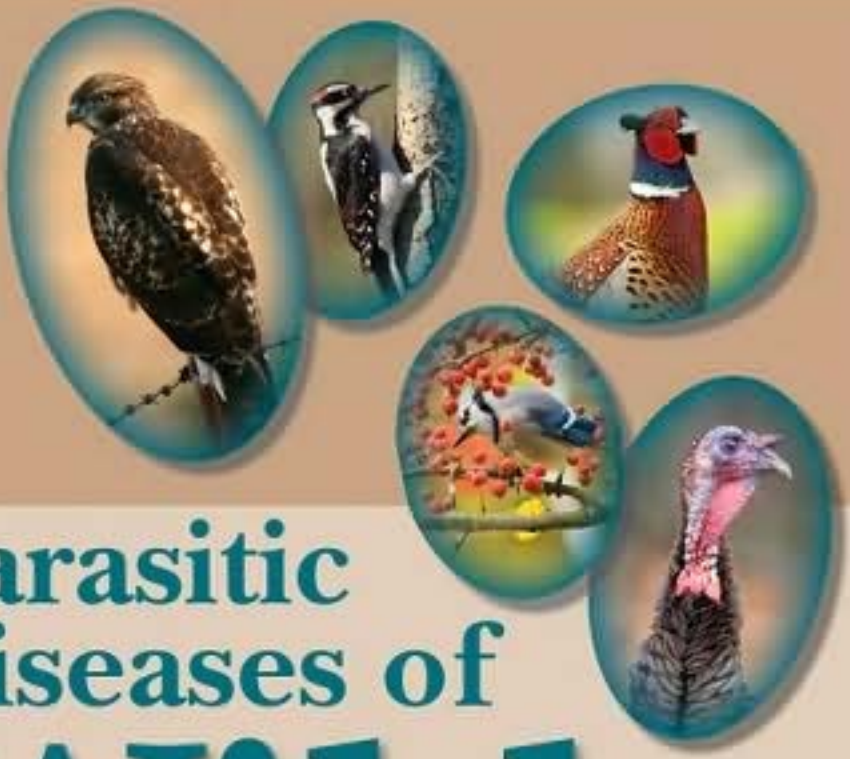


Parasitic Diseases of Wild Birds

Edited by
Carter T. Atkinson, Nancy J. Thomas & D. Bruce Hunter

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Contents

Preface vii
Contributors ix

Section I Introduction

1. Parasitism: Costs and Effects 3
Gary A. Wobeser

Section II Protozoa

2. *Haemoproteus* 13
Carter T. Atkinson

3. Avian Malaria 35
Carter T. Atkinson

4. Leucocytozoonosis 54
Donald J. Forrester and Ellis C. Greiner

5. *Isospora, Atoxoplasma*, and *Sarcocystis* 108
Ellis C. Greiner

6. Trichomonosis 120
Donald J. Forrester and Garry W. Foster

7. *Histomonas* 154
William R. Davidson

8. *Eimeria* 162
Michael J. Yabsley

9. Disseminated Visceral Coccidiosis in Cranes 181
Marilyn G. Spalding, James W. Carpenter, and Meliton N. Novilla

10. *Cryptosporidium* 195
David S. Lindsay and Byron L. Blagburn

11. *Toxoplasma* 204
J. P. Dubey

Section III Helminths

12. Trematodes 225
Jane E. Huffman

13. Schistosomes 246
Jane E. Huffman and Bernard Fried

14. Cestodes 261
J. Daniel McLaughlin

15. Acanthocephala 277
Dennis J. Richardson and Brent B. Nickol

16. Eustrongylidosis 289
Marilyn G. Spalding and Donald J. Forrester

17. *Trichostrongylus* 316
Daniel M. Tompkins

18. *Dispharynx, Echinuria*, and *Streptocara* 326
Ramon A. Carreno

19. Tracheal Worms 343
M. A. Fernando and John R. Barta

20. *Amidostomum* and *Epomidiostomum* 355
Alan M. Fedynich and Nancy J. Thomas

21. Tetrameridosis 376
John M. Kinsella and Donald J. Forrester

22. Avioserpensosis 384
John M. Kinsella

23. *Heterakis* and *Ascaridia* 388
Alan M. Fedynich

24. Ascaridoid Nematodes: *Contracaecum, Porrocaecum*, and *Baylisascaris* 413
Hans-Peter Fagerholm and Robin M. Overstreet

25. *Diplotriaena, Serratospiculum,* and *Serratospiculoides* 434
Mauritz C. Sterner III and Rebecca A. Cole

26. Filarioid Nematodes 439
Cheryl M. Bartlett

27. Capillarid Nematodes 463
Michael J. Yabsley

Section IV Leeches

28. Leech Parasites of Birds 501
Ronald W. Davies, Fredric R. Govedich, and William E. Moser

Section V Arthropods

29. Phthiraptera, the Chewing Lice 515
Dale H. Clayton, Richard J. Adams, and Sarah E. Bush

30. Acariasis 527
Danny B. Pence

31. Black Flies (Diptera: Simuliidae) 537
Douglas C. Currie and D. Bruce Hunter

32. Myiasis in Wild Birds 546
Susan E. Little

Index 557

Preface

More that 30 years ago, John W. Davis, Roy C. Anderson, Lars Karstad, and Daniel O. Trainer edited the first edition of *Infectious and Parasitic Diseases of Wild Birds*. Since then there has been an explosion of new knowledge about parasitic diseases of wild birds, as wildlife disease specialists, ecologists, and evolutionary biologists have continued to unravel how parasitic protozoans, helminths, and ectoparasites affect wildlife populations. We continue in the footsteps of the first editors of this work by significantly expanding and updating the parasite portion of their original book. This work is a companion volume to *Infectious Diseases of Wild Birds*, which was published in 2007 by Blackwell Publishing, and complements *Infectious Diseases of Wild Mammals*, 3rd edition, edited by Elizabeth S. Williams and Ian K. Barker, and *Parasitic Diseases of Wild Mammals*, 2nd edition, edited by William M. Samuel, Margo J. Pybus, and A. Alan Kocan (Iowa State University Press). Taken together, these four volumes provide an important source of reference material for biologists and wildlife mangers, wildlife and veterinary students, professionals in the fields of animal health and wildlife disease, and evolutionary biologists with interests in disease ecology. We gratefully acknowledge our colleagues who established such excellent models for us to follow.

This book focuses on the disease conditions produced by parasitic protozoans, helminths, leeches, and ectoparasitic arthropods, e.g. mites, and biting flies in free-living wild birds. Unlike most parasitology texts, this book emphasizes effects on the host rather than the parasites themselves, but still includes important information about their etiology, life cycles, transmission, and diagnosis. While no single work can cover the entire spectrum of wildlife parasites, we have attempted to assemble chapters that are both specific (e.g., Chapter 9, Disseminated Visceral Coccidiosis in Cranes) and general (e.g., Chapter 14, Cestodes) in their treatment of some of the diverse groups of organisms that use wild birds as intermediate or definitive hosts. In all cases, we have urged authors to avoid generalities and include specific examples of host–parasite associations that can lead to clinical disease. We owe a great debt to the authors of these chapters both for their expertise in the material and for their willingness to endure the inevitable delays and revisions that are inherent in multiauthored works.

Each chapter provides a classical description of the history, effects on the host, and causative agent, but the authors were also challenged to provide perspectives on the significance of the disease to wild birds and to document population impacts, an aspect that is particularly difficult to quantify in the wild. Unlike other volumes in this series, we elected to begin this book with an introductory chapter by Gary A. Wobeser who discusses some of the costs and effects of parasitism in wild avian populations. This chapter provides a succinct discussion of some of the difficulties in assessing impacts of parasitism on wild birds and provides a good framework for assimilating the detailed information in the sections that follow.

We used *The Clements Checklist of Birds of the World*, 6th edition (Cornell University Press, 2007), as the authority for avian nomenclature and elected to allow authors to make individual decisions about whether to follow the proposed standardized nomenclature for parasitic diseases (SNOPAD; http://www.waavp.org/node/40). As a result, some chapters follow this terminology (e.g., Chapter 4, Leucocytozoonosis) while others retain the more traditional terminology (e.g., Chapter 7, *Histomonas*). Because many unpublished data on wild bird diseases have been compiled in laboratory and diagnostic files, citations of unpublished data were allowed for repositories of large, permanent, accessible institutions, such as the Canadian Cooperative Wildlife Health Centre, U.S. Geological Survey National Wildlife Health Center, and Southeastern Cooperative Wildlife Disease Study.

Grateful acknowledgment goes to the Iowa State University Press, which guided this project through its initial stages, and to Blackwell Publishing, which took it over and shepherded it through to completion. We owe sincere debts of gratitude to Donald J. Forrester who was instrumental in the initial organization of the book and to Amy Miller for her significant

contribution in the technical editing of the final manuscript. We acknowledge the support of the U.S. Geological Survey, Wildlife and Terrestrial Resources Program, and the University of Guelph. This book is dedicated to the Wildlife Disease Association, whose members initiated the revision of this book series and who continue to provide the backbone of growing knowledge in the field of wildlife disease. Royalties that accrue from sales of this book will be provided to the Wildlife Disease Association.

Carter T. Atkinson
Nancy J. Thomas
D. Bruce Hunter

Contributors

Richard J. Adams
Department of Biology
University of Utah
Salt Lake City, Utah, U.S.A.

Carter T. Atkinson
U.S. Geological Survey
Pacific Island Ecosystems Research Center
Hawaii National Park, Hawaii, U.S.A.

John R. Barta
Department of Pathobiology
Ontario Veterinary College
University of Guelph
Guelph, Ontario, Canada

Cheryl M. Bartlett
Department of Biology
Cape Breton University
Sydney, Nova Scotia, Canada

Byron L. Blagburn
Department of Pathobiology
College of Veterinary Medicine
Auburn University, Alabama, U.S.A.

Sarah E. Bush
Natural History Museum *and*
Biodiversity Research Center
University of Kansas
Lawrence, Kansas, U.S.A.

James W. Carpenter
Department of Clinical Sciences
College of Veterinary Medicine
Kansas State University
Manhattan, Kansas, U.S.A.

Ramon A. Carreno
Department of Zoology
Ohio Wesleyan University
Delaware, Ohio, U.S.A.

Dale H. Clayton
Department of Biology
University of Utah
Salt Lake City, Utah, U.S.A.

Rebecca A. Cole
U.S. Geological Survey
National Wildlife Health Center
Madison, Wisconsin, U.S.A.

Douglas C. Currie
Royal Ontario Museum
Toronto, Ontario, Canada
and Department of Ecology
and Evolutionary Biology
University of Toronto
Toronto, Ontario, Canada

William R. Davidson
D. B. Warnell School of Forestry and Natural
Resources *and* Southeastern
Cooperative Wildlife Disease Study
College of Veterinary Medicine
University of Georgia
Athens, Georgia, U.S.A.

Ronald W. Davies
Department of Biological Sciences
University of Calgary
Calgary, Alberta, Canada

J.P. Dubey
Animal Parasitic Diseases Laboratory
Animal and Natural Resources Institute
Agricultural Research Service
U.S. Department of Agriculture
Beltsville, Maryland, U.S.A.

Hans-Peter Fagerholm
Laboratory of Aquatic Pathobiology
Department of Biology
Abo Akademi University
Åbo, Finland

Alan M. Fedynich
Caesar Kleberg Wildlife Research Institute
Texas A&M University-Kingsville
Kingsville, Texas, U.S.A.

M.A. Fernando
Department of Pathobiology
Ontario Veterinary College
University of Guelph
Guelph, Ontario, Canada

Donald J. Forrester
Department of Infectious
Diseases and Pathology
College of Veterinary Medicine
University of Florida
Gainesville, Florida, U.S.A.

Garry W. Foster
Department of Infectious
Diseases and Pathology
College of Veterinary Medicine
University of Florida
Gainesville, Florida, U.S.A.

Bernard Fried
Department of Biology
Lafayette College
Easton, Pennsylvania, U.S.A.

Fredric R. Govedich
Department of Biological Sciences
Southern Utah University
Cedar City, Utah, U.S.A.

Ellis C. Greiner
Department of Infectious
Diseases and Pathology
College of Veterinary Medicine
University of Florida
Gainesville, Florida, U.S.A.

Jane E. Huffman
Department of Biological Sciences
Applied DNA Sciences
Fish and Wildlife Microbiology Laboratory
East Stroudsburg University
East Stroudsburg, Pennsylvania, U.S.A.

D. Bruce Hunter
Department of Pathobiology
Ontario Veterinary College
University of Guelph
Guelph, Ontario, Canada

John M. Kinsella
Department of Infectious
Diseases and Pathology
College of Veterinary Medicine
University of Florida
Gainesville, Florida, U.S.A.

David S. Lindsay
Department of Biomedical
Sciences and Pathobiology
Virginia-Maryland Regional
College of Veterinary Medicine
Virginia Polytechnic Institute
and State University
Blacksburg, Virginia, U.S.A.

Susan E. Little
Department of Veterinary Pathobiology
Center for Veterinary Health Sciences
Oklahoma State University
Stillwater, Oklahoma, U.S.A.

J. Daniel McLaughlin
Department of Biology
Concordia University
Montreal, Quebec, Canada

William E. Moser
Department of Invertebrate Zoology
National Museum of Natural History
Smithsonian Institution
Washington, District of Columbia, U.S.A.

Brent B. Nickol
School of Biological Sciences
University of Nebraska-Lincoln
Lincoln, Nebraska, U.S.A.

Meliton N. Novilla
WIL Research Laboratories—Biotechnics LLC
Greenfield, Indiana, U.S.A.

Robin M. Overstreet
The University of Southern Mississippi
Gulf Coast Research Laboratory
Ocean Springs, Mississippi, U.S.A.

Danny B. Pence
Department of Pathology
Texas Tech University Health
Sciences Center
Lubbock, Texas, U.S.A.

Dennis J. Richardson
Department of Biological Sciences
Quinnipiac University
Hamden, Connecticut, U.S.A.

Marilyn G. Spalding
Department of Infectious
Diseases and Pathology
College of Veterinary Medicine
University of Florida
Gainesville, Florida, U.S.A.

Mauritz C. Sterner III
U.S. Geological Survey
National Wildlife Health Center
Madison, Wisconsin, U.S.A.

Nancy J. Thomas
U.S. Geological Survey
National Wildlife Health Center
Madison, Wisconsin, U.S.A.

Daniel M. Tompkins
Landcare Research
Dunedin, New Zealand

Gary A. Wobeser
Department of Veterinary Pathology
Western College of Veterinary Medicine
University of Saskatchewan
Saskatoon, Saskatchewan, Canada

Michael J. Yabsley
D. B. Warnell School of
Forestry and Natural Resources
and Southeastern Cooperative
Wildlife Disease Study
Department of Population Health
College of Veterinary Medicine
University of Georgia
Athens, Georgia, U.S.A.

Section I: Introduction

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1
Parasitism: Costs and Effects

Gary A. Wobeser

Parasitism has been defined in many ways, but in terms of wildlife disease, it is usually taken to mean an obligatory trophic association between individuals of two species in which one (the parasite) derives its food from a living organism of the other species (the host). An individual host bird can be viewed as an island of habitat that provides resources for parasites, with the parasites deriving benefits while the host is harmed or bears some cost. Parasitism is common in nature; for example, Price (1980) estimated that half of all animal taxa are parasitic. Parasitism is ubiquitous in wild birds and individual birds are affected by many different parasites during their lifetime, but our understanding of the parasites that occur in wild birds is fragmentary.

Moore and Clayton (1997) concluded that the majority of parasites of wild birds have yet to be described taxonomically. Some groups, such as blood-inhabiting protozoa (the hematozoa), have been studied widely, perhaps because of the ease with which blood can be collected from living birds, while little is known about other groups such as intestinal flagellates. But even within the hematozoa, species diversity has probably been greatly underestimated (Bensch et al. 2007). Similarly, more is known about the effects of arthropod ectoparasites than about the effect of protozoa and helminths on birds, and cavity nesting birds have been studied more extensively than most other species because of the relative ease in capturing, examining, and following these birds.

Studying parasitism in wild birds is subject to a number of constraints that make working with disease in any free-ranging species more difficult than studying humans or domestic animals. These include:

- inadequate baseline information about the host species. Knowledge of avian life history traits is rudimentary (Zera and Harshman 2001), and so one often must extrapolate from other species and collect information about the basic biology of the host while trying to understand a host–parasite relationship;
- difficulty in quantifying factors related to disease. It is impossible to assess the significance of a parasite for a population without the ability to calculate basic epidemiological proportions such as prevalence, incidence, morbidity, and mortality rates. The number of individuals affected by a parasite (the numerator for such calculations) is usually difficult to determine and the population at risk (the denominator) rarely can be measured adequately;
- the need to consider the long-term effect of a parasite in wild birds. This may be very difficult, even when the number affected and the population at risk can be determined. If a disease, such as coccidiosis, occurs in a flock of chickens and 15% die, the significance of the disease is that 15% fewer chickens go to market. However, a similar 15% loss in a wild bird population might result in more resources per capita for the remaining birds, leading to reduced mortality from other factors and/or improved reproduction. The potential for compensation or other delayed effects may be very important in assessing the impact of a parasite on wild birds at the population level;
- the sample of wild birds available for study is usually biased by the method of collection and may not represent the actual state of nature. Depending on the method of collection, affected birds may be under- or overrepresented, even in groups collected by mass-capture methods (Sulzbach and Cooke 1978); and
- the anonymity of wild birds, except for the small number marked by the researcher. For instance, although age is an important disease determinant, the age of wild birds often cannot be determined except to differentiate hatch-year from after-hatch-year birds. Individuals seldom can be traced back in time to discover previous exposure to disease agents or forward in time to discover

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their fate. Commonly used techniques such as retrospective and prospective case–control studies that are useful in human and veterinary epidemiology are impossible except in unusual circumstances, such as in birds with a high degree of nest site fidelity.

A fundamental feature of parasitism is that the presence of a parasite involves a cost to the host. The costs of parasitism may include:

- loss of resources extracted by the parasite directly from the host, for example, loss of blood to blood-feeding ectoparasites;
- competition between the parasite and the host for resources, as occurs with cestodes that absorb nutrients from the host's gut content;
- costs to the host for defense against parasites. These may include foregoing resource-rich areas to avoid areas where parasites may be present, costs for grooming, moving away from parasites, or abandoning a nest, costs to develop and maintain innate and acquired resistance, and costs to activate these systems;
- costs resulting from tissue injury related to the parasite. This may be direct damage caused by the parasite or, more often, injury from the inflammatory and immune response to the parasite. Some injuries may result in dysfunction, such as reduced mobility, reduced digestive efficiency, or increased loss of nutrients through intestinal or kidney injury, that interfere with obtaining or retaining resources;
- costs related to improper development as a result of parasitism early in life (e.g., Spencer et al. 2005); and
- costs to repair or replace damaged tissues.

The diversity of parasites and the variety of ways that they interact with hosts make it difficult to measure the cost of a single parasite species; to compare the relative cost of different parasites such as the lice, intestinal coccidia, and tracheal worms, all of which might be infecting a single host; or to understand how these parasites may interact with each other and with other environmental factors to affect a host population. The costs described above are related to resources, and particularly to energy ["*the single common denominator of life*"; "*something that is absolutely essential and involved in every action large or small*" (Odum 1993)]. Energy is a measure of the ability to do work and is a "currency" that can be used to consider the costs of all types of parasitism, at least conceptually if not quantitatively at this time. Four basic features must be considered when using energy as a currency to consider parasitism:

- The supply of energy is limited. Most birds are unable to increase their intake of energy readily, and so they must function within a finite budget. In other words, a bird cannot use more energy than it can assimilate or has in storage.
- The amount of energy available and accessible is not constant or uniform. The energy available to a bird varies with the time of year, weather, habitat conditions, and the number of competitors for that energy. Not all individuals in a population have equal access to the resources that are available; thus, within a group or population some birds may have abundant resources while others do not.
- Use of energy for one purpose reduces the amount available for other uses. Most of the energy assimilated by a bird is used for maintenance, that is, keeping the body functioning, repaired, maintaining a high core temperature, avoiding predators, and defending against disease. Energy that remains can be used for production (growth and reproduction) or stored as fat for future use. If extra energy is used to defend against parasites or to repair tissue injured by parasites, the energy available for reproduction or growth is reduced. For instance, the cost of producing antibody to a novel antigen is equivalent to that of producing half an egg in female House Sparrows (*Passer domesticus*) (Martin et al. 2003) and mounting an immune response resulted in asymmetry of flight feathers in nestling Mountain Chickadees (*Poecile gambeli*) (Whitaker and Fair 2002). Conversely, increased reproductive effort may result in reduced ability to mount a defense against parasites (Deerenberg et al. 1997).
- The need for energy for various purposes is highly variable among individuals and at different times of year.

Because an individual cannot maximize all life history traits simultaneously, life history theory suggests that a bird should adopt a strategy that optimizes energy use among resource-demanding activities, such as defense and reproduction, to maximize lifetime fitness. Ecologists use the term "trade-off" for this process of making physiological choices among competing needs for resources that should maximize the chances of an individual's genes being passed on to the next generation. Individuals that make the wrong choices are less successful or "fit," and this may provide a basis for genetic selection.

As a result of heterogeneity in both the supply of energy and the need for energy, the appropriate physiological trade-offs in relation to parasitism vary among individual birds and for different parasites, and the pattern of trade-offs is different seasonally and annually. For this reason, the reaction to parasites and the effects of parasitism must always be considered in terms of the context in which parasitism is occurring and of how the situation might influence resource trade-offs. For instance, during one season a bird may be in poor nutritional condition and need to direct all its available resources to simply staying alive, with little or no ability to mount an effective defense against parasites or to grow or reproduce. At another time of year the same bird may have ample resources to meet all needs, and so it can afford strong resistance to parasites and still be able to grow and reproduce effectively.

Young birds may have different priorities than adults and the sexes may have different strategies and trade-offs. For instance, Tschirren et al. (2003) suggested that a greater need for carotenoid-based coloration for signaling by male Great Tits (*Parus major*) might lead to a trade-off that results in reduced immunocompetence in males. Privileged individuals within the population, such as birds that possess a territory, may have a totally different context for trade-offs related to parasites than do the "have-nots" within the population. Changes in environmental conditions may change the context; for example, Blow Fly (*Protocalliphora braueri*) larvae had no effect on Sage Thrasher (*Oreoscoptes montanus*) nestling weight, size at fledging, or mean fledgling age, but in a year with cold wet weather, survival and fledging success were markedly reduced among parasitized birds compared to unparasitized birds (Howe 1992).

Knowledge of how trade-offs occur in relation to parasitism is fragmentary at this time and general rules about which activity (reproduction, growth, defense against predators or parasites) should take precedence for resources are likely subject to many exceptions. For instance, hosts may be selected to develop acquired immunity to only some of the disease agents that they encounter (Boots and Bowers 2004). While mounting a strong defensive response to parasites is likely a "good" thing generally, in some situations it may be adaptive to suppress the defensive response. This may be the case in nesting Common Eiders (*Somateria mollissima*). Female eiders do not feed during breeding and face severe resource restrictions while incubating. Birds that do not begin with adequate resources abandon their nest in order to survive.

Hanssen et al. (2004) immunized incubating female eiders with nonpathogenic antigens, including sheep red blood cells. Not surprisingly, the rate of successful immunization was not very good compared to what would be expected at other times of year. Under these circumstances, it appears that the appropriate choice for many eiders is to use their limited resources to survive and reproduce rather than to mount an immune response. A second part of the same study compared survival of birds that mounted an immune response to that of birds that did not produce antibodies. Both responding and nonresponding eiders had sufficient resources to complete reproduction; however, only about 27% of birds that produced antibody to sheep red blood cells returned to the colony in subsequent years, compared with approximately 72% of birds that did *not* produce antibody. Under these conditions, females that invested in an immune response "*experienced considerably impaired long-term survival*" compared to females that did not respond. This example also serves to illustrate that the effect of a trade-off on fitness may be delayed.

The cost to the host is not obvious for most parasites encountered in wild birds. It is only in a minority of situations, described elsewhere in this book, that parasitism is clearly associated with recognizable functional impairment of the host that we can characterize as disease. The apparently "benign" nature of many parasites could be because:

- the effect of the parasites actually is so trivial as to be undetectable;
- the cost is not trivial but it is tolerable; that is, the bird has sufficient resources to cover the costs without significant negative effects on other functions *under conditions at the time the effect was measured*;
- the cost of parasitism is obscured by other more proximate regulatory factors such as predation and competition. Predation is thought to be a major factor in shaping the life history of birds (Zera and Harshman 2001) and parasitized prey may be taken disproportionately by predators (Temple 1987). In some situations the parasite benefits if the infected host is eaten by an appropriate predator (parasite-induced trophic transmission; Lafferty 1999). But infections in which there is no apparent benefit to the parasite may make animals more susceptible to predators, perhaps because of the pathology induced by the parasite. Hudson et al. (1992a) found that Red Grouse (*Lagopus lagopus scotica*) killed by predators were more heavily parasitized by the cecal nematode (*Trichostrongylus tenuis*) than were hunter-killed birds and that birds with many worms may emit more scent and, hence, be more vulnerable to mammalian predators. In some situations, increased vulnerability to predators may be related to energy trade-offs and reduced resources for

predator vigilance or avoidance. For instance, Common Redshanks (*Tringa totanus*) that are energetically stressed (as might result from parasitism) respond by taking risks that increase the probability of predation (Quinn and Cresswell 2004). The interaction between predation and parasitism is undoubtedly complex. Navarro et al. (2004) found that House Sparrows exposed to potential predators (cat or owl) had reduced T-cell-mediated immune response and a higher prevalence and intensity of infection with *Haemoproteus* spp. than did sparrows exposed to nonthreatening animals (rabbit or pigeon), suggesting that even the threat of predation may alter trade-offs that influence parasitism. Although little is known about the effect of parasitism on intraspecific competition, this may be an important factor. For instance, male Greater Sage-Grouse (*Centrocercus urophasianus*) infested with lice are discriminated against for breeding (Spurrier et al. 1991). Females appear to recognize infected males by the occurrence of petechial hemorrhages on the air sacs and males infested with lice are shunned, and so their reproductive input to the population is minimal; that is, their fitness is very low and there is likely negative selection against their genotype. In a similar manner, male Red Grouse infected with *T. tenuis* may have difficulty defending a territory (Delahay et al. 1995). Consideration of interactions between parasitism and competition must also include competition among species that share parasites, such as the Ring-necked Pheasant (*Phasianus colchicus*) and Gray Partridge (*Perdix perdix*) that share *Heterakis gallinarum,* with asymmetrically severe effects on the partridge (Tompkins et al. 2001b); and

- the cost is not trivial but it goes undetected because of insensitivity of the methods used to look for effects. For instance, it would be very easy to dismiss the tiny hemorrhages caused by lice as inconsequential to male Greater Sage-Grouse, without even considering that they might have a profound effect on behavior, reproductive success, and natural selection. The costs of parasitism could also be overlooked because the wrong individuals within the population are examined, the interaction between parasite and host is examined in an inappropriate context (e.g., at the wrong time of year or in an experimental situation in which resources are not limited), inappropriate parameters are measured, or because the long-term (lifetime) consequences of parasitism are not measured. Møller (1994) suggested that the cost of parasitism in nestling birds could be paid by the nestlings through reduced growth or survival or by the parents through reduced survival or future reproductive success as a result of having to provide additional resources to the parasitized young. Bize et al. (2003) found that nestling Alpine Swifts (*Tachymarptis melba*) can compensate for early growth retardation by rapid feather growth, so that if measured at fledging no effect might be obvious; however, rapid feather growth may result in poor feather quality with later effects (Dawson et al. 2000). Nutrient shortage in early development can have other serious long-term consequences including effects on adult dominance rank, morphology, and lifespan (Metcalfe and Monaghan 2001). Island Canaries (*Serinus canaria*) infected with plasmodia as nestlings have structural changes in their brain and reduced song repertoire as adults (Spencer et al. 2005). The effects of parasites are usually not distributed evenly or fairly among all members of a population, which complicates measuring their cost. Metazoa characteristically are distributed in an aggregated manner within the host population (Shaw et al. 1998). Most hosts have few or no parasites and a few individuals have many parasites (often referred to as the 20:80 rule: 20% of the population carries 80% of the parasites). Severe effects are likely to be confined to those individuals with many parasites. Measures of central tendency, such as average intensity of infection and average cost of parasitism, may not be helpful in understanding the significance of the parasite if effects are concentrated in a small group of heavily infected individuals. These animals at the extreme end of the distribution are also important as the major source of infection within the population, but samples drawn from the population are unlikely to contain these individuals unless the sample is very large. Much of the information available on the occurrence of parasites in wild birds comes from the study of birds that died of other causes, because it is inappropriate to kill large samples of birds simply to record their parasites. At one extreme, such a sample may primarily consist of the survivors of conditions that were severe, resulting in underestimation of the cost of parasitism. At the opposite extreme, the sample may contain the few significantly affected individuals in the population, and so the cost to the population is overestimated.

"*While the study of specific host–parasite relationships have proven insightful, they reflect only a small part of the wealth of parasites and pathogens in an*

animal's internal and external environment" (Lochmiller and Deerenberg 2000). Virtually all the information available about parasites of birds relates to the effects of individual parasite species, but individual birds are host to many different parasites, often simultaneously; for example, a single feather may be infested with 6 species of feather mite (Pérez and Atyeo 1984) and a group of 45 Lesser Scaup (*Aythya affinis*) were infected by almost 1 million individuals of 52 different helminth species (Bush and Holmes 1986). Examining the effect of parasitism as the interaction between two species fails to account for interactions among parasites that might be additive, synergistic, or antagonistic. Almost nothing is known about the effects or dynamics of parasite assemblages or communities in wild birds.

The largest challenge for those interested in parasites of birds is to answer the question "Do parasites influence bird populations?" Most ecologists and wildlife managers have assumed that the answer is "No" (Tompkins et al. 2001a), but modeling suggests that parasites could regulate host populations if they reduce host survival and/or fecundity in a density-dependent manner (Anderson and May 1978; May and Anderson 1978). To understand the effect of a parasite on the host population, one needs to understand the effect of the parasite on the individual host, the prevalence and intensity of parasite infection within the host population, and the context within which the interaction is occurring. Parasites rarely result in obvious piles of dead birds but many studies have concentrated on the direct effect of parasites on mortality although "*. . . highly pathogenic parasites tend not to have an impact at the population level. . . .*" (Hudson and Dobson 1997), because this type of parasite may kill the host rapidly, thus limiting transmission to other individuals. Sublethal effects of chronic infections that are mediated through reduced fecundity are more likely to have an effect at the population level.

Much of the information available about parasites in birds is descriptive. More than 70 years ago, Aldo Leopold recognized that observational and correlational studies have limited ability to lead to an understanding of disease in wild species (Leopold 1933). Marzal et al. (2005) observed that knowledge of causal relationships of disease caused by parasites of birds "*is still rudimentary due to a scarcity of experimental manipulation*," and Tompkins and Begon (2000) stated that "*regulation by parasites can be established only by experimentally perturbing host/parasite systems away from their equilibrium levels and monitoring subsequent changes in both parasite and host densities relative to control*." Studies that include intervention through treatment of parasites in natural populations, such as by Hudson et al. (1992b, 1998) (*T. tenuis* and Red Grouse), Merino et al. (2000) (hematazoa in Eurasian Blue Tits, *Cyanistes caeruleus*), Hoodless et al. (2002) (ticks and Ring-necked Pheasants) and Marzal et al. (2005) (*Haemoproteus prognei* in House Martins, *Delichon urbicum*), and through experimental infection (e.g., Spencer et al. 2005), have provided insights into parasitism that would be unattainable with traditional observational study. As in all aspects of the study of parasitism, it is important to consider the long-term effects of such interventions. For instance, Hanssen et al. (2003) studied the effect of antiparasite treatment on nesting female eiders. There was no effect of treatment on nest success or on the survival to the next year of birds that nested successfully. However, among the females that were unsuccessful in nesting, 69% of treated birds survived compared with 18% of untreated birds. This suggests that birds that nested successfully were able to tolerate the effects of parasitism, while unsuccessful females were less able to bear the costs from parasites, resulting in a delayed effect on survival. In another example, McCutchan et al. (2004) found that a vaccine significantly protected canaries against natural infection with *Plasmodium relictum* in the year of vaccination. In the following year, survivors in the vaccinated group suffered much higher mortality than unvaccinated birds that had survived exposure in year 1, presumably because vaccine-induced immunity prevented acquisition of protective natural immunity.

Wild birds have developed a suite of trade-offs that allow them to be successful under a particular set of conditions. Environmental cues, such as photoperiod, may guide the timing of these trade-offs. Our world is changing rapidly and dramatically, especially for many wild species. With rapid anthropogenic alterations, such as climate change and environmental contamination, cues that were reliable may no longer be associated with adaptive outcomes (Schlaepfer et al. 2002). If birds are trapped by their evolutionary response to cues, they may find themselves equipped with attributes that are no longer optimal. Schlaepfer et al. (2002) used the term "*evolutionary trap*" for decisions that are now maladaptive because of a sudden anthropogenic disruption. For instance, the optimal time for reproduction by seasonally breeding birds matches peak food supply with peak nestling demand. If birds schedule reproduction based on photoperiod while food supply is determined by temperature, a mismatch in timing may result in peak nestling demand occurring while food supplies are declining, with serious consequences for fitness (e.g., Thomas et al. 2001). The effect of this type of evolutionary trap on parasitism has not been explored, but mismatches between the phenology of parasites or disease vectors and birds, as well as range expansion by parasites as a result of

climate change and interactions among parasites and contaminants, could result in parasites assuming different or greater significance in altered environments.

In summary, although parasitism is a universal phenomenon in wild birds and many parasites have been observed and described, the information is still fragmentary and largely descriptive in nature. Little is known about the effect of most parasites on their hosts and almost nothing is known about interactions among the parasites that make up parasite assemblages or communities. The cost of parasites to their hosts is difficult to measure, but using energy as a currency may be a fruitful way to understand how costs are incurred, why birds must make trade-offs that influence both their exposure and resistance to parasites, and how being parasitized may affect basic life history traits including reproduction and susceptibility to predation. Parasitism can never be considered in isolation; it must always be considered in terms of the context in which it is occurring and this consideration must include the potential effects of anthropogenic changes.

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Section II: Protozoa

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2
Haemoproteus

Carter T. Atkinson

INTRODUCTION

The species of *Haemoproteus* that infect birds are vector-transmitted intraerythrocytic parasites closely allied to the true malarial parasites of vertebrates. Unlike their close relatives in the genus *Plasmodium*, they undergo asexual reproduction or merogony within tissues rather than circulating erythrocytes. They are some of the most common and widespread blood parasites of wild birds, yet their potential significance as disease agents in wild bird populations is largely unknown. They are receiving increasing attention by avian ecologists as models for testing evolutionary theories about effects of disease on host fitness and sexual selection, but these efforts have been hampered by lack of basic knowledge about their life cycles, vectors, and epizootiology.

Some species of *Haemoproteus* can be highly pathogenic and cause severe myositis in avian hosts, but well-documented cases are still rare. These include reports of disease associated with developing tissue stages in Northern Bobwhite (*Colinus virginianus*) (Gardiner et al. 1984; Cardona et al. 2002), Luzon Bleeding-heart (*Gallicolumba luzonica*) (Earle et al. 1993), Rock Pigeons (*Columba livia*) (Farmer 1965), House Sparrows (*Passer domesticus biblicus*) (Paperna and Gil 2003), Blossom-headed Parakeets (*Psittacula roseata*) (Miltgen et al. 1981), and Wild Turkeys (*Meleagris gallopavo*) (Atkinson and Forrester 1987). Some of these reports reflect abnormal host–parasite associations, where susceptible hosts were moved outside of their natural ranges and exposed to haemoproteid parasites from closely related host species.

SYNONYMS

Haemosporidiosis. Infection with avian species of *Haemoproteus* is sometimes referred to as avian malaria, particularly in the recent ecological literature, but distinctive life history characteristics clearly distinguish them from the true malarial parasites in the genus *Plasmodium* (Valkiūnas et al. 2005).

HISTORY

The species of *Haemoproteus* that infect birds were first observed on unstained blood smears along with other intraerythrocytic hemosporidian parasites by the Russian zoologist V. Ya. Danilewsky as "... clear, colorless, transparent vacuoles, variable in shape and size, in which are present several refractile glossy-black granules" (cited in Hewitt 1940). With the advent of Giemsa staining to differentiate parasites from host cells (Garnham 1966), the diversity and broad host range of these parasites became evident, but their host specificity, life cycles, and vectors were not known. Considerable confusion existed as to what comprised a species, and the taxonomy of this group has been in a continual state of flux for over a hundred years.

Major historical milestones over the past century include discovery that *Haemoproteus columbae* of pigeons and doves can be transmitted by the bite of ectoparasitic hippoboscid flies (Sergent and Sergent 1906) and the discovery that ceratopogonid flies in the genus *Culicoides* can transmit other species of *Haemoproteus* (Fallis and Wood 1957). Early recognition of the sexual stages of *Haemoproteus* (MacCallum 1898), the hippoboscid vectors (Sergent and Sergent 1906), and preerythrocytic tissue stages of *H. columbae* (Aragão 1908a) led to a number of classic investigations of the sporogonic or asexual stages of the parasite within the invertebrate vector and the preerythrocytic development of *H. columbae* within the avian host (Acton and Knowles 1914; Adie 1915, 1924; Coatney 1933). These formed an important framework during the first two decades of the twentieth century for understanding the life cycles and development of closely related haemosporidia in the genera *Plasmodium* and *Leucocytozoon*.

The vast bulk of published studies on avian species of *Haemoproteus* over the past 50 years have been surveys and taxonomic descriptions by parasitologists and disease workers. It is only in the past few years that there has been a renaissance in interest in these parasites by avian ecologists and evolutionary biologists

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because ease of sampling wild birds by noninvasive blood collection makes them potentially good models for testing evolutionary hypotheses. The role that these parasites may play as pathogens in wild birds has been speculated about since their discovery, but it is only in the past 20 years that clear evidence that they can have some measurable effects on host survival and reproduction has accumulated.

DISTRIBUTION

Avian haemoproteids have a worldwide distribution in temperate and tropical climates. This wide distribution is most likely a function of the diverse habitats occupied by their ceratopogonid and hippoboscid vectors (Greiner et al. 1975). Haemoproteids have been recorded from most parts of the globe where hippoboscid and ceratopogonid vectors occur, including remote islands in the central Pacific (Work and Raymeyer 1996; Padilla et al. 2004). The greatest diversity of species occurs in the Holarctic, Ethiopian, and Oriental zoogeographic regions, with fewer numbers of species recorded from both the Neotropical and Australian regions (Valkiūnas 2005). In both North and South America, haemoproteids tend to have a relatively uniform distribution across the continent and are virtually absent in the high arctic tundra, most likely because of the absence of suitable vectors (Greiner et al. 1975; White et al. 1978; Bennett et al. 1992).

HOST RANGE

Over 130 species of *Haemoproteus* have been reported from 72 families of birds, depending on authority (Peirce 2005; Valkiūnas 2005). Diversity in terms of number of distinct morphological forms and species is highest among the Coraciiformes (kingfishers), Piciformes (woodpeckers), and Galliformes, but the highest number of species occurs within the Passeriformes (perching birds) (Bennett 1993). Of interest is the wide disparity in occurrence of haemoproteid infections among the avian orders (Bennett 1993; Valkiūnas 2005). *Haemoproteus* has not been reported in many of the more primitive orders of birds, but is very common among the Passeriformes (Table 2.1). Some of these differences are clearly related to vector distribution

Table 2.1. Host distribution of avian haemoproteids by avian order.

Avian order	Host species	Number examined	Number infected	Percent infected
Sphenisciformes	16	16	0	0
Gaviiformes	4	3	0	0
Podicipediformes	21	7	0	0
Procellariiformes	100	30	0	0
Pelecaniformes	57	44	0	0
Tinamiformes	47	12	0	0
Apterygiformes	3	1	0	0
Struthioniformes	8	4	0	0
Ciconiiformes	124	89	40	45
Falconiformes	296	168	83	49
Strigiformes	162	66	49	74
Anseriformes	154	113	56	50
Galliformes	270	133	74	56
Gruiformes	203	87	47	54
Charadriiformes	339	154	36	23
Columbiformes	323	135	87	64
Psittaciformes	344	143	43	30
Cuculiformes	153	84	40	48
Caprimulgiformes	106	51	8	16
Apodiformes	414	75	20	27
Piciformes	402	201	60	30
Coliiformes	6	3	0	0
Coraciiformes	202	118	13	11
Trogoniformes	39	20	7	35
Passeriformes	5,211	2,409	2,047	85

Note: Data are summarized from Table 1 in Bennett (1993) and represent number of reported host species for each avian order that are infected with one or more species of *Haemoproteus*.

and abundance, with correspondingly low prevalence in seabirds and shorebirds that have limited exposure to hippoboscid or ceratopogonid flies (Mendes et al. 2005), while others may be related to differences in host resistance and immune competence (Ricklefs 1992; Sol et al. 2003).

ETIOLOGY

Members of this genus are classified as members of the phylum Apicomplexa, class Aconoidasida, order Haemospororida, family Plasmodiidae, and are defined primarily by their intraerthrocytic development, production of prominent golden-brown or black pigment granules from digestion of host hemoglobin, and absence of asexual reproduction in the circulating blood cells (Peirce 2000). Virtually all species in this genus are distinguished by morphology of the circulating gametocytes, their presumed host specificity, and by distinctive changes in host erythrocyte morphology (Figures 2.1a, b, and 2.2). Five different morphological types of gametocytes are recognized that differ in shape (round or elongated) and how far they reach around the erythrocyte nucleus (Figure 2.2).

Recent phylogenetic analyses based on mitochondrial gene sequences have placed *Haemoproteus* as a polyphyletic group within the same clade as *Plasmodium* (Perkins and Schall 2002). More recent analyses based on four genes find that the avian haemoproteids fall into two clades that are sister to *Plasmodium*: a basal group of columbiform parasites that uses hippoboscid flies as vectors and a second distinct group that is transmitted by ceratopogonid flies (Martinsen et al. 2008). These new analyses support the proposal by Bennett et al. (1965) to subdivide the genus, keeping columbiform parasites transmitted by hippoboscid flies in the genus *Haemoproteus* and moving the bulk of species that are likely transmitted by ceratopogonid flies into the genus *Parahaemoproteus* (Martinsen et al. 2008). While this distinction is currently made at the level of subgenus (Valkiūnas 2005), these recent phylogenetic studies suggest that the proposal by Bennett et al. (1965) should be revived.

The most recent taxonomic revisions of this genus are by Peirce (2005) and Valkiūnas (2005). Peirce

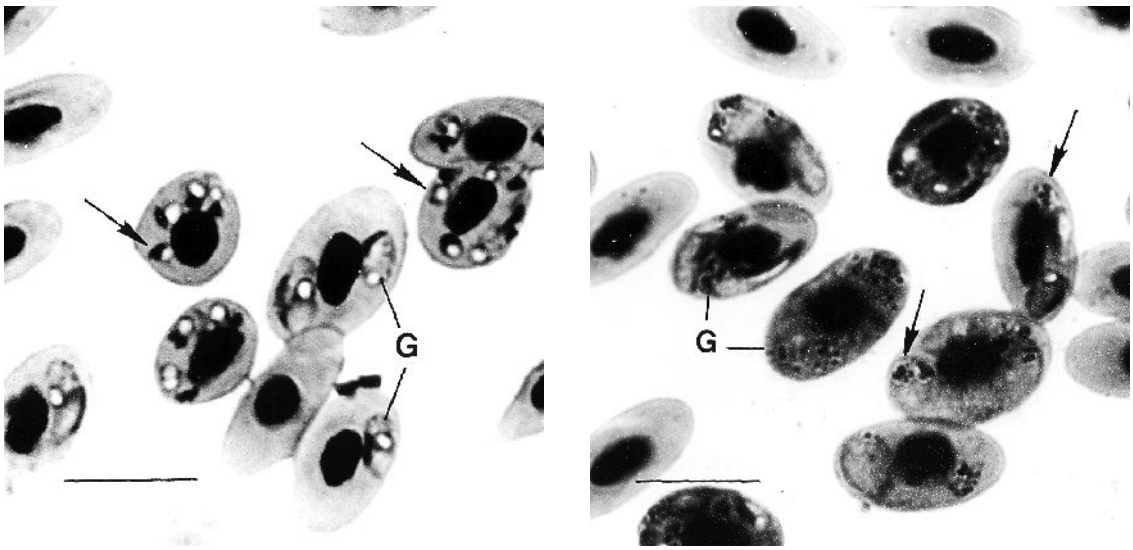


Figure 2.1. Gametocytes of *Haemoproteus meleagridis* in erythrocytes of an experimentally infected domestic turkey. (a) After release from preerythrocytic meronts, merozoites (arrows) invade erythrocytes and develop into mature gametocytes. Intraerythrocytic merozoites have a large vacuole and small nucleus. As merozoites transform into young gametocytes (G), they become elongated and sausage-shaped, eventually encircling the erythrocyte nucleus. As many as seven gametocytes may be found within individual erythrocytes in intense infections. (b) Gametocytes (G) reach maturity within 7 days after invading erythrocytes. Pigment granules (arrows) become visible only during later stages of development. Giemsa stain, bar = 10 μm. Reproduced from Atkinson (1991a), with permission of the *Journal of Vector Ecology*.

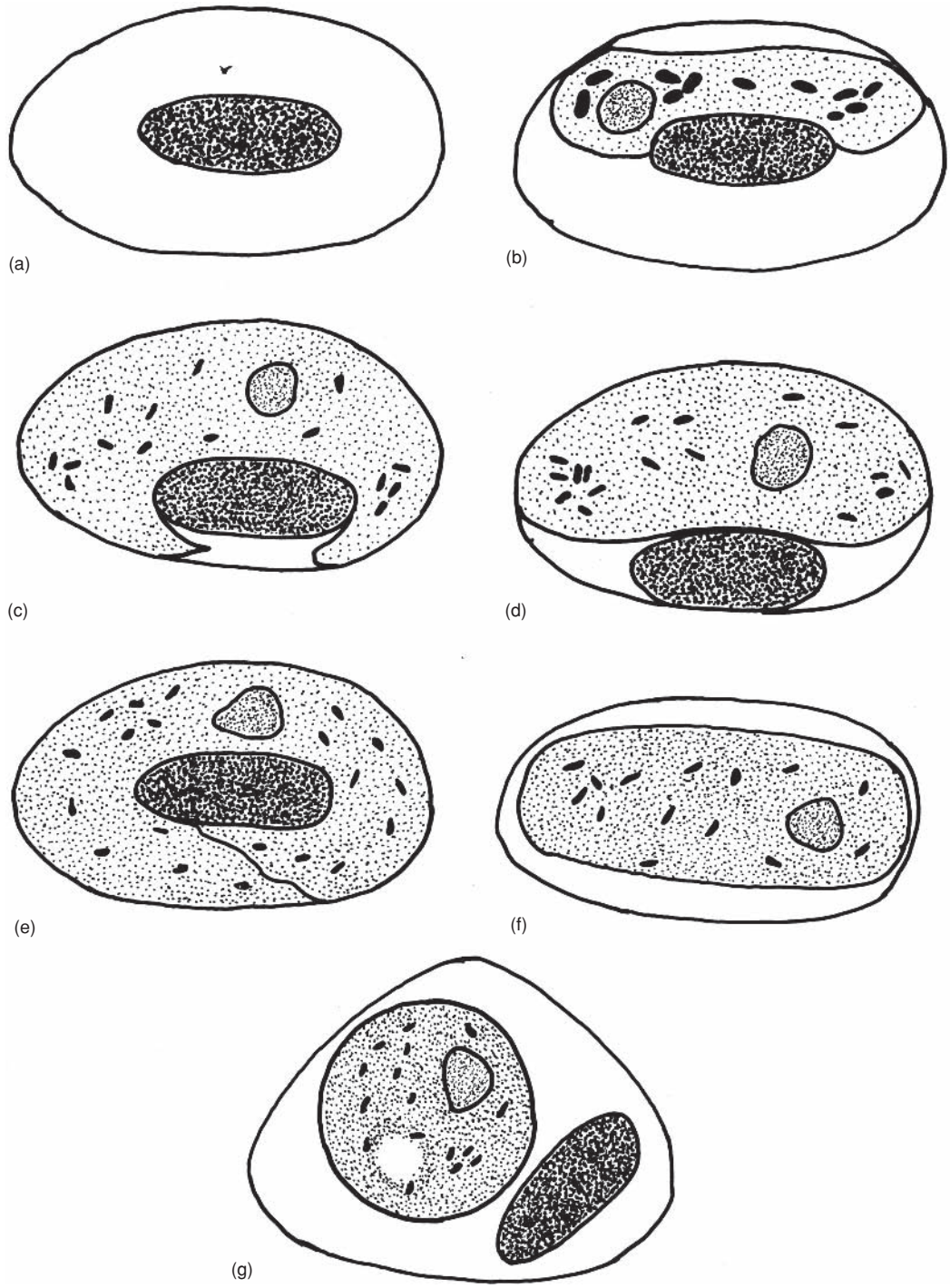


Figure 2.2. Five basic morphological forms of the mature gametocytes of avian species of *Haemoproteus*: (a) normal erythrocyte, (b) microhalteridial gametocyte, (c, d) halteridial gametocyte, (e) circumnuclear gametocyte, (f) rhabdosomal gametocyte, and (g) discosomal gametocyte. Reproduced from Bennett et al. (1988), with permission of the *Journal of Natural History* and Taylor & Francis Ltd. (http://www.informaworld.com).

(2005) lists 147 species in 72 avian families, while Valkiūnas synonymizes some species based on host range and lists 132 valid species. Early efforts by Gorden Bennett and coworkers to make some taxonomic sense out of the bewildering diversity of avian haemoproteids led to separation of morphologically similar forms by host family, based on limited experimental evidence indicating that species are specific to family (Bennett and Peirce 1988). As is the case with closely related parasites in the genus *Leucocytozoon* (Chapter 4), much of this evidence was based on small sample sizes and only a few attempts to actually infect members of different host families and orders (Valkiūnas 2005). Problems with this taxonomy have been summarized by Valkiūnas (2005), and he argues effectively that available evidence only supports specificity to level of host order.

Our understanding of the relationships between traditional morphological species and parasite lineages defined by mitochondrial and nuclear gene sequences is rapidly evolving. Recent studies of diversity of mitochondrial and nuclear genes of avian haemoproteids suggest that the true number of species may be several orders of magnitude higher with multiple parasite lineages that can coexist within the same host and with host ranges that extend well beyond single avian families (Bensch et al. 2000, 2004; Ricklefs and Fallon 2002). As a result, many traditional species defined by morphological characteristics and host family may be composed of multiple cryptic species, while many others that are defined only by occurrence in different host families will need to be synonymized. It is likely that major strides in our understanding of the taxonomy, host specificity, and evolutionary relationships among these parasites and closely related species in the genus *Plasmodium* will occur in future years as molecular data are reconciled with life history characteristics of these organisms.

EPIZOOTIOLOGY

The complex life cycle of *Haemoproteus* involves both sexual (gametogenesis and fertilization) and asexual (sporogony) reproduction in the vector and asexual reproduction (merogony) in the avian host. Proven vectors of avian haemoproteids include both ceratopogonid flies in the genus *Culicoides* and ectoparasitic hippoboscid flies (Table 2.2). The sexual cycle begins when a blood meal containing mature sexual stages of the parasite, female macrogametocytes and male microgametocytes, is taken from an infected host. The sexual stages undergo gametogenesis and fertilization in the midgut of the vector and produce a motile zygote called the ookinete. Ookinetes subsequently penetrate the midgut wall and develop under the midgut basal lamina as spherical oocysts during the asexual sporogonic cycle.

Development of the parasite in both vectors is similar, but size of oocysts, number of sporozoites produced, and duration of sporogony differs. In ceratopogonid flies, oocysts measure approximately 10 μm in diameter, while in hippoboscid flies oocysts are considerably larger and reach diameters of approximately 40 μm (Adie 1924; Fallis and Bennett 1960; Atkinson 1991b). Sporogony typically takes 4–6 days in ceratopogonid flies, eventually producing fewer than 100 sporozoites that bud from a single sporoblast. Sporogony in hippoboscid flies typically takes up to 10 days, eventually producing thousands of sporozoites that bud from multiple sporoblasts within the oocysts (Adie 1915, 1924). Oocysts subsequently rupture, releasing sporozoites into the haemocoel of the insect. These invade the salivary glands and pass through the salivary ducts during the next blood meal.

The factors that affect the ability of particular species of *Haemoproteus* to develop in a particular species of arthropod vector are poorly understood. It is clear that individual species of avian *Haemoproteus* can be transmitted by a number of different hippoboscid or ceratopogonid vectors (Table 2.2), but successful development as measured by ability to complete sporogony and produce sporozoites that can reach the salivary glands varies in each species of *Culicoides* (Atkinson 1991a; Valkiūnas et al. 2002). It is not known whether blocks in development occur in the midgut, during passage through the peritrophic membrane that surrounds the blood meal during digestion, or within the midgut epithelium.

It has never been demonstrated by experimental methods that haemoproteids transmitted by hippoboscid flies can also be transmitted by ceratopogonid flies, although complete development of *Haemoproteus lophortyx* from Northern Bobwhites in *Culicoides bottimeri*, *Stilbometopa impressa*, and *Lynchia hirsuta* suggests that this is possible (O'Roke 1930; Tarshis 1955; Mullens et al. 2006). However, the original experimental work on hippoboscid transmission of *H. lophortyx* (O'Roke 1930; Tarshis 1955) was done in facilities that were not adequately screened to prevent entry by ceratopogonid flies (Valkiūnas 2005; Table 2.2). Among other species of *Haemoproteus*, the rare occurrence of hippoboscid flies on Mourning Doves (*Zenaida macroura*) and high prevalence of *Haemoproteus sacharovi* strongly suggest that ceratopogonid flies may be involved in transmission of this parasite, but this possibility has not been investigated (Bennett and Peirce 1990). Given the results of recent phylogenetic studies (Martinsen et al. 2008), experimental tests of vector specificity of these two groups of haemoproteids should be pursued.

Table 2.2. Known species of hippoboscid (*Lynchia*, *Microlynchia*, *Ornithomyia*, *Pseudolynchia*, *Stilbometopa*) and ceratopogonid (*Culicoides*) flies that can support complete asexual sporogonic development of *Haemoproteus*.

Species	Host order	Vector	Authors
Haemoproteus nettionis	Anseriformes	*Culicoides downesi*	Fallis and Wood (1957)
Haemoproteus columbae	Columbiformes	*Pseudolynchia canariensis*	Sergent and Sergent (1906)
		Pseudolynchia brunnea	Aragão (1908b)
		Microlynchia pusilla	Aragão (1916)
Haemoproteus sacharovi[*]	Columbiformes	*Pseudolynchia canariensis*	Huff (1932)
Haemoproteus maccallumi	Columbiformes	*Pseudolynchia canariensis*	Huff (1932)
Haemoproteus turtur[†]	Columbiformes	*Pseudolynchia canariensis*	Rashdan (1998)
Haemoproteus palumbis	Columbiformes	*Ornithomyia aviculria*	Baker (1963, 1966)
Haemoproteus lophortyx[‡]	Galliformes	*Stilbometopa impressa*	O'Roke (1930)
		Lynchia hirsuta	Tarshis (1955)
		Culicoides bottimeri	Mullens et al. (2006)
Haemoproteus mansoni	Galliformes	*Culicoides sphagnumensis*	Fallis and Bennett (1960)
Haemoproteus meleagridis[§]	Galliformes	*Culicoides edeni*	Atkinson et al. (1983)
		Culicoides hinmani	Atkinson et al. (1983)
		Culicoides arboricola	Atkinson et al. (1983)
		Culicoides haematopotus	Atkinson (1988)
		Culicoides knowltoni	Atkinson (1988)
Haemoproteus handai	Psittaciformes	*Culicoides nubeculosus*	Miltgen et al. (1981)
Haemoproteus velans	Passeriformes	*Culicoides stilobezziodes*	Khan and Fallis (1971)
		Culicoides sphagnumensis	Khan and Fallis (1971)
Haemoproteus fringillae	Passeriformes	*Culicoides crepuscularis*	Fallis and Bennett (1961)
		Culicoides stilobezziodes	Fallis and Bennett (1961)
		Culicoides sphagnumensis	Fallis and Bennett (1961)
		Culicoides impunctatus	Valkiūnas (1997)
Haemoproteus danilewskii	Passeriformes	*Culicoides crepuscularis*	Bennett and Fallis (1960)
		Culicoides stilobezziodes	Bennett and Fallis (1960)
		Culicoides sphagnumensis	Fallis and Bennett (1961)
		Culicoides edeni	Garvin and Greiner (2003a)
		Culicoides knowltoni	Garvin and Greiner (2003a)
		Culicoides arboricola	Garvin and Greiner (2003a)
Haemoproteus balmorali	Passeriformes	*Culicoides impunctatus*	Valkiūnas et al. (2002)
Haemoproteus dolniki	Passeriformes	*Culicoides impunctatus*	Valkiūnas et al. (2002)
Haemoproteus tartakovskyi	Passeriformes	*Culicoides impunctatus*	Valkiūnas et al. (2002)
Haemoproteus belopolskyi	Passeriformes	*Culicoides impunctatus*	Valkiūnas and Iezhova (2004)
Haemoproteus lanii	Passeriformes	*Culicoides impunctatus*	Valkiūnas and Iezhova (2004)

[*]Circumstantial evidence indicates that one or more species of *Culicoides* may also be involved in natural transmission of *Haemoproteus sacharovi* (Bennett and Peirce 1990).

[†]*Haemoproteus turtur* is recognized as distinct from *Haemoproteus columbae* by Valkiūnas (2005), but considered a synonym of *H. columbae* by Peirce (2005).

[‡]Tarshis (1955) was unable to demonstrate sporozoites of *Haemoproteus lophortyx* in *Stilbometopa impressa* and *Lynchia hirsuta* in spite of repeated attempts to infect them in the laboratory, but did successfully transmit *H. lophortyx* when flies were allowed to bite uninfected birds. Valkiūnas (2005) suggests that experimental cages may not have been impervious to ceratopogonid flies, based on unusually long prepatent periods for experimental infections and use of screened outdoor aviaries. O'Roke (1930), however, describes oocysts and sporozoites in *L. hirsuta* that fed on infected quail. Given the recent finding that *Culicoides bottimeri* is a likely natural vector, experiments with both *S. impressa* and *L. hirsuta* should be repeated.

[§]*Haemoproteus meleagridis* is considered a junior synonym of *Haemoproteus canachites* by Valkiūnas (2005).

Complete life cycles are known for only a handful of avian haemoproteids and we still have only a rudimentary knowledge about the preerythrocytic development of these parasites. *Haemoproteus columbae* from pigeons and doves, *Haemoproteus meleagridis* from Wild Turkeys, and *Haemoproteus danilewskii* from Blue Jays (*Cyanocitta cristata*) have received the most detailed experimental study.

Endogenous development of all three of these species begins when infective sporozoites are inoculated at the site where the vector takes a blood meal. These sporozoites develop within cells of the lymphoid–macrophage system, capillary endothelium, and/or myofibroblasts, undergoing one or more generations of asexual reproduction or merogony before penetrating circulating erythrocytes (Mohammed 1965; Atkinson et al. 1986). Here they develop as gametocytes, becoming infective to vectors within 7–10 days after invading the blood cells.

At least two generations of preerythrocytic merogony occur in skeletal and cardiac muscle of domestic turkeys experimentally infected with *H. meleagridis*. The first begins when infective sporozoites invade capillary endothelial cells and myofibroblasts and develop into thin-walled round or oval meronts measuring 12–20 μm in diameter. These produce long, slender merozoites between 5 and 8 days postinfection that subsequently invade new capillary endothelial cells in skeletal and cardiac muscle and develop as second-generation meronts. Early second-generation meronts are 5–8 μm in diameter and 28 μm in length. These grow rapidly to form large, fusiform, thick-walled megalomeronts measuring up to 500 μm in length (Figure 2.3). Megalomeronts reach maturity at 17 days postinfection and rupture to release small spherical merozoites that invade erythrocytes and develop into gametocytes (Figure 2.1a). Mature gametocytes that completely encircle the host erythrocyte nucleus develop within 7–10 days after red blood cells are invaded (Figure 2.1b). Parasitemias reach their peak intensity in the peripheral circulation at approximately 21 days postinfection and fall rapidly within 7 days to low intensities. A second, smaller peak in parasitemia may occur at approximately 35 days postinfection (Atkinson et al. 1986). The number of generations of preerythrocytic merogony has not been defined for *H. columbae* and *H. danilewskii*, but it is likely that they also undergo two or more cycles of asexual reproduction before invading erythrocytes. In these two species, the parasites invade capillary endothelial cells of the lungs where they undergo preerythrocytic development to form thin-walled, oval or branching meronts that radiate along pulmonary capillaries (Mohammed 1965; Garnham 1966; Garvin et al. 2003a; Valkiūnas 2005). Similar, thin-walled branching meronts have been reported in a variety of other naturally infected avian hosts (Figure 2.4). Thick-walled megalomeronts have been reported in Luzon Bleeding-hearts (*Gallicolumba luzonica*) infected with *H. columbae* (Earle et al. 1993), but their relationship to the thin-walled, branching meronts of *H. columbae* described from Rock Pigeons is unclear and possibly related to presence of a mixed infection with another unidentified parasite (Peirce et al. 2004).

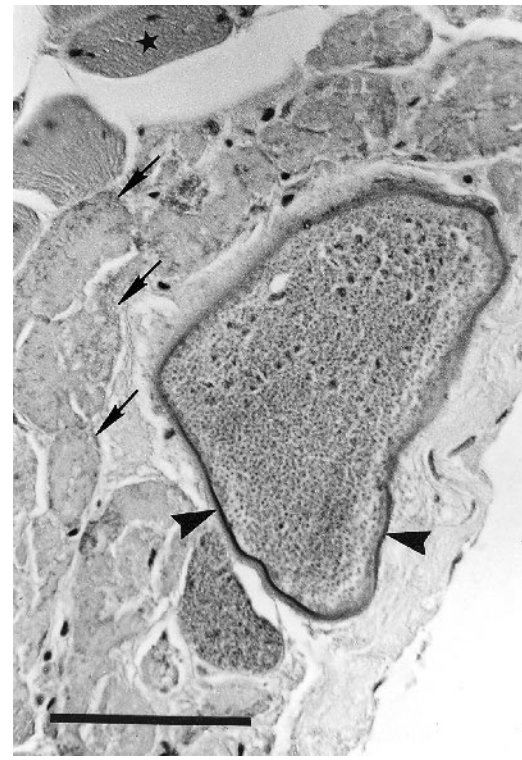


Figure 2.3. Megalomeront of *Haemoproteus meleagridis* from the pectoral muscle of a naturally infected Wild Turkey (*Meleagris gallopavo*). The megalomeront is surrounded by a thick, hyaline wall (arrowheads) and is packed with spherical merozoites. Muscle fibers surrounding the megalomeront are swollen, pale, and hyaline and contain scattered basophilic granules (arrows). Note adjacent normal tissue (*). Hematoxylin and eosin, bar = 50 μm. Reproduced from Atkinson and Forrester (1987), with permission of the *Journal of Wildlife Diseases*.

Among haemoproteids transmitted by *Culicoides*, prepatent periods vary from 11 to 12 days for

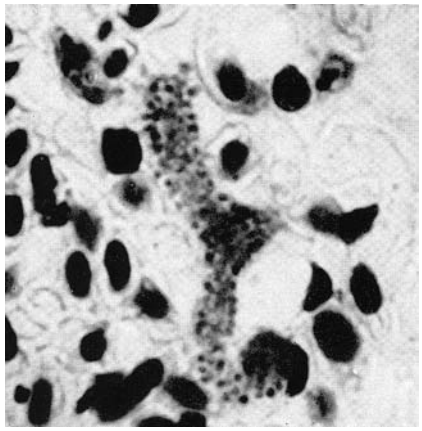


Figure 2.4. Thin-walled, irregularly shaped meront in lung tissue from a White-throated Sparrow (*Zonotrichia albicollis*) infected naturally with *Haemoproteus coatneyi*. Reproduced from Khan and Fallis (1969), with permission of the *Canadian Journal of Zoology*.

Haemoproteus belopolskyi of Blackcaps (*Sylvia atricapilla*) (Valkiūnas and Iezhova 2004), from 11 to 14 days for *Haemoproteus velans* of woodpeckers (Khan and Fallis 1971), 14 days for *Haemoproteus mansoni* of Ruffed Grouse (*Bonasa umbellus*) (Fallis and Bennett 1960), approximately 16 days for *Haemoproteus nettionis* of ducks (Fallis and Wood 1957), 14 days for *H. danilewskii* of Blue Jays (Garvin et al. 2003a), and 17 days for *H. meleagridis* of Wild Turkeys (Atkinson et al. 1986).

Among haemoproteids transmitted by hippoboscid flies, the prepatent period ranges from 17 to 37 days for *H. columbae* of Rock Pigeons and is about 14 days for *Haemoproteus palumbis* of Common Wood-Pigeons (*Columba palumbus*) (Baker 1966). Like the species of *Haemoproteus* that are transmitted by *Culicoides*, merozoites in circulating erythrocytes develop to mature microgametocytes and macrogametocytes that encircle the erythrocyte nucleus within approximately 5–10 days. Gametocyte numbers peak in the peripheral circulation approximately 10–20 days after first appearing in the circulation and then decline in numbers.

Among species of *Haemoproteus* transmitted by ceratopogonid flies, transmission is seasonal and limited to the spring and summer months in more temperate parts of their range (Bennett and Fallis 1960), but can occur throughout the year in subtropical habitats in Florida and most likely other parts of the world where suitable vectors are present year round (Atkinson et al. 1988a). In temperate North America, by contrast, transmission of *H. columbae* by hippoboscid flies is seasonal and closely correlated with changes in vector populations, generally increasing in the fall and winter months and then declining as vector density decreases (Klei and DeGiusti 1975). More limited data from tropical and subtropical parts of the world where populations of hippoboscid flies remain more constant indicate that high rates of transmission and high prevalences of infection can be maintained throughout the year (Ayala et al. 1977; Sol et al. 2000).

The role that host migratory behavior plays in cycles of transmission of avian haemoproteids is significant because of the potential of long distance migrants to disperse parasites both within and between continental landmasses (Laird 1960; Waldenström et al. 2002; Hasselquist et al. 2007). Limited information from the Nearctic and Palearctic indicates that some species of avian haemoproteids are transmitted on the breeding grounds, while others are transmitted in wintering areas in the tropics and subtropics, while others may be transmitted in both locations. This suggests that transmission may be linked in some cases to particular geographic locations or vector–parasite associations (Valkiūnas 1993; Valkiūnas and Iezhova 2001; Waldenström et al. 2002; Garvin et al. 2003b, 2004; Hasselquist et al. 2007; Hellgren et al. 2007b).

Within individual hosts, intensity of infection varies after the initial acute phase and appears to be influenced by the complex interplay of host immunity, seasonal changes in photoperiod, and hormonal changes associated with reproduction. In temperate climates, a seasonal increase in intensity, termed the spring relapse, coincides with the breeding season when populations of blood-sucking insects typically increase and recently fledged susceptible birds are increasing in the population (Atkinson and van Riper 1991; Valkiūnas et al. 2004). Relapse of chronic *Plasmodium* infections can be triggered by corticosterone (Applegate and Beaudoin 1970) and other experimental evidence suggests that increases in photoperiod and subsequent physiological changes in levels of hormones such as melatonin that regulate circadian rhythms may also be important stimuli for initiating relapses among species of *Haemoproteus* (Valkiūnas et al. 2004).

Other factors affecting intensity include stress-mediated changes in the immune system that are associated with reproductive effort (Siikamäki et al. 1997), food availability (Appleby et al. 1999), concomitant infection with other parasites (Cox 1987), and exposure to predators (Navarro et al. 2004).

Attempts to identify broad patterns and relationships in the prevalence of avian haemoproteids have met with variable success because of the diversity of this group of parasites. Much may depend on how prevalence data

are lumped, with positive relationships more evident where other species of hematozoans are included in the analyses. A wide variety of both intrinsic and extrinsic factors have been identified, including host specificity of the parasites (Bennett 1993), immune competency (Ricklefs 1992), host genotype (Bonneaud et al. 2006), host age and sex (Davidar and Morton 1993; Powers et al. 1994; McCurdy et al. 1998), geographic range of host species (Tella et al. 1999), whether or not host species are migratory (Bennett and Fallis 1960; Peirce and Mead 1978; Figuerola and Green 2000; Smith et al. 2004), plumage coloration (Yezerinac and Weatherhead 1995), and host foraging or nesting behavior (Greiner et al. 1975; Garvin and Remsen 1997).

Extrinsic factors such as habitat, geographical region, and season are critically important because they can influence the distribution and abundance of vectors (Weatherhead and Bennett 1991, 1992; Sol et al. 2000; Mendes et al. 2005). Prevalence in the same host species can vary significantly across both large and small landscapes (Atkinson et al. 1988a; Sol et al. 2000; Wood et al. 2007), suggesting that vector distribution and abundance may be the most important determinant of prevalence. However, other factors may cause seasonal changes in parasite prevalence, including winter mortality in infected birds, and new infections associated with emergence of insect vectors and transmission to uninfected juvenile birds.

CLINICAL SIGNS

Clinical signs are usually not evident in low-intensity infections, but can become evident during acute phase infections when erythrocytic parasitemias and numbers of tissue meronts reach high intensities. Domestic turkey poults with experimental infections of *H. meleagridis* are lame in one or both legs and have lower weights and growth rates than do uninfected controls (Atkinson et al. 1988b). Similarly, Northern Bobwhites with natural infections of *H. lophortyx* are reluctant to move, have a ruffled, depressed appearance, and exhibit neurological signs such as loss of balance and difficulty walking (Cardona et al. 2002). Signs of infection in Rock Pigeons include weakness, anemia, and anorexia (Acton and Knowles 1914; Coatney 1933).

Elevation in numbers of circulating lymphocytes, heterophils, basophils, eosinophils, and monocyte numbers has been observed in both natural and experimental infections with *Haemoproteus*, and it is likely that these increases represent a cell-mediated response to both erythrocytic and preerythrocytic stages of the parasite, particularly as the latter mature and rupture to release merozoites that invade erythrocytes (Ots and Hõrak 1998; Garvin et al. 2003a). No significant overall difference in plasma protein concentration, hemoglobin concentration, packed cell volume, or weight was observed between infected and uninfected Blue Jays (Garvin et al. 2003a). Other studies have also failed to report significant anemia in infections with *Haemoproteus*, including *H. meleagridis* in experimentally infected domestic turkeys (Atkinson et al. 1988b) and *Haemoproteus* spp. in Great Tits (*Parus major*) (Ots and Hõrak 1998). By contrast, O'Roke (1930) and Cardona et al. (2002) detected severe anemia in California Quail (*Callipepla californica*) and captive Northern Bobwhites with natural infections of *H. lophortyx*. Severe regenerative anemia with marked polychromasia has also been reported in Snowy Owls (*Bubo scandiacus*) infected with *Haemoproteus noctuae* (Evans and Otter 1998) and in Snowy Owls, Tawny Owls (*Strix aluco*), and Great Horned Owls (*Bubo virginianus*) infected with *Haemoproteus syrnii* (Mutlow and Forbes 1999). Mechanisms responsible for development of anemia in these host species are not known, although there may be a fine balance between removal of parasitized erythrocytes by the spleen and their replacement with immature red blood cells (Atkinson et al. 1988b). When an infected host lacks the physiological resources to replace infected blood cells because of stress associated with reproduction or limited food resources, anemia may result.

PATHOGENESIS AND PATHOLOGY

Virtually nothing is known about the pathogenesis of haemoproteid infections because so little is known about their development within natural and experimental hosts. Few host responses have been associated with development of thin-walled branching meronts that frequently occur in lung tissue (Mohammed 1965; Baker 1966; Garnham 1966) (Table 2.3). In one of the most detailed studies to date, no host responses were associated with preerythrocytic meronts at day 31 postinfection in Blue Jays infected experimentally with *H. danilewskii*. However by day 57 postinfection, juvenile jays had lesions in liver, spleen, and lung tissue. These included periportal and random individual cell necrosis in liver and lymphocytic infiltrates and epithelial hyperplasia around tertiary bronchi in lung tissue. Histological changes in splenic tissue included hyperplasia of white pulp arteriolar endothelium, random necrosis of lymphocytes, and increases in the number of macrophages, plasma cells, and Mott cells (Garvin et al. 2003a). The authors suggested that the lesions developed only after meronts matured and ruptured.

Severe myositis has been reported in association with thick-walled megalomeronts in a variety of avian species (Table 2.3). These lesions are associated with intact and ruptured megalomeronts and are grossly visible as white flecks or dark hemorrhagic streaks

Table 2.3. Preerythrocytic meronts and host responses reported from wild (W), domestic (D), captive (C), or experimentally infected (E) avian hosts.

Host species	Host order	Parasite	Status	Tissue	Pathology	Citations
Thin-walled oval or branching meronts						
Wood Duck (*Aix sponsa*)	Anseriformes	*Haemoproteus nettionis*	E	Lungs, heart, spleen	No	Sibley and Werner (1984)
Black Crowned-Crane (*Balearica pavonina*)	Gruiformes	*Haemoproteus balearicae*	W	Lung	No	Peirce (1973)
Rock Pigeon (*Columba livia*)	Columbiformes	*Haemoproteus columbae*	W, E, D	Lungs, rarely liver and spleen	None reported or tissue displacement, blockage of vessels	Aragão (1908b), Mohammed (1965), and Peirce et al. (2004)
Common Wood-Pigeon (*Columba palumbus*)	Columbiformes	*Haemoproteus palumbis*	W	Lungs, heart	No	Baker (1966)
Mourning Dove (*Zenaida macroura*)	Columbiformes	*Haemoproteus sacharovi*	W	Lung	No	Greiner (1971)
Mourning Dove (*Zenaida macroura*)	Columbiformes	*Haemoproteus maccallumi*	W	Lung	No	Greiner (1971)
Blue Jay (*Cyanocitta cristata*)	Passeriformes	*Haemoproteus danilewskii*	E	Liver, spleen, lung	Minor inflammation	Garvin et al. (2003a)
House Sparrow (*Passer domesticus*)	Passeriformes	*Haemoproteus passeris*	W	Lungs, liver	No	Peirce (1976)
Noisy Miner (*Manorina melanocephala*)	Passeriformes	*Haemoproteus ptilotis**	W	Heart and spleen	Tissue displacement, inflammation	Peirce et al. (2004)
Noisy Friarbird (*Philemon corniculatus*)	Passeriformes	*Haemoproteus ptilotis**	W	Liver	No	Peirce et al. (2004)
European Robin (*Erithacus rubecula*)	Passeriformes	*Haemoproteus attenuatus*	W	Lungs, spleen	No	Valkiūnas (2005)
White-throated Sparrow (*Zonotrichia albicollis*)	Passeriformes	*Haemoproteus coatneyi*	W	Lungs, heart, liver, spleen, cecum, kidneys	No	Khan and Fallis (1969)

Thick-walled meglomeronts						
Lesser Flamingo (*Phoenicopterus minor*)	Phoenicopteriformes	*Haemoproteus* sp.†	C	Liver	Hepatic necrosis, hemorrhage, inflammation	Ferrell et al. (2007)
Muscovy Duck (*Cairina moschata*)	Anseriformes	Undetermined*	D	Heart, lungs, liver, kidneys, spleen	Inflammation	Commichau and Jonas (1977) and Kŭcera et al. (1982)
Northern Bobwhite (*Colinus virginianus*)	Galliformes	*Haemoproteus lophortyx*	C	Skeletal muscle	Myopathy	Cardona et al. (2002)
Domestic Turkey, Wild Turkey (*Meleagris gallopavo*)	Galliformes	*Haemoproteus meleagridis*	W, E	Cardiac and skeletal muscle	Myopathy	Atkinson and Forrester (1987) and Atkinson et al. (1988b)
Domestic chicken, Red Jungle Fowl (*Gallus gallus*)	Galliformes	*Arthrocystis galli**	D	Skeletal and cardiac muscle	Myopathy	Levine et al. (1970) and Opitz et al. (1982)
Luzon Bleeding-heart (*Gallicolumba luzonica*)	Columbiformes	*Haemoproteus columbae*	C	Cardiac and skeletal muscle, gizzard, proventriculus	Myopathy	Earle et al. (1993)
Mourning Dove (*Zenaida macroura*)	Columbiformes	*Haemoproteus sacharovi*	W	Gizzard	No	Farmer (1965)
Blossom-headed Parakeet (*Psittacula roseata*)	Psittaciformes	*Haemoproteus handai*	C	Cardiac and skeletal muscle	Myopathy	Miltgen et al. (1981)
Monk Parakeet (*Myiopsitta monachus*)	Psittaciformes	Undetermined*	C	Skeletal muscle	Myopathy	Borst and Zwart (1972)
Parakeet (species not reported)	Psittaciformes	Undetermined*	C	Heart, gizzard	Hemorrhage	Fowler and Forbes (1972) and Walker and Garnham (1972)

(*continues*)

Table 2.3. (*Continued*)

Host species	Host order	Parasite	Status	Tissue	Pathology	Citations
Snowy Owl (*Bubo scandiacus*)	Strigiformes	*Haemoproteus syrnii*	W	Skeletal muscle	No	Mutlow and Forbes (1999)
Israeli House Sparrow (*Passer domesticus biblicus*)	Passeriformes	*Haemoproteus passeris*	W	Liver, lungs, kidney	Inflammation	Garnham (1966) and Paperna and Gil (2003)
Java Sparrow (*Padda oryzivora*)	Passeriformes	Undetermined*	W	Kidney	No	Garnham (1966)
Sacred Kingfisher (*Todiramphus sanctus*)	Passeriformes	*Haemoproteus halcyonis*	W	Skeletal muscle	No	Peirce et al. (2004)
Pied Currawong (*Strepera graculina*)	Passeriformes	Undetermined*	W	Heart, skeletal muscle, gizzard	Myopathy	Lederer et al. (2002)
Green Jay (*Cyanocorax yncas*)	Passeriformes	*Haemoproteus* sp.†	C	Liver	Hepatic necrosis, hemorrhage, inflammation	Ferrell et al. (2007)
Montezuma Oropendola (*Gymnostinops montezuma*)	Passeriformes	*Haemoproteus* sp.†	C	Liver	Hepatic necrosis, hemorrhage, inflammation	Ferrell et al. (2007)

Note: Two primary types of preerythrocytic meronts have been reported: thin-walled, oval or branching forms that are associated with limited host reaction; and thick-walled, round or fusiform forms that occur in skeletal, gizzard, and cardiac muscle, as well as liver, spleen, and lung tissue. The most definitive associations are in birds with experimental infections with *Haemoproteus nettionis*, *Haemoproteus columbae*, *Haemoproteus meleagridis*, and *Haemoproteus danilewskii*. Remaining examples should be viewed with caution since hosts may have been infected with more than one parasite and often did not have circulating gametocytes. Table includes hosts infected with megalomeronts of undetermined or questionable taxonomic status that are suspected to belong to species of *Haemoproteus*.

*No parasitemia observed, possibly *Leucocytozoon* or other undetermined protozoan.

†Identity determined by PCR amplification and sequencing of parasite cytochrome *b* gene.

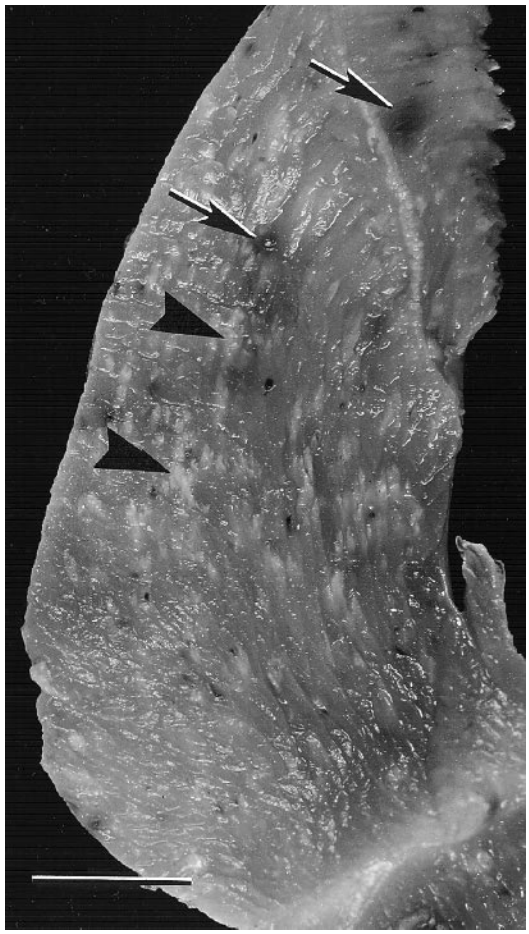


Figure 2.5. Formalin-fixed pectoral muscle from a domestic turkey with an experimental infection with *Haemoproteus meleagridis*. Note the scattered white streaks (arrowheads) and darkened hemorrhagic areas (arrows) that correspond to megalomeronts in histological sections. Hematoxylin and eosin, bar = 0.5 cm. Reproduced from Atkinson et al. (1988b), with permission of the *Journal of Parasitology*.

in skeletal and cardiac muscle. The lesions superficially resemble those from infections with *Sarcocystis* (Figure 2.5). Microscopically, megalomeronts are surrounded by mixed inflammatory infiltrates composed of macrophages, heterophils, giant cells, and red blood cells, and adjacent muscle fibers are often necrotic and calcified (Miltgen et al. 1981; Atkinson et al. 1988b; Cardona et al. 2002) (Figures 2.6 and 2.7). Other lesions include extensive deposition of parasite pigment in tissue macrophages of the liver and spleen and enlargement of these organs (Atkinson et al. 1986, 1988b; Atkinson and Forrester 1987).

Megalomeronts with associated muscle pathology have been reported in a variety of other naturally infected avian hosts, but their role in life cycles of specific species of *Haemoproteus* is difficult to determine without experimental studies (Peirce et al. 2004; Table 2.3).

DIAGNOSIS

The gold standard for diagnosis of *Haemoproteus* is a Giemsa-stained thin blood smear where it is possible to demonstrate the presence of erythrocytic gametocytes with prominent golden-brown or black pigment granules and absence of erythrocytic meronts that are diagnostic for *Plasmodium* spp. Individual species are traditionally defined by morphology of intraerythrocytic gametocytes (Figure 2.2) and host specificity, but this will likely undergo extensive revision in future years. Molecular methods are beginning to be applied to differentiation of genera and identification of unique parasite lineages. Their high sensitivity make them valuable for identifying birds with very low intensity infections, but these methods have not been refined to the point where they can be used to distinguish individual species. Recent studies, though, suggest that this may eventually be feasible (Hellgren et al. 2007a; Valkiūnas et al. 2007).

Species of *Haemoproteus* may be difficult to distinguish from avian species of *Plasmodium*, particularly in chronic infections where number of circulating gametocytes is low and where it may be difficult to determine whether the intracellular meronts characteristic of *Plasmodium* are present or absent. Several recent sets of primers designed to amplify portions of parasite mitochondrial genome can distinguish *Haemoproteus* and *Plasmodium* from *Leucocytozoon* (Hellgren et al. 2004) or all three genera from each other following restriction digests of polymerase chain reaction (PCR) products (Beadell and Fleischer 2005). However, sequencing of PCR products is necessary for identifying individual parasite lineages and determining phylogenetic relationships.

The morphology of tissue stages is difficult to use alone for making accurate diagnosis of infection with *Haemoproteus*. The thin-walled oval or branching meronts that are characteristic of some species of columbiform haemoproteids are similar in morphology to tissue stages of both *Leucocytozoon* and *Plasmodium*. Megalomeronts of *Haemoproteus* may be difficult to distinguish from those of *Leucocytozoon*. A variety of megalomeronts have been reported as aberrant *Leucocytozoon* infections (Levine et al. 1970; Borst and Zwart 1972; Fowler and Forbes 1972; Walker and

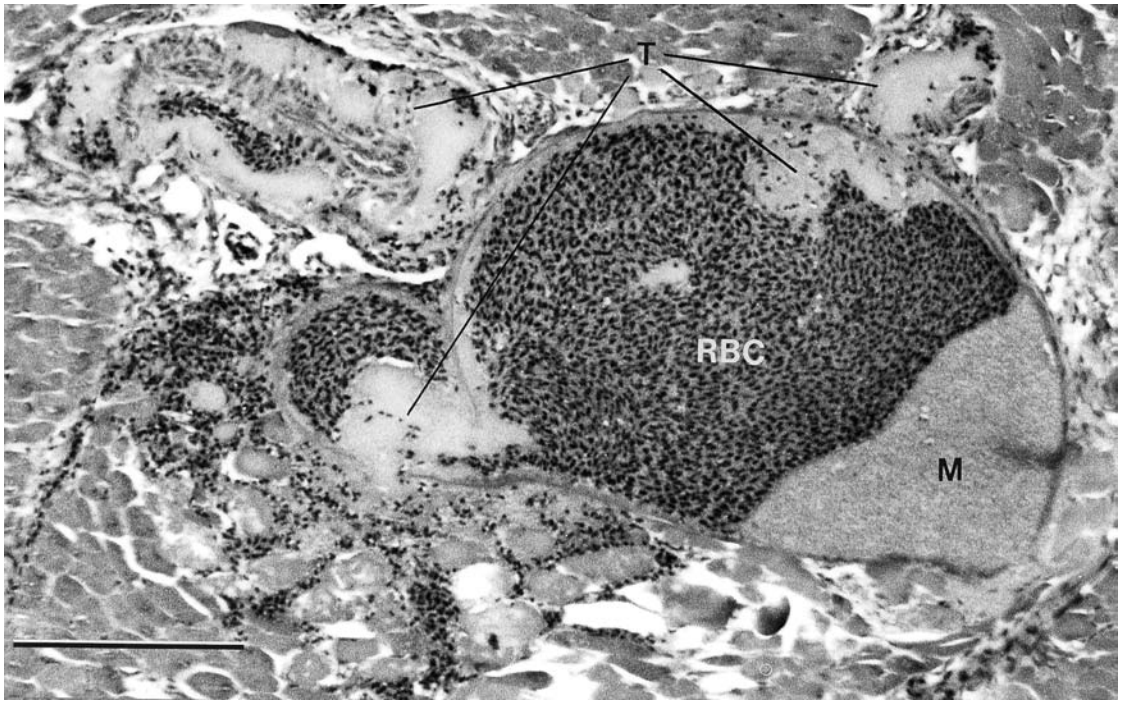


Figure 2.6. Ruptured megalomeront from pectoral muscle of a domestic turkey with an experimental infection with *Haemoproteus meleagridis*. Hemorrhagic megalomeront (M) is surrounded and partially filled by red blood cells (RBCs). Thrombi (T) with embedded RBCs are adjacent to or within the megalomeront. Hematoxylin and eosin, bar = 100 μm. Reproduced from Atkinson et al. (1988b), with permission of the *Journal of Parasitology*.

Garnham 1972; Hartley et al. 1981; Simpson 1991; Pennycott et al. 2006) or possible *Besnoitia* infections (Bennett et al. 1993; Peirce et al. 2004), but erythrocytic parasites were absent and it is possible that some of these reports may prove to be tissue stages of *Haemoproteus*. The difficulties in making diagnoses from wild birds that may be infected with multiple species of haemosporidians have been discussed by Lederer et al. (2002) who pointed out that accurate association between megalomeronts and infection with *Haemoproteus* or *Leucocytozoon* in wild birds requires experimental studies. The recent use of molecular methods for diagnosis may help resolve some of these problems. For example, hepatic megalomeronts in three species of captive birds in a zoo collection in Texas were recently shown to be associated with infection with an undetermined species of *Haemoproteus* by PCR amplification of a portion of the parasite mitochondrial cytochrome *b* gene (Ferrell et al. 2007).

Haemoproteus appears to be antigenically distinct from *Plasmodium* and crude antigen extracts have been used to develop an ELISA test for *H. columbae* in Rock Pigeons (Graczyk et al. 1994). The specificity and sensitivity of this serological test with other avian haemoproteids are not known, but they may prove useful for making genus level diagnoses in birds with low-intensity infections.

IMMUNITY

Virtually nothing is known about immune mechanisms in haemoproteid infections. Spontaneous recovery from infections with *H. columbae* has been reported in Rock Pigeons, with no immunity conferred to second infection (Sergent and Béquet 1914; Ahmed and Mohammed 1978). In most cases birds probably remain infected for long periods of time and have spontaneous relapses that may decrease in frequency, eventually leading to recovery (Coatney 1933; Ahmed and Mohammed 1978). Experimental evidence for this is very limited, however, and restricted to *H. columbae* of Rock Pigeons. Limited experimental data indicate that birds with chronic infections have concomitant immunity where a persistent chronic infection stimulates immunity to reinfection with homologous parasites of the same species (Coatney 1933; Ahmed and Mohammed

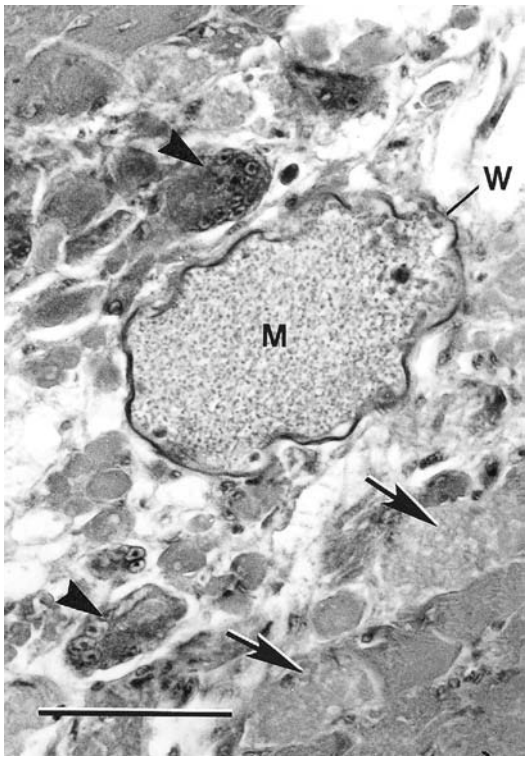


Figure 2.7. Intact megalomeront from pectoral muscle of a domestic turkey with an experimental infection with *Haemoproteus meleagridis*. Megalomeront (M) is surrounded by giant cells (arrowheads) and hyaline and necrotic muscle fibers (arrows). Note thick hyaline wall (W) surrounding the megalomeront. Hematoxylin and eosin, bar = 50 μm. Reproduced from Atkinson et al. (1988b), with permission of the *Journal of Parasitology*.

1978). The relapses associated with chronic infections most likely originate from persistent tissue stages, but this has not been proven by experimental studies.

PUBLIC HEALTH CONCERNS

Infected birds pose no health hazards to humans.

DOMESTICATED ANIMAL HEALTH CONCERNS

Haemoproteus meleagridis of Wild Turkeys is a potential threat to domestic turkey production, but in practice this has never materialized—possibly because of separation of most commercial poultry facilities from habitats where Wild Turkeys range.

There are multiple reports of pathogenic infections of *Haemoproteus* in pigeons and doves. These are usually associated with high parasitemias (Coatney 1933) and the occurrence of megalomeronts (Farmer 1965; Earle et al. 1993), but most individuals appear to be able to tolerate very high parasitemias with no clinical signs of infection.

Major outbreaks of infection with *H. lophortyx* have been reported in Northern Bobwhite raised in California where the natural reservoir host is California Quail. Outbreaks occur during warm weather when ceratopogonid populations increase (Cardona et al. 2002). Similarly, there have been a substantial number of reports of lethal *Leucocytozoon*-like infections affecting captive birds, particularly parakeets, that may actually be caused by species of *Haemoproteus* (Fowler and Forbes 1972; Smith 1972; Walker and Garnham 1972; Simpson 1991; Pennycott et al. 2006; Ferrell et al. 2007). In all these instances, captive birds were introduced to areas outside of their natural range.

WILDLIFE POPULATION IMPACTS

The effects of individual *Haemoproteus* infections are difficult to discern in wild hosts. The vast majority of studies are correlational and the avian hosts under investigation are frequently infected with other hematozoan parasites, including *Leucocytozoon*, *Plasmodium*, and *Trypanosoma*. In a thorough review of over 5,000 papers on avian blood parasites, Bennett et al. (1993) found that only about 4% reported mortality or pathogenicity in birds, with most dealing with domestic birds or birds in zoological collections. Mortality associated with *Haemoproteus* and other blood parasites in wild birds probably occurs more frequently than reported because sick individuals may be difficult to find for sampling or recover from the wild for necropsy. Epizootics are often hard to document for small passerines in areas where carcasses are rapidly scavenged (Bennett et al. 1993).

Since the life cycle of *Haemoproteus* requires a vector, experimental manipulations of naturally acquired infections in the wild are difficult. One approach that has been successful is use of a single subcutaneous dose of primaquine to control *H. majoris* and *Leucocytozoon majoris* in naturally infected Eurasian Blue Tits (*Cyanistes caeruleus*) (Merino et al. 2000). The treated group had higher fledging success and lower nestling mortality, but the relative contributions of *Haemoproteus* and *Leucocytozoon* to decreased fledging success were not determined.

Some studies have reported reduced survival in birds infected with *Haemoproteus* (Nordling et al. 1998;

Dawson and Bortolotti 2000; Hõrak et al 2001; Sol et al. 2003) and negative effects on indices of immunity, condition, and reproductive success of their hosts (Allander and Bennett 1995; Ots and Hõrak 1998; Merino et al. 2000; Sanz et al. 2001). While some studies suggest that these changes may be reflected in plumage coloration (Hõrak et al. 2001), others have found limited association (Kirkpatrick et al. 1991). Effects of infection with *Haemoproteus* can also have indirect effects on host reproduction. Female Eurasian Kestrels (*Falco tinnunculus*) with *Haemoproteus*-infected mates laid smaller and later clutches than did females with unparasitized males (Korpimäki et al. 1995). Among American Kestrels (*Falco sparverius*) infected with *Haemoproteus*, pairs with lower intensity infections fledged more young than birds with higher intensities (Apanius 1991).

There is growing evidence for a trade-off between reproductive effort and resistance to parasites that is thought to arise when limited resources must be partitioned between reproductive effort and disease resistance (Chapter 1). Parasite intensity (as measured by numbers of circulating gametocytes) increases with the degree of effort expended in reproduction (Norris et al. 1994; Ots and Horak 1996; Allander 1997; Siikamäki et al. 1997; Nordling et al. 1998) and may decrease when food resources are abundant (Wiehn and Korpimaki 1998).

From other studies of the subclinical impacts of *Haemoproteus* infections on wild birds, results have often been conflicting and dependent on the particular host–parasite association under investigation, whether or not hosts had concurrent infections with other hematozoan parasites, whether stage of infection was acute or chronic, and age of the hosts. A number of studies have been unable to establish a relationship between infection with *Haemoproteus* and survivorship, mating success, reproductive success, host condition, or clinical chemistry (Bennett et al. 1988; Weatherhead and Bennett 1992; Davidar and Morton 1993; Powers et al. 1994; Korpimäki et al. 1995; Dale et al. 1996; Hõrak et al. 1998; Dawson and Bortolotti 2000; Schrader et al. 2003), yet others find subtle effects that are either difficult to detect or are equivocal (Dawson and Bortolotti 2000). For example, no association was detected between infection with *Haemoproteus tinnunculi* and return rates of American Kestrels when data from both sexes were combined, but there was a significant negative association between return rates and intensity of infection in females (Dawson and Bortolotti 2000). This suggests that acute or recrudescing infections may have more impact on host survivorship than chronic, low-intensity infections, but that effects may be subtle and easily masked when data for males and females are combined. By contrast, Purple Martins (*Progne subis*) infected with *Haemoproteus prognei* returned to breeding sites earlier than uninfected birds, and infected females had higher numbers of fledged young than uninfected birds (Davidar and Morton 1993). These authors hypothesized that recovery from acute phases of infection of *Haemoproteus* was evidence of immunological superiority in surviving hosts and may actually be a measure of superior fitness.

TREATMENT AND CONTROL

A number of antimalarial compounds are effective for reducing intensity of parasitemia in both wild and domestic birds with infections with *Haemoproteus*. These include atebrine, plasmochin, chloroquine sulfate, primaquine, and mefloquine (Coatney 1935; Evans and Otter 1998; Mutlow and Forbes 1999; Remple 2004) as well as the antitheilerial drug buparvaquone (El-Metenawy 1999). Other antimalarials may be effective including pyrimethamine, pyrimethamine–sulfadoxine combinations, and tetracyclines, but their effectiveness in birds is not wildly established (Mutlow and Forbes 1999).

In captive situations, infections with *Haemoproteus* can be controlled by housing birds in screened, *Culicoides*-proof facilities and dusting birds to reduce or eliminate ectoparasitic hippoboscid flies.

MANAGEMENT IMPLICATIONS

There are currently no broad-scale strategies for prevention or control of infections with *Haemoproteus* in wild birds. While reduction of vector populations will decrease transmission of species of *Haemoproteus*, this approach is currently not feasible for the many species of ceratopogonids that have larval habitats in damp soil and tree cavities (Blanton and Wirth 1979) or for ectoparasitic hippoboscid flies that occur on wild birds. It is likely that some species of *Haemoproteus* may become emerging disease threats in the event of global climate change as the range of hosts and vectors change, bringing previously isolated populations into contact with vectors and parasites to which they had no prior exposure. On a smaller scale, similar circumstances occur when avian species are transported or relocated outside of their normal range. Good examples are the recent epizootics of *H. lophortyx* in Northern Bobwhites that were relocated in California (Cardona et al. 2002), sporadic reports of myopathy from megalomeronts in captive psittacines (Pennycott et al. 2006), and periodic outbreaks in other captive birds and zoos where new exotic hosts are exposed to endemic vectors and parasites (Ferrell et al. 2007).

DISCLAIMER

Any use of trade, product, or firm names in this publication is for descriptive purposes only and does not imply endorsement by the U.S. government.

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3
Avian Malaria

Carter T. Atkinson

INTRODUCTION

Avian malaria is a common mosquito-transmitted disease of wild birds that is caused by protozoan parasites in the genus *Plasmodium*. Infections are caused by a complex of more than 40 species that differ widely in host range, geographic distribution, vectors, and pathogenicity. The avian species of *Plasmodium* share morphological and developmental features with closely related haemosporidian parasites in the genera *Haemoproteus* and *Leucocytozoon* (Chapters 2 and 4), but are distinguished from both by the presence of asexual reproduction (merogony) in circulating erythrocytes.

While there are numerous reports of individual birds with acute, pathogenic infections with *Plasmodium*, reports of epizootics are rare and mostly associated with captive birds in zoological collections and abnormal host–parasite associations following introductions of parasites or mosquito vectors to remote islands. *Plasmodium relictum*, one of the most widely distributed species of avian malaria (Beadell et al. 2006), continues to play an important role as a limiting factor in the current distribution and abundance of native Hawaiian forest birds (Warner 1968; Woodworth et al. 2005; Foster et al. 2007).

SYNONYMS

Avian malaria, haemoproteosis. Many reports in the recent ecological literature lump *Plasmodium* with *Haemoproteus* and refer to both genera as avian malaria, making it difficult to identify which genus is being discussed. Clear differences in life history characteristics of these two genera justify their continued separation (Valkiūnas et al. 2005) even though they are closely related (Martinsen et al. 2008).

HISTORY

The avian species of *Plasmodium* have played a seminal role as models for human malaria since they were first recognized as common intraerythrocytic parasites of wild birds (Danilewsky 1889). The early years of this field have been reviewed in detail by Hewitt (1940), Garnham (1966), and Valkiūnas (2005), and it is clear that most of the major milestones in the field of human malariology were associated in one way or another with avian parasites. Highlights include the first descriptions of the characteristic pathological lesions of malaria in birds by Danilewsky (1889), the discovery of the mosquito transmission of *P. relictum* by Sir Ronald Ross (1898), discovery of the exoerythrocytic merogony of *Plasmodium elongatum* in reticuloendothelial cells in bone marrow and other organs in birds (Raffaele 1934), and the development of the theory of premunition or a resistance to reinfection that is conferred by a chronic malarial infection in avian hosts (Sergent and Sergent 1956).

It was recognized relatively early that both wild and captive birds experience significant disease following infection with avian malaria, with reports as early as 1905 of die-offs from infection with *Plasmodium* in Gray Partridges (*Perdix perdix*) that were imported from Hungary and released in France (Garnham 1966). Despite this lengthy history, the number of reports of large-scale epizootics from avian malaria over the past 100 years are surprisingly limited, with most associated with wild Ciconiiformes in Venezuela (Gabaldon and Ulloa 1980), captive penguins (Fix et al. 1988), and native Hawaiian forest birds (Warner 1968).

There has been a recent renaissance in the use of prevalence data on hematozoan infections in birds to investigate ecological and evolutionary hypotheses about sexual selection and the physiological costs of parasitism in wild bird populations (Hamilton and Zuk 1982; Kilpatrick et al. 2006; Gilman et al. 2007). Some of this work is based on the use of molecular methods to diagnose very low intensity infections and track host specificity and geographic distribution of mitochondrial lineages of these parasites (Ricklefs et al. 2005). These new tools are leading to fundamental revisions in how we define species of *Plasmodium* and will play

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an important role in assessing their impact on wildlife populations.

DISTRIBUTION

The species of *Plasmodium* that infect birds have a cosmopolitan distribution and are found in all major zoogeographic regions of the world with the exception of Antarctica, where mosquito vectors responsible for their transmission do not occur. Reports of *Plasmodium* from the Australian region are notably fewer than others, but it is not clear whether this is because this region has not been adequately sampled or whether it reflects a true distributional anomaly (Bennett et al. 1993; Valkiūnas 2005).

Seven species of *Plasmodium* have a cosmopolitan distribution and broad host range, with reports from as few as 67 species of avian hosts for *P. elongatum* to as many as 419 different species of birds for *P. relictum* (Bennett et al. 1993; Valkiūnas 2005). *Plasmodium relictum* and *P. circumflexum* have the broadest geographic distribution and are reported from the Nearctic, Palearctic, Oriental, Ethiopian, Neotropical, and Australian regions. *Plasmodium vaughani*, *P. cathemerium*, *P. nucleophilum*, *P. rouxi*, and *P. elongatum* have been reported from all regions with the exception of the Australian region (Bennett et al. 1993).

HOST RANGE

Infections with *Plasmodium* have been reported in birds from all avian orders with the exception of the Struthioniformes (ostriches), the Coliiformes (mousebirds), and the Trogoniformes (trogons and quetzals), but only about half of all avian species have been examined for these parasites. The greatest diversity of species of *Plasmodium* is recorded from the Galliformes, Columbiformes, and Passeriformes (Valkiūnas 2005). Important resources for locating host records and early literature on *Plasmodium* infections in wild birds have been prepared by the International Reference Centre for Avian Hematozoa (Herman et al. 1976; Bennett et al. 1981, 1982; Bishop and Bennett 1992).

Plasmodium relictum has one of the widest host ranges of the avian plasmodia, occurring naturally in 70 different avian families. The relatively broad host range of most species of *Plasmodium* from birds is considered to be characteristic of the avian species of this genus, but exceptions are common. Based on identifications made by traditional morphological methods, some species appear to have very restricted host distributions in wild populations. For example, *Plasmodium hermani* and *Plasmodium kempi* have been described from domestic and Wild Turkeys (*Meleagris gallopavo*) in North America, yet are different enough in morphological features to be described as separate species. *Plasmodium hermani* has also been found in Northern Bobwhite (*Colinus virginianus*) from the same habitats as Wild Turkeys in Florida, USA (Forrester et al. 1987), but prevalence in other species of wild birds from the same habitats is not known. While *P. kempi* is capable of infecting species of Galliformes and Anseriformes in the laboratory, Wild Turkeys are the only known natural host of this parasite (Christensen et al. 1983).

Recent application of molecular methods to screen avian hosts has revealed a far greater complexity of genetic lineages of *Plasmodium* and the closely related genus *Haemoproteus* that are currently difficult to relate to more traditional morphological species (Bensch et al. 2004). Multiple lineages can occur in the same host individual, and their occurrence in species from a wide range of avian orders, families, and species is much broader than previously recognized (Fallon et al. 2005; Ricklefs et al. 2005; Szymanski and Lovette 2005).

ETIOLOGY

Members of this genus are classified as members of the phylum Apicomplexa, class Aconoidasida, order Haemospororida, family Plasmodiidae and are defined primarily by their intraerthrocytic development and asexual reproduction (merogony, also called schizogony) in the circulating blood cells (Peirce 2000). All members of this genus produce prominent golden-brown or black pigment granules from digestion of host hemoglobin. The species of *Plasmodium* that infect birds are divided into five subgenera based on morphology of circulating gametocytes and meronts and on preference for mature or immature erythrocytes (Table 3.1; Figure 3.1; Valkiūnas 2005). Peirce and Bennett (1996) recognize a sixth subgenus among the avian parasites, *Plasmodioides*, that was erected for a single species, *Fallisia neotropicalis*, from pigeons and Ciconiiformes in Venezuela (Gabaldon et al. 1985). This unusual avian parasite lacks pigment granules in all stages of development and develops exclusively in circulating leukocytes and thrombocytes. Peirce and Bennett (1996) argue that similarities in life history characteristics justify including this parasite among the avian malarial parasites as a species of *Plasmodium*, but most workers now place this subgenus in the family Garniidae (genus *Fallisia*) with reptilian blood parasites that also undergo merogony in circulating leukocytes (Valkiūnas 2005).

Species of *Plasmodium* are further distinguished by host range, vectors, and developmental characteristics of exoerythrocytic tissue stages. More than 40

Table 3.1. Subgenera and species of avian *Plasmodium* and characteristics of erythrocytic stages of development.

Subgenus	Characteristics	Species
Haemamoeba	Gametocytes round and exceed size of host cell nucleus Mature parasites displace host cell nucleus Meronts present in mature erythrocytes	*Plasmodium relictum* *Plasmodium subpraecox* *Plasmodium cathemerium* *Plasmodium gallinaceum* *Plasmodium matutinum* *Plasmodium lutzi* *Plasmodium giovannolai* *Plasmodium griffithsi* *Plasmodium tejerai* *Plasmodium coturnixi* *Plasmodium parvulum*
Giovannolaia	Gametocytes elongate Mature parasites do not displace host cell nucleus Meronts present in mature erythrocytes Meronts larger than erythrocyte nucleus, with plentiful cytoplasm	*Plasmodium fallax* *Plasmodium circumflexum* *Plasmodium polare* *Plasmodium lophurae* *Plasmodium durae* *Plasmodium pedioecetae* *Plasmodium pinottii* *Plasmodium formosanum* *Plasmodium gundersi* *Plasmodium anasum* *Plasmodium garnhami* *Plasmodium hegneri* *Plasmodium octamerium* *Plasmodium gabaldoni* *Plasmodium leanucleus*
Novyella	Gametocytes elongate Mature parasites do not displace host cell nucleus Meronts present in mature erythrocytes Meronts smaller than erythrocyte nucleus, without noticeable cytoplasm	*Plasmodium vaughani* *Plasmodium columbae* *Plasmodium rouxi* *Plasmodium hexamerium* *Plasmodium nucleophilum* *Plasmodium dissanaikei* *Plasmodium paranucleophilum* *Plasmodium bertii* *Plasmodium kempi* *Plasmodium forresteri* *Plasmodium ashfordi*
Bennettinia	Gametocytes, round or oval, do not exceed size of host cell nucleus and stick to host nucleus Meronts present in mature erythrocytes Meronts round with scant cytoplasm and stick to host nucleus	*Plasmodium juxtanucleare*
Huffia	Gametocytes elongate Mature parasites do not displace host cell nucleus Meronts variable in form and size Meronts present in circulating erythrocyte precursors	*Plasmodium elongatum* *Plasmodium huffi* *Plasmodium hermani*

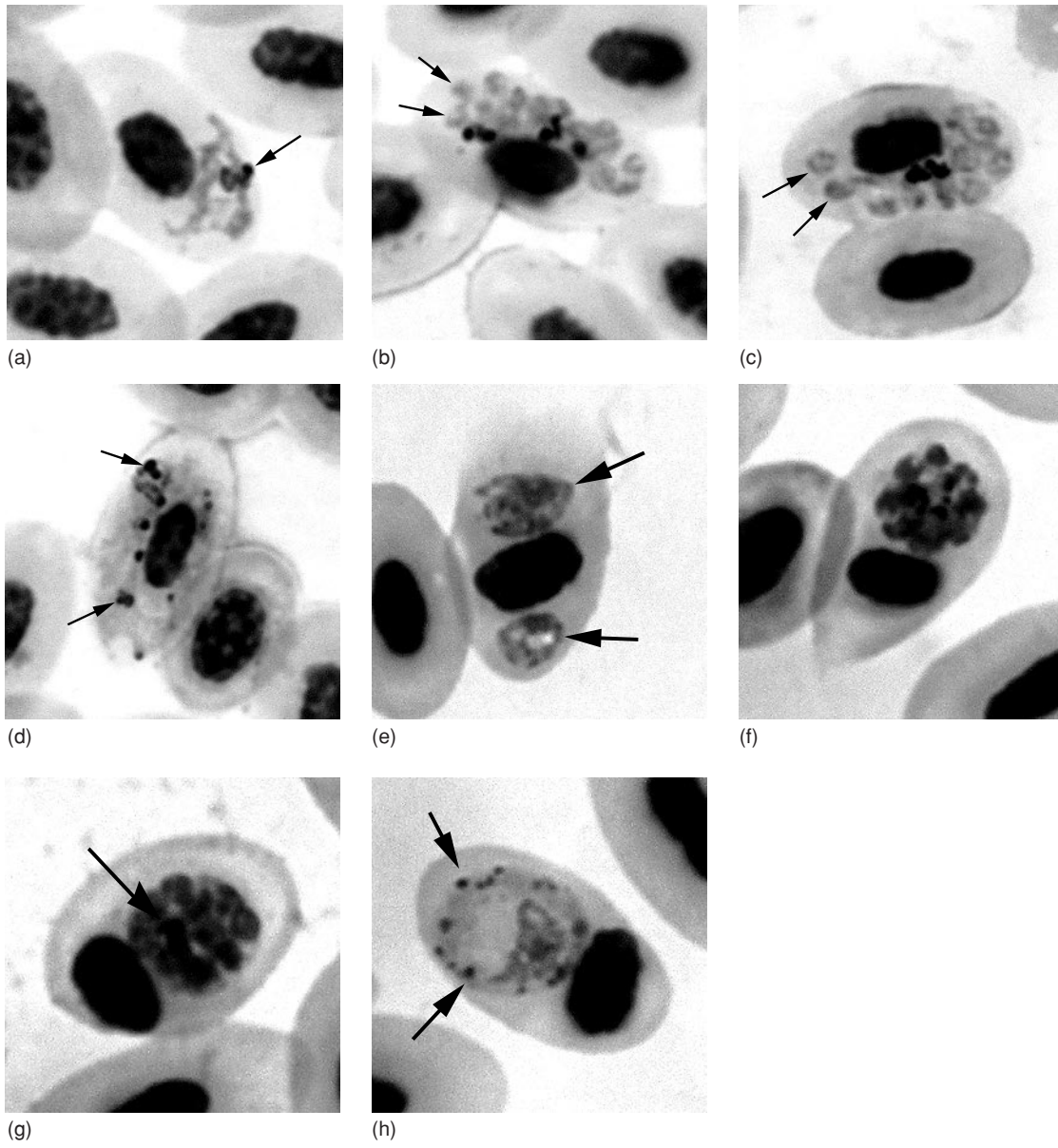


Figure 3.1. Erythrocytic stages of *Plasmodium circumflexum* (subgenus *Novyella*) (a–d) and *Plasmodium relictum* (subgenus *Haemamoeba*) (e–h). Note elongated shape of meronts (b, c) and gametocyte that encircles the host erythrocyte nucleus (d). The latter is characteristic of species of *Plasmodium* in the subgenus *Novyella*. Note shift in host erythrocyte nuclei (e–h) and round shape of gametocyte (h) that is characteristic of species of *Plasmodium* in the subgenus *Haemamoeba*. (a) Trophozoite. Note cluster of pigment granules (arrow). (b) Mature meront. Individual merozoites (arrows) are evident. (c) Mature meront. Note individual merozoites (arrows). (d) Gametocyte. Gametocyte surrounds the host erythrocyte nucleus, filling the erythrocyte cytoplasm. Pigment granules (arrows) are scattered through the parasite cytoplasm. (e) A pair of trophozoites (arrows). (f) Mature meront. Developing merozoites surround a central mass of pigment. (g) Mature meront. Developing merozoites surround a central mass of pigment (arrow). (h) Gametocyte. Note round shape, displaced host erythrocyte nucleus, and scattered pigment granules (arrows).

species are currently recognized, but this number is in a continual state of flux as existing species are synonomized as more information is learned about their biological characteristics and new species are described (Table 3.1).

The recent application of molecular methods to the taxonomy of this group has identified a bewildering array of lineages that are currently defined by sequence of mitochondrial genes (Bensch et al. 2004). In most cases, we know nothing about their erythrocytic morphology, natural vectors, or other life history characteristics and are only just beginning to link this information to the more traditional morphological and biological definition of individual species (Valkiūnas et al. 2007). Recent efforts to combine molecular data with life history information from members of five subgenera (*Haemamaoba*, *Huffia*, *Bennettinia*, *Novyella*, and *Giovannolaia*) indicate that the very distinctive characteristics of the subgenera *Haemamoeba* (large, round gametocytes and prominent host nucleus displacement), *Huffia* (predilection for immature erythrocytes), and *Bennettinia* (unusual morphology of the erythrocytic and sporogonic stages) are consistent with monophyletic origins for each of these subgenera. By contrast, *Novyella* and *Giovannolaia* form a clade composed of representatives from both subgenera, indicating that the less distinctive morphology of these parasites appears to be more plastic over evolutionary time (Martinsen et al. 2008). These findings suggest that some of the key morphological features used by parasitologists to distinguish these subgenera may not reflect true phylogenetic relationships (Martinsen et al. 2008). It is clear that our understanding of the taxonomy and phylogenetics of these parasites is rapidly evolving, and their classification will likely undergo further revision in future years.

EPIZOOTIOLOGY

Much of what we know about the detailed life cycle of species of *Plasmodium* from birds is based on a series of classic experiments by Clay Huff and coworkers with *Plasmodium gallinaceum*. In these studies, chickens and other birds were exposed to infective mosquito bites and examined at sequential time intervals to determine the location and morphology of the parasites (Huff and Coulston 1944; Huff 1951). These studies have provided us with specific details about how some of these parasites develop, but variations in the life cycle have been documented among other species of *Plasmodium* and further studies are needed.

The life cycle of *P. gallinaceum* begins when infective sporozoites are inoculated by a mosquito vector into a susceptible host (Huff and Coulston 1944). Sporozoites invade macrophages and fibroblasts near the site of the mosquito bite and undergo an initial generation of asexual reproduction (merogony) as cryptozoites. These are relatively small in diameter and mature in approximately 36–48 h to release ovoid merozoites that invade cells of the lymphoid–macrophage system in brain, spleen, kidney, lung, and liver tissue to begin a second generation of merogony as metacryptozoites. Metacryptozoites mature and release merozoites that are capable of invading circulating erythrocytes and capillary endothelial cells of the major organs. The first two generations of merogony are referred to as the preerythrocytic stages of infection. Merozoites that continue with a third generation of merogony in stationary tissues of the host are called phanerozoites. Once they invade capillary endothelial cells and begin to reproduce by asexual merogony, they are referred to as exoerythrocytic meronts. Merozoites released from exoerythrocytic meronts can either invade circulating erythrocytes or reinvade endothelial cells to continue additional generations of merogony in stationary tissues. The exoerythrocytic meronts that occur in capillary endothelial cells are oval, elongate, or branching and similar in morphology to thin-walled meronts of *Haemoproteus* (Chapter 2). They are significantly larger than the preerythrocytic meronts and may contain hundreds of nuclei (Garnham 1966).

Merozoites that invade the circulating erythrocytes undergo merogony and develop within 24–48 h into either mature meronts containing 8–32 ovoid merozoites or gametocytes that are infective to mosquito vectors (Table 3.1). Depending on the species of *Plasmodium*, meronts may be either round or elongate and produce numbers of merozoites that may be characteristic for particular species. Merozoites typically bud from a central residual mass and destroy their host erythrocyte when they are released. By contrast, gametocytes are elongate or round and have a single nucleus. The male gametocytes (microgametocytes) typically stain pink with Giemsa stain, while female gametocytes (macrogametocytes) stain pale blue.

During growth in the erythrocyte, the parasites ingest host erythrocyte cytoplasm through a specialized structure known as a cytostome and digest host hemoglobin within one or more food vacuoles scattered throughout the cytoplasm of the parasite. Malarial pigment or hematozoin is produced as a by-product of the digestion of hemoglobin and may appear as golden-brown or black granules in the parasite cytoplasm. Clear food vacuoles with one or more pigment granules may be visible by light microscopy, depending on size of the vacuoles. Merogony may continue indefinitely in the circulating erythrocytes, and evidence suggests that merozoites from some erythrocytic meronts can reinvade stationary tissues and continue development as phanerozoites (Garnham 1966).

Unlike *P. gallinaceum* (subgenus *Haemamoeba*), *P. elongatum* and other species in the subgenus *Huffia* do not develop in capillary endothelial cells of the major organs, but instead undergo exoerythrocytic merogony in hematopoietic tissues of the host (Garnham 1966). Specific details about preerythrocytic stages of development are not known.

Gametocytes of all species of avian *Plasmodium* remain in the circulation and do not continue development until they are ingested by an arthropod vector. Once in the midgut of a suitable mosquito vector, they leave their host cells and undergo gametogenesis to form gametes. Male gametocytes undergo a process call exflagellation to produce up to eight, flagellated microgametes. One microgamete will fertilize a macrogamete and within 24 h a motile zygote develops, which is capable of penetrating the midgut wall and beginning development as an oocyst under the basal membrane of the mosquito midgut. These initial stages of gametogenesis and fertilization exhibit little or no host specificity for mosquito vectors and can be completed in vitro. It is only during invasion of the peritrophic membrane that surrounds the blood meal and subsequent penetration of the midgut epithelium that blocks in development of particular species of malaria, in particular mosquito hosts, can occur (Michel and Kafatos 2005).

Oocysts undergo a type of asexual reproduction called sporogony and eventually produce thousands of sporozoites through a process of budding from multiple residual masses or sporoblasts. Oocysts mature within approximately 7 days after reaching a diameter of approximately 40 μm, depending on ambient temperature, and rupture to release sporozoites into the hemoceol of the mosquito. Sporozoites move via the hemocoel to the salivary glands, penetrate the glandular cells, and eventually gain access to the salivary ducts. When a mosquito takes a blood meal, these pass with the saliva into a new avian host to initiate a new infection.

Birds typically undergo an acute phase of infection where parasitemia increases steadily to reach a peak in numbers, called the crisis, approximately 6–12 days after parasites first appear in the blood. This is followed by a rapid decline in intensity of infection to chronic levels as the host immune system begins to bring the infection under control. Chronic infections most likely persist for the lifetime of infected birds, and both circulating parasites and persistent exoerythrocytic meronts can serve as a source for recrudescing infections (Manwell 1934; Bishop et al. 1938; Garnham 1966).

More than 60 different species of culicine and anopheline mosquitoes are capable of supporting experimental development of a variety of species of *Plasmodium* from avian hosts (Huff 1965), but surprisingly, few natural mosquito vectors are known (Table 3.2). For example, more than 20 species of anopheline and culicine mosquitoes in four different genera (*Culex*, *Aedes*, *Culiseta*, and *Anopheles*) are capable of transmitting *P. relictum* in the laboratory, but only three—*Culex quinquefasciatus*, *Culex tarsalis*, and *Culex stigmatasoma*—are proven natural vectors of *P. relictum* in California and Hawaii (Reeves et al. 1954; LaPointe et al. 2005).

After the initial acute phase of infection, intensity appears to be influenced by the complex interplay of host immunity, seasonal changes in photoperiod, and hormonal changes associated with reproduction. As has been described for other hematozoan parasites (Chapters 2 and 4), an increase in intensity of infection coincides with the breeding season when populations of blood-sucking insects typically increase, and recently fledged susceptible birds are increasing in the population (Atkinson and van Riper 1991; Valkiūnas et al. 2004). Termed the "spring relapse," the increase in numbers of parasites in the peripheral circulation can be triggered by corticosterone (Applegate and Beaudoin 1970), increases in photoperiod, and subsequent physiological changes in levels of hormones such as melatonin that regulate circadian rhythms (Valkiūnas et al. 2004). Many of the same factors that affect intensity of infection with *Haemoproteus* (Chapter 2) probably affect intensity of infection with *Plasmodium*. These include stress-mediated changes in the immune system that are associated with reproductive effort (Siikamäki et al. 1997), food availability (Appleby et al. 1999), concomitant infection with other parasites (Wright et al. 2005), and exposure to predators (Navarro et al. 2004).

While it is clear that most transmission of avian *Plasmodium* takes place during the spring and summer months in temperate climates, relatively little is known about dynamics of infection in tropical parts of the world. In Hawaii, transmission of *P. relictum* at lower elevations can take place throughout the year (Woodworth et al. 2005), but is more seasonal at higher elevations where both temperature and rainfall have significant effects on vector populations (Ahumada et al. 2004). By contrast, transmission of *P. hermani* in Wild Turkeys in subtropical Florida is limited primarily to late summer and early fall when populations of the primary vector, *Culex nigripalpus*, reach a peak. As is the case with *Haemoproteus* (Chapter 2), both the spatial and seasonal patterns of transmission depend on availability of suitable mosquito vectors and susceptible avian hosts. Among migratory species, recent evidence indicates that transmission of some species of *Plasmodium* and other haemosporidian parasites can occur on both the breeding and the wintering grounds,

Table 3.2. Proven and suspected natural vectors of species of *Plasmodium* from birds, based on demonstration of oocysts or sporozoites from wild mosquitoes or transmission by wild-captured mosquitoes.

Parasite species	Locality	Mosquito vector	Reference
Plasmodium relictum	California, USA	*Culex stimatosoma*	Reeves et al. (1954)
	California, USA	*Culex tarsalis*	Reeves et al. (1954)
	Hawaii, USA	*Culex quinquefasciatus*	LaPointe et al. (2005) and Woodworth et al. (2005)
Plasmodium gallinaceum	Sri Lanka	*Mansonia crassipes*	Niles et al. (1965) and Garnham (1966)
Plasmodium circumflexum	Sri Lanka	*Mansonia crassipes*	Niles et al. (1965) and Garnham (1966)
	New Brunswick, Canada	*Culiseta morsitans**	Meyer et al. (1974)
Plasmodium rouxi	Algeria	*Culex pipiens*	Sergent et al. (1928) and Garnham (1966)
Plasmodium juxtanucleare	Malaysia	*Culex sitiens*	Bennett et al. (1966)
		Culex annulus	Bennett and Warren (1966)
	Brazil	*Culex saltanensis*	Lourenco-de-Oliveira and de Castro (1991)
Plasmodium hermani	Florida, USA	*Culex nigripalpus*	Forrester et al. (1980)
Plasmodium elongatum	Maryland, USA	*Culex pipiens**	Beier and Trpis (1981)
		*Culex restuans**	Beier and Trpis (1981)
Plasmodium (*Novyella*) sp.	Venezuela	*Aedeomyia squamipennis*	Gabaldon et al. (1977) and Gabaldon and Ulloa (1980)
Plasmodium (*Giovannolaia*) sp.	Venezuela	*Aedeomyia squamipennis*	Gabaldon et al. (1977) and Gabaldon and Ulloa (1980)

Note: Numerous other species of mosquitoes are capable of supporting development of avian species of *Plasmodium* under laboratory conditions (Huff 1965), but few studies have isolated *Plasmodium* from naturally infected vectors or linked these with demonstrated transmission in the wild.

*Sporozoites or oocysts of undetermined species were demonstrated in wild mosquitoes; laboratory susceptibility was confirmed.

leading to increases in parasite dispersal (Pérez-Tris and Bensch 2005; Hellgren et al. 2007).

Both intrinsic and extrinsic factors affect the distribution and prevalence of the closely related genus *Haemoproteus* (Chapter 2). Many of these same factors also determine the prevalence of *Plasmodium*, but this has not been examined in as much detail. Based on surveys by microscopy, prevalence of *Plasmodium* is four to five times lower than either *Haemoproteus* or *Leucocytozoon*, with an overall prevalence of less than 4% in a sample of over 2,000 birds from North America (Greiner et al. 1975). Prevalence of *Plasmodium* differed in specific physiographic regions of the continent, ranging as high as almost 10% in the southeastern US to less than 1% in the arctic barrens (Greiner et al. 1975). Very low prevalences of *Plasmodium* relative to *Haemoproteus* and *Leucocytozoon* may largely be a sampling artifact because very low intensity chronic infections are extremely difficult to detect by microscopy. Prevalence of *Plasmodium* is much higher when more sensitive diagnostic methods are used, such as those based on the polymerase chain reaction (PCR). For example, prevalence of *Plasmodium* in forest birds from American Samoa is 1% by microscopy, but approximately 60% by PCR amplification of parasite ribosomal genes (Jarvi et al. 2003; Atkinson et al. 2006).

Given their higher sensitivity, molecular methods may be valuable for investigating the effects of host behavior and ecology on prevalence of infection. In a large study of host and parasite community relationships in southern Missouri, USA, prevalence was weakly correlated with host body mass, but not with foraging stratum, nest height, nest type, plumage brightness, sexual dichromatism, age, or sex (Ricklefs et al. 2005). Significant relationships may have been obscured, however, by analysis of multiple parasite lineages that may differ in specific life history

characteristics. In more intensive studies of individual species of *Plasmodium* in defined host populations, prevalence may differ by both age and sex. For example, prevalence of infection with *P. circumflexum* and *P. cathemerium* is significantly higher in adults rather than juvenile Red-winged Blackbirds (*Agelaius phoeniceus*; Herman 1938), and differences in prevalence of *Plasmodium* by sex have been reported in other studies of this host species (Weatherhead and Bennett 1991).

The potential confounding effects of simultaneous infection with other haemosporidian parasites may also influence prevalence of *Plasmodium* by maintaining infections at higher frequencies than might be expected. For example, specific *Mhc* alleles that seem to be associated with susceptibility to *Plasmodium* may be maintained in a population of House Sparrows (*Passer domesticus*) because they confer resistance to a coinfecting strain of *Haemoproteus* (Loiseau et al. 2008).

CLINICAL SIGNS

Infections with *P. relictum* (canaries, Hawaiian honeycreepers, penguins), *P. gallinaceum* (domestic chickens), *P. juxtanucleare* (domestic chickens), *P. elongatum* (penguins), and *P. durae* (domestic turkeys) can be extremely pathogenic during acute phases of infection in their respective hosts (Garnham 1966; Stoskopf and Beier 1979; Huchzermeyer 1993a; Yorinks and Atkinson 2000; Williams 2005). Infected birds are typically anemic, lethargic, anorexic, and have ruffled feathers. Hematocrits may fall by more than 50% (Figure 3.2). Domestic chickens infected with *P. gallinaceum* and *P. juxtanucleare* have been described as lethargic, having pale combs, green droppings, diarrhea, and partial or total paralysis (Garnham 1966). Young turkeys with infections of *P. durae* exhibit few clinical signs until immediately before death, when severe convulsions may occur (Garnham 1966). Adult turkeys typically become lethargic, anorexic, and often develop right pulmonary hypertension as a consequence of hypoxic pulmonary arterial hypertension (Huchzermeyer 1988). Adult birds may also develop edematous legs and gangrene of the wattles. Cerebral capillaries may be blocked by developing exoerythrocytic meronts, and infected birds may exhibit neurological signs and paralysis before death (Garnham 1966).

During the crisis, when peripheral parasitemias reach their peak, chickens infected with *P. gallinaceum* have reduced plasma albumin and α_2-globulin as well as significant increases in γ_1- and γ_2-globulin (Williams 2005). These changes coincide with significant increases in plasma total protein and aspartate aminotransferase, glutamate dehydrogenase, and γ-glutamyltransferase and a decrease in creatinine that likely reflect tissue damage caused by developing both erythrocytic and exoerythrocytic parasites (Williams 2005). Increases in white blood cell counts, relative and absolute lymphocytosis, and total plasma solids have been documented in Hawaiian Crows (*Corvus*

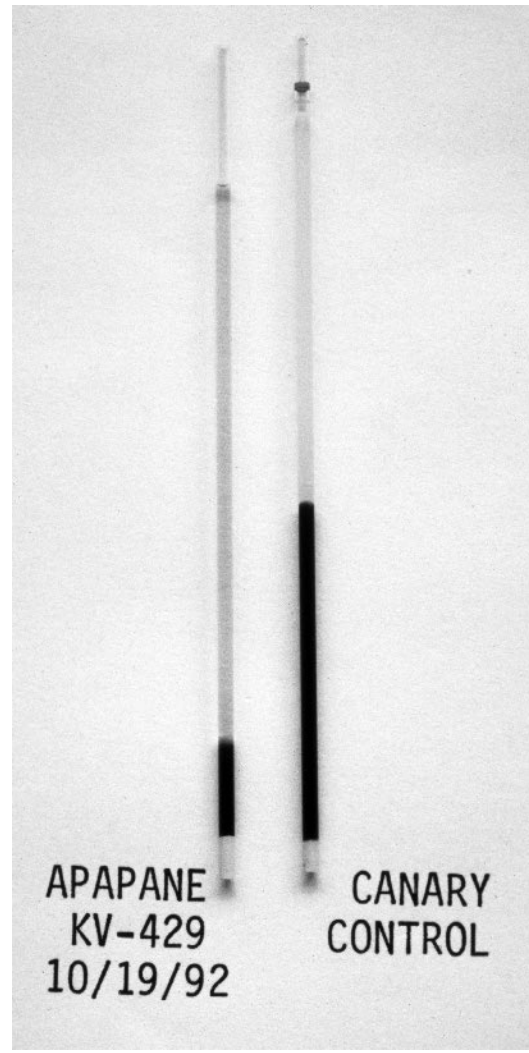


Figure 3.2. Hematocrit for a Wild Apapane (*Himatione sanguinea*) (left) with an acute natural infection with *Plasmodium relictum*. The hematocrit from an uninfected control canary (right) illustrates the severity of the anemia. Birds with acute infections of this intensity are rarely captured with mist nets in the wild.

hawaiiensis) and penguins with acute infections with *P. relictum* (Graczyk et al. 1994c; Massey et al. 1996). Hematological changes are much less evident in birds with chronic infections (Ricklefs and Sheldon 2007).

PATHOLOGY AND PATHOGENESIS

Avian malaria is primarily a disease of the blood and reticuloendothelial system, and the progress of the disease and clinical signs closely parallel increases in the number of parasites in the peripheral circulation (van Riper et al. 1994). In detailed studies of *P. gallinaceum* in experimentally infected chickens, clinical signs first become evident from 5 to 7 days after inoculation of infected blood (Williams 2005). These correspond to rapid increases in peripheral parasitemia and declines in hematocrit (Figure 3.3). Hemolysis of both infected and uninfected erythrocytes and catabolism of hemoglobin leads to production of excess biliverdin, which is excreted in the feces (Williams 1985). Infected birds begin to excrete green feces approximately 4 days after infection. During phase I, lasting only a few hours, feces are normal in form with green pigment confined to the fecal portion of the dropping. Thin, mucoid, brilliant green diarrhea develops by day 5 (phase II), which persists about 2 days among birds that survive infection. During phase III, birds are recovering from infection, and green coloration of the droppings is intermediate in intensity between that observed during phase I and phase II. Droppings lose all green color by the time that parasitemia becomes undetectable (Williams 2005).

It is not clear whether acute infections with *Plasmodium* cause the febrile paroxysms in birds that are so characteristic of human malarial infections. Increases in cloacal temperature have been measured during acute phases of infection with *P. gallinaceum* in chickens (Williams 2005). As is the case with human infections, the febrile period was relatively short-lived and closely paralleled increases in peripheral parasitemia. Following the crisis, cloacal temperatures fell and then remained below normal for several days (Figure 3.3).

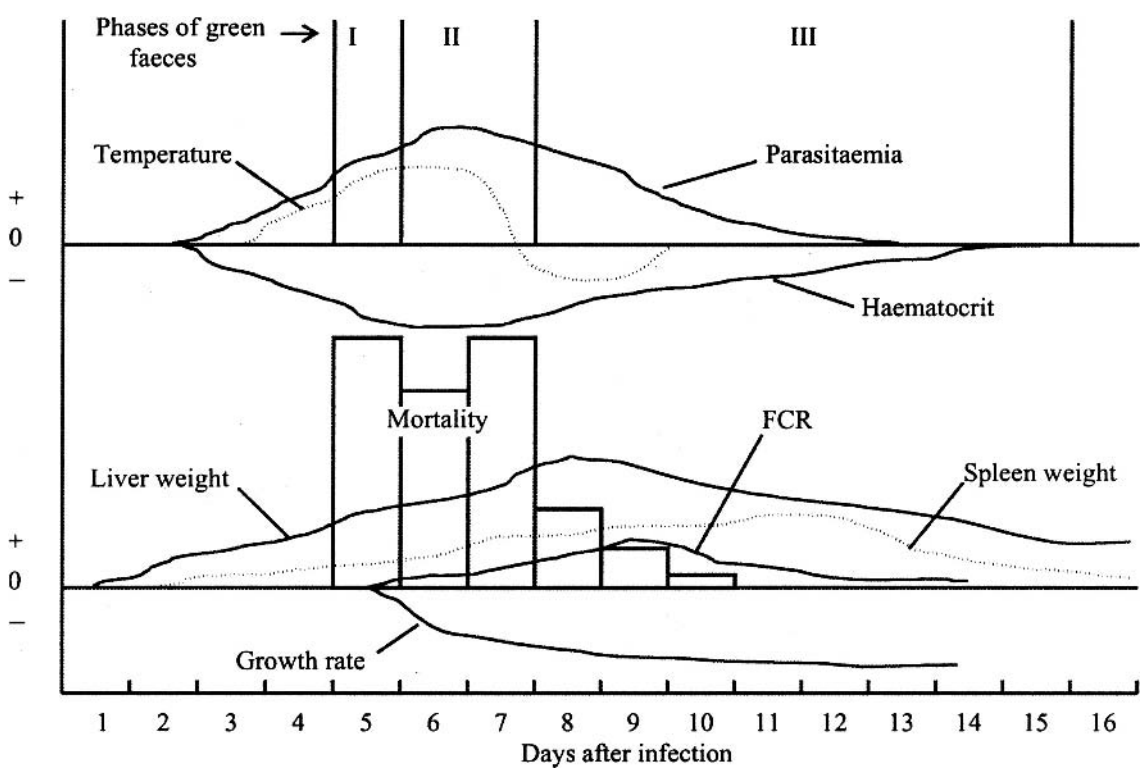


Figure 3.3. Relative timing of clinical signs of *Plasmodium gallinaceum* in domestic chickens following a blood-induced infection. Lines represent deviations from baseline conditions in healthy birds. FCR, food conversion ratio. Reproduced from Williams (2005), with permission of Taylor & Francis Ltd. (http://www.informaworld.com).



Figure 3.4. Livers and spleens from an uninfected control canary (right) and a canary with an experimental acute infection with *Plasmodium relictum* (left). Infected liver (bottom left) is enlarged, has rounded borders, is discolored from deposition of malarial pigment in tissue macrophages, and has multifocal areas of necrosis. Infected spleen (top left) is similarly enlarged and discolored from deposition of malarial pigment in tissue macrophages. Tissue has been fixed in 10% buffered formalin.

By contrast, canaries infected with *P. relictum* have significant declines in core body temperature during acute phases of infection and appear to lose the ability to thermoregulate (Hayworth et al. 1987).

The hallmark gross lesions produced by acute infections with *Plasmodium* include thin, watery blood, and enlargement and discoloration of the liver and spleen by deposition of malarial pigment in tissue macrophages (Figure 3.4). Enlargement of these organs is due to hypercellularity and increased phagocytic activity of macrophages rather than edema (Al-Dabagh 1966). Development of gross lesions closely corresponds to a steady increase in peripheral parasitemia, intravascular hemolysis of infected erythrocytes as meronts mature, phagocytosis of parasitized erythrocytes, and increased fragility of unparasitized erythrocytes (Al-Dabagh 1966; Seed and Kreier 1972; van Riper et al. 1994; Williams 2005). Regenerative, hemolytic anemia is associated with a drop in erythrocyte counts, replacement with immature erythrocytes, and drops in hemoglobin concentration that peak during the crisis (Figure 3.5). Anoxia and intravascular agglutinations of erythrocytes ("sludging" of blood) may lead to damage of endothelial cells lining the capillaries (Al-Dabagh 1966). Deposition of malarial pigment in macrophages of various organs, particularly liver and spleen, as infected cells are removed from the circulation can be extensive. In intense fatal infections, thrombi or emboli can form in some organs, particularly the spleen. Secondary shock may also occur during the terminal stages of some acute infections, resulting from destruction of large numbers of infected and uninfected erythrocytes. Capillaries and venules may be dilated and exhibit increased permeability, edema, and stasis of blood flow. Hemorrhage may be evident within the capillaries. Lowered blood pressure, lowered blood volume, disturbed fluid balance, increased coagulation times, and increased levels of potassium may also be evident in severe infections (Al-Dabagh 1966).

Infections with *P. cathemerium* produce inflammatory myopathy in skeletal muscle of experimentally infected canaries. This is characterized by degeneration of capillaries and muscle fibers and presence of mononuclear cell infiltrates. Carmona et al. (1996) suggest that this may be related to obstruction of capillaries

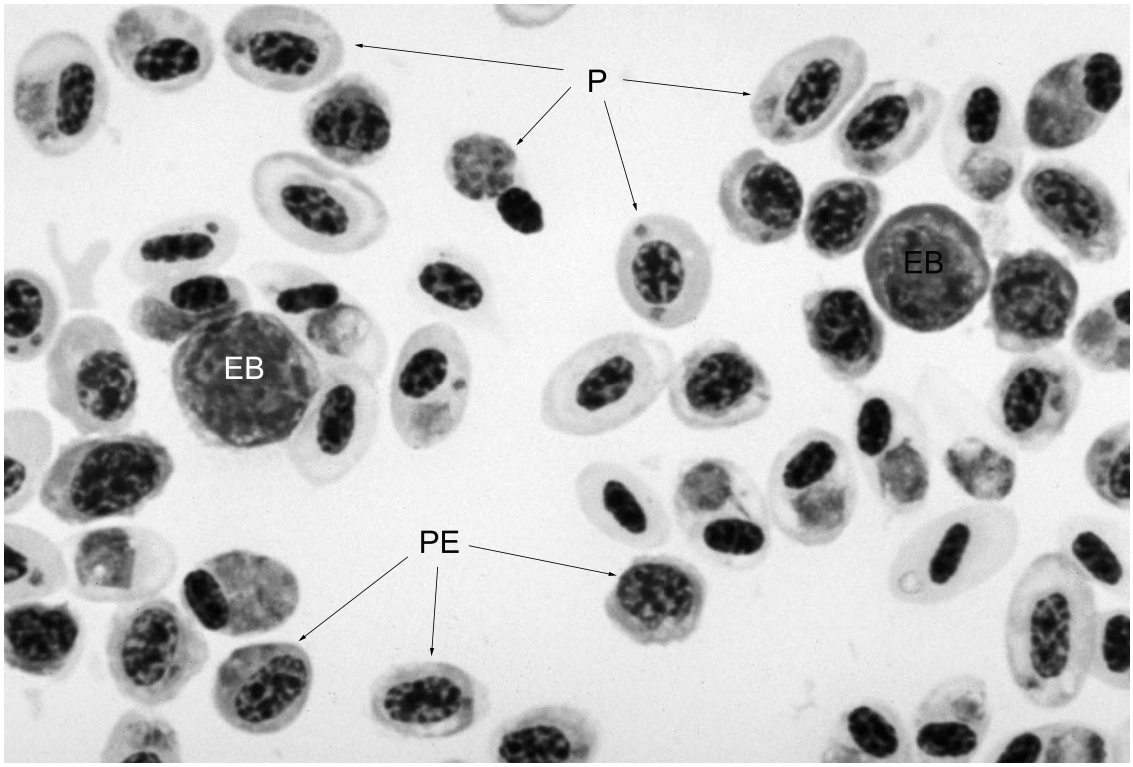


Figure 3.5. Blood smear from an Iiwi (*Vestiaria coccinea*) with an experimental infection with *Plasmodium relictum*. The normal cellular makeup of the blood is profoundly altered, with mature erythrocytes being replaced by erythroblasts (EB) and early polychromatic erythrocytes (PE). P, parasitized erythrocytes (Atkinson et al. 1995).

by infected erythrocytes. Anemia may also lead to circulatory deficiency that is compensated in part by increased cardiac output and dilation and hypertrophy of heart muscle (Al-Dabagh 1966).

While little or no host response is evident around preerythrocytic meronts of *Plasmodium*, the exoerythrocytic meronts of some species, for example, *P. gallinaceum* and *P. durae*, may partially or completely block capillaries, leading to leakage of plasma proteins, edema, and hemorrhage. These lesions may occur in the heart, lungs, renal glomeruli, and brain. When they occur in the brain, neurologic symptoms may appear and death can be sudden.

There is a clear association between the severity of disease and dose. This has been demonstrated experimentally with both blood-induced infections (Permin and Juhl 2002) and sporozoite-induced infections (Atkinson et al. 1995). Birds exposed to higher numbers of infective sporozoites have higher parasitemias, more severe gross and microscopic lesions, and higher mortality (Atkinson et al. 1995, 2000).

DIAGNOSIS

The gold standard for diagnosis of *Plasmodium* is a Giemsa-stained thin blood smear where it is possible to demonstrate the presence of erythrocytic meronts and gametocytes with prominent golden-brown or black pigment granules. Individual species are traditionally defined by size and shape of intraerythrocytic gametocytes and meronts (Table 3.1; Figure 3.1), number of merozoites produced by mature meronts, changes in morphology of the host erythrocyte, and other biological characteristics such as host range, susceptibility to species of mosquitoes, morphology, and location of exoerythrocytic meronts (Garnham 1966; Valkiūnas 2005). Since most identifications are made from blood smears, life history characteristics may be unknown, and it becomes essential to be able to find enough mature meronts and gametocytes on a smear to be able to make an accurate assessment of parasite morphology. Detailed keys and species descriptions have been recently revised by Valkiūnas (2005), and his monograph is currently the most

up-to-date resource for identifying species of avian *Plasmodium*.

Most infections of *Plasmodium* in wild birds are chronic, however, and intensity may be extremely low. In these cases, it may be impossible to identify parasites below level of subgenus. When erythrocytic meronts are not present, it may become difficult to distinguish gametocytes of *Plasmodium* from those of *Haemoproteus*, although gametocytes of *Haemoproteus* are often thicker and more robust than those of *Plasmodium*. The fact that species of *Plasmodium* have circulating meronts while species of *Haemoproteus* do not can be used to both isolate and identify an unknown species of *Plasmodium* if susceptible domestic or captive wild birds are available for experimental subinoculation of blood from the suspect bird. While it was common knowledge among early malariologists that *Plasmodium* can be passed to a new host by blood inoculation, Manwell and Herman (1935) and later Herman (1938) were the first to apply this method to diagnose infections with *Plasmodium* in wild birds. Blood from an infected host is passed by intravenous, intraperitoneal, or intramuscular inoculation into an uninfected host of the same species, and blood smears are prepared from the inoculated host for several weeks after injection. If the host is susceptible to the parasites, an acute phase infection will often result and meronts and gametocytes can be readily found for morphological analysis. When parasitemia is high, blood can be collected, treated with glycerin or dimethyl sulfoxide, aliquoted, and frozen in liquid nitrogen to create a frozen stabilite for further experimental studies (Garnham 1966).

Given the importance of morphological characters to identify species of *Plasmodium* from birds, their consistency and stability between hosts of different species is critical for making accurate identifications. Surprisingly, few studies have looked at this issue in detail. In one of the most widely cited examples, when *P. relictum* from Silver Gulls (*Larus novaehollandiae*) was passed by sporozoites to sparrows and canaries, merozoite, and gametocyte morphology changed significantly (Lawrence and Bearup 1961). In gulls, gametocytes were elongate and mature meronts had 10 merozoites. In sparrows, morphology was more typical of *P. relictum* and gametocytes were round or oval, and mature meronts had on average 14 merozoites. Other reports have documented changes in morphology when parasites are inoculated into atypical hosts (Garnham 1966) or when parasitemias are extremely high in immature erythrocytes (Laird and van Riper 1981). By contrast, other reports have documented relatively constant morphology in hosts from multiple avian species and orders (van Riper et al. 1986; Iezhova et al. 2005; Valkiūnas et al. 2007; C. T. Atkinson, unpublished observations). This issue clearly needs further study, and the relatively recent development of molecular methods to diagnose avian malaria with PCR primers to ribosomal and mitochondrial genes may help to resolve this problem. Despite their higher sensitivity, PCR methods may still miss infections that have extremely low parasitemias (Jarvi et al. 2002), although the recent application of real-time methods to malarial diagnostics may eventually solve these problems (Boonma et al. 2007).

Several recent sets of primers designed to amplify portions of the parasite mitochondrial genome can distinguish *Haemoproteus* and *Plasmodium* from *Leucocytozoon* (Hellgren et al. 2004) or all three genera from each other following restriction digests of PCR products (Beadell and Fleischer 2005). However, sequencing of PCR products is necessary for identifying individual parasite lineages and determining phylogenetic relationships. Since so few isolates of avian *Plasmodium* of known identity have been sequenced and typed, it is often not known how to relate unknown mitochondrial lineages to traditional morphological species. Recent rapid progress in the molecular diagnosis of avian species of *Plasmodium* may eventually make it possible to identify species based on mitochondrial lineage (Valkiūnas et al. 2007).

Plasmodium appears to be antigenically distinct from *Haemoproteus*, and crude antigen extracts have been used to develop an ELISA test for *P. relictum* in captive and wild penguins (Graczyk et al. 1994a, b). Standard immunoblotting techniques can also be used to identify antibodies to *Plasmodium* in wild and experimentally infected passerines (Atkinson et al. 2001). Although neither ELISA nor immunoblotting can distinguish species of *Plasmodium*, the techniques are useful for making diagnoses to level of genus in birds with low-intensity infections that may be missed by microscopy or PCR.

IMMUNITY

Birds infected with avian species of *Plasmodium* develop strong antibody and cell-mediated responses to erythrocytic parasites (van Riper et al. 1994), but appear to be unable to completely clear their infections. Limited evidence based on experimental studies in canaries (*P. relictum*), Hawaii Amakihi (*Hemignathus virens*) (*P. relictum*), and domestic turkeys (*P. hermani*) indicates that birds likely remain infected for life, but at chronic levels that stimulate immunity to reinfection with homologous strains of the parasite (Bishop et al. 1938; Jarvi et al. 2002; Young et al. 2004). This phenomenon, termed premunition, was recognized in the early part of the twentieth century (Hewitt 1940; Sergent and Sergent 1956). When birds with blood or sporozoite-induced infections are rechallenged, they

may have only brief, low-intensity increases in peripheral parasitemia (Hewitt 1940; Atkinson et al. 2001; Paulman and Mcallister 2005).

The persistence of subclinical infections may make birds vulnerable to the recrudescence of erythrocytic parasites if host immunity is compromised by stress or infection with other pathogens and provides an indirect measure of the cost of mounting an immune response. Experimental manipulation of clutch size led to increases in prevalence of *Plasmodium* in female Great Tits (*Parus major*) that laid more eggs, supporting the idea that there is a trade-off between the energetic costs of egg production and defense against parasites (Oppliger et al. 1996). Similarly, male Great Tits that expended extra energy to provision larger broods had a higher prevalence of malarial infection (Richner et al. 1995).

Exposure to other infectious diseases that compromise the immune system may also lead to recrudescing infections. When Wild Turkeys are exposed simultaneously or sequentially to turkeypox virus and *P. hermani*, both parasitemia and mortality are higher in 1-week-old poults infected with both agents than those exposed to either malaria or pox alone (Wright et al. 2005). These effects are less evident in older poults, suggesting that the host age may also play a role in pathogenesis of concomitant infections.

PUBLIC HEALTH CONCERNS

Avian species of *Plasmodium* do not infect humans, and infected birds pose no health risks to humans.

DOMESTICATED ANIMAL HEALTH CONCERNS

Domestic poultry are susceptible to several species of avian malaria, but their most significant effects occur outside of North America and Europe and specifically where wild reservoir hosts serve as sources of infection for domestic birds. *Plasmodium gallinaceum* is highly pathogenic in domestic chickens, particularly when European breeds are introduced to endemic areas in southeastern Asia, Malaysia, India, and Sri Lanka where the natural host is the Red Junglefowl (*Gallus gallus*; Garnham 1966). The distribution of the parasite in domestic chickens coincides with the geographic range of the natural host and has not expanded with the movement of domestic poultry to other parts of the world. *Plasmodium juxtanucleare* is also a significant pathogen in domestic chickens in South America, southern Africa, and southeastern Asia. Proven wild reservoirs of this species are found in India, Malaysia, South Africa, and Taiwan and include Red Junglefowl, Gray-winged Francolins (*Francolinus africanus*), and Chinese Bamboo-Partridges (*Bambusicola thoracicus*), but natural hosts are not known for other parts of its range (Garnham 1966; Fernando and Dissanaike 1975; Manwell et al. 1976; Earle et al. 1991).

Domestic turkeys are highly susceptible to *P. durae* in sub-Saharan Africa. This species is a parasite of wild francolins that infects domestic turkeys when wild reservoir hosts and vectors are present (Huchzermeyer 1993b). *P. durae* is highly pathogenic in domestic turkeys, and mortalities can be as high as 90% in young poults. Both *P. kempi* and *P. hermani* infect Wild Turkeys in North America, but have not reported to be a problem in domestic birds.

WILDLIFE POPULATION IMPACTS

There is relatively little evidence that species of avian *Plasmodium* are causes of major epizootic die-offs in their natural hosts. In a frequently cited example, high rates of transmission of species of *Plasmodium* from several subgenera have been documented in Venezuela among nesting Ciconiiformes, but clear evidence of malarial mortality in dead nestlings is not provided (Gabaldon and Ulloa 1980). In a thorough review of over 5,000 papers on avian blood parasites, Bennett et al. (1993) found that only about 4% reported mortality or pathogenicity in birds, with most dealing with domestic birds or birds in zoological collections.

Evidence is beginning to accumulate, however, that both direct and indirect effects of acute and chronic infections can have measurable impacts on the lifetime reproductive success of their avian hosts. In a study of singing behavior in White-crowned Sparrows (*Zonotrichia leucophrys oriantha*), song consistency was influenced by infection with *Plasmodium* and *Leucocytozoon*. Birds infected with *Plasmodium* also sang fewer songs following experimental playback of recorded songs (Gilman et al. 2007). This could have a significant impact on mate choice and reproductive success of infected males. Similarly, the behavioral effects of acute infections may lead to increased predation of infected hosts (Yorinks and Atkinson 2000; Møller and Nielsen 2007). These questions are just beginning to be explored in detail in ecological studies of wild birds, and the careful integration of both field and laboratory studies may lead to significant progress in our understanding of the more subtle costs of infection with these parasites.

The most significant reports of pathogenicity among species of *Plasmodium* that infect birds are in captive birds, zoological collections, and on isolated islands when new host–parasite associations become established. Avian malaria is particularly pathogenic in

captive penguins whenever they are exposed to mosquito vectors outside of their natural range (Stoskopf and Beier 1979; Fix et al. 1988). Well-documented cases of mortality from *Plasmodium* have not been reported in wild penguins (Jones and Shellam 1999; Sturrock and Tompkins 2007), but the introduction and spread of new mosquito vectors and the potential effects of global climate change may begin to place wild colonies at risk in future years (Miller et al. 2001; Tompkins and Gleeson 2006).

The threat that introduced avian malaria poses to endemic birds on isolated islands is substantial. The accidental introduction of *P. relictum* and the southern house mosquito (*C. quinquefasciatus*) to the Hawaiian Islands has had a devastating impact on native Hawaiian forest birds (Warner 1968; van Riper et al. 1986) and continues to play a significant role in limiting the current geographic and altitudinal distribution of remaining species (Atkinson et al. 1995; Benning et al. 2002). Of more than 70 species and subspecies of endemic forest birds present at the end of the eighteenth century, at least 23 are now extinct and 30 of the remaining species and subspecies are listed as endangered by the U.S. Fish and Wildlife Service (Jacobi and Atkinson 1995). While numerous limiting factors have contributed to these extinctions, high susceptibility to malaria is believed to be one of the most important reasons why populations of native species have collapsed at low elevations in areas where suitable habitat still exists (van Riper et al. 1986; Atkinson et al. 1995). High rates of transmission are maintained by the extremely high susceptibility of native honeycreepers (Drepanidinae) to *P. relictum* (Atkinson et al. 1995, 2000), presence of high rates of malaria transmission in the lowlands (Woodworth et al. 2005), and presence of disease-free refugia on the highest mountaintops that provide a continual source of nonimmune birds for initiating epizootics at lower elevations (Atkinson and LaPointe 2009). While many of the more rare native species are continuing to decline, at least one, Hawaii Amakihi (*Hemignathus virens*), appears to be evolving some resistance to infection, and lowland populations in some parts of Hawaii have started to rebound in recent years (Woodworth et al. 2005; Foster et al. 2007).

TREATMENT AND CONTROL

Chloroquine phosphate, primaquine phosphate, pyrimethamine–sulfadoxine combinations, and mefloquine are effective in treating canaries, penguins, and raptors with avian malaria (Remple 2004). The anticoccidial drugs sulfamonomethoxine, sulfachloropyrazine, and halofuginone are somewhat effective in treating *P. durae* in domestic turkeys and may also be effective against *P. gallinaceum*. Sulfamonomethoxine suppresses parasitemia, but does not provide full protection from mortality when given after the appearance of circulating parasites. Sulfachloropyrazine reduces mortality, but has no effect on parasitemia, suggesting that it has some efficacy against exoerythrocytic schizonts. Halofuginone delays parasitemia, but suppresses it to only a minor extent (Huchzermeyer 1993a).

While birds were some of the first experimental models for development of vaccines against *Plasmodium*, practical methods for immunizing wild birds have not been developed and this probably presents the most significant challenge to controlling infection with this approach. A variety of different experimental vaccines have been used, including use of ultraviolet light-inactivated, formalin-inactivated, and irradiated sporozoites, merozoites, and gametes, and synthetic vaccines based on parasite surface molecules (van Riper et al. 1994). Two DNA vaccines based on the circumsporozoite protein of *P. gallinaceum* and *P. relictum* have recently been evaluated in Jackass Penguins (African Black-footed Penguins, *Spheniscus demersus*; Grim et al. 2004), and canaries (McCutchan et al. 2004) exposed to natural transmission of *P. relictum* in a zoological park. Both provided protection to natural exposure to *P. relictum*, but immunity was short-lived in canaries, and birds were just as susceptible as unvaccinated controls when exposed to mosquito vectors 1 year later.

As has been demonstrated with human malaria, reductions of populations of mosquito vectors can reduce transmission of *Plasmodium*, but this method has not been widely used to control infections in wild or captive birds. Efforts to control avian malaria in Hawaiian forest birds have focused on reducing larval habitat for the introduced mosquito, *C. quinquefasciatus* (Reiter and LaPointe 2007; LaPointe et al. in press). The most cost-effective measures for captive or domestic birds include housing cage birds in screened, mosquito-proof buildings, or locating birds in areas that are isolated from wild reservoir hosts.

MANAGEMENT IMPLICATIONS

The potential risk of exposure to avian malaria should be considered when threatened or endangered species are moved outside of their normal ranges and maintained in captive propagation facilities or zoological parks where they may be introduced to new vectors and locally transmitted strains of *Plasmodium*. This risk is well documented for penguins, but should also be considered for species of birds from remote and isolated island systems that may have no prior exposure to these parasites. Similarly, the unintentional introduction of both parasites and mosquito vectors to new

habitats should be avoided to prevent establishment of new host–parasite associations that may be highly pathogenic (LaPointe et al. 2009).

DISCLAIMER

Any use of trade, product, or firm names in this publication is for descriptive purposes only and does not imply endorsement by the U.S. government.

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4
Leucocytozoonosis

Donald J. Forrester and Ellis C. Greiner

INTRODUCTION

Leucocytozoonosis is a vector-borne protozoan disease of birds caused by several species of Apicomplexa in the genus *Leucocytozoon*. There are many species of *Leucocytozoon*, but only a few are known to be pathogenic to their hosts. Avian groups at risk include waterfowl, pigeons, galliforms, raptors, and ostriches (Bennett et al. 1993b; Valkiūnas 2005). Several species cause significant mortality in domestic waterfowl and poultry, and one species (*Leucocytozoon simondi*) causes localized epizootics in wild ducks and geese (O'Roke 1931; Herman et al. 1975). Other species of *Leucocytozoon* cause disease on a smaller scale, but have not been studied extensively (Valkiūnas 2005). Undoubtedly, as more details on the life cycles and other biological aspects of the many relatively unstudied species are determined, others will be found to be pathogenic. All leucocytozoids are host specific at the avian order level and in some cases at the family level (e.g., *Leucocytozoon simondi*) and some even at the species level (e.g., *Leucocytozoon caulleryi* and *Leucocytozoon smithi*). They are closely related to species of the genera *Plasmodium* and *Haemoproteus* with similar life cycles, but are transmitted by black flies of the family Simuliidae, except for *L. caulleryi*, which is vectored by biting midges of the family Ceratopogonidae (Akiba 1960; Valkiūnas 2005).

There is a considerable body of literature on the various species of *Leucocytozoon*, only a part of which is discussed here. Several monographs and reviews have been published (Sambon 1908; Hewitt 1940; Bennett et al. 1965; Garnham 1966; Cook 1971; Fallis et al. 1974; Kučera 1981a, b; Atkinson and van Riper 1991; Greiner 1991; Desser and Bennett 1993; Valkiūnas 2005).

SYNONYMS

Hematozoan disease, haemosporidian disease, blood parasite disease, *Leucocytozoon* disease, and Bangkok hemorrhagic disease (refers specifically to *L. caulleryi* infection in domestic chickens in South and Southeast Asia).

HISTORY

The first publication on *Leucocytozoon* was by Sakharoff (1893) and was a morphological study of leucocytozoids of crows, magpies, and rooks in the Russian state of Georgia. This was followed by a paper written by Ziemann (1898) that included a description of leucocytozoids in the Little Owl (*Athene noctua*). He described the species as *Leukocytozoon* (sic) *danilewskyi* and was the first to stain blood films; his paper contained color illustrations of the gametocytes of this leucocytozoid (Ziemann 1898). The genus name *Leucocytozoon* was first used by Berestneff (1904) who described several species from owls, rooks, and crows, but Sambon (1908) was the first to define the genus *Leucocytozoon*. The family Leucocytozoidae was established later by Fallis and Bennett (1961). In 1965, the genus *Akiba* was established for one species of leucocytozoid (*Akiba caulleryi*) that is transmitted by biting midges rather than black flies (Bennett et al. 1965). This genus is currently considered by most as a subgenus of the genus *Leucocytozoon* (Valkiūnas 2005). It is interesting that the name *Leucocytozoon* was chosen originally for these blood protozoans because it was believed that they occupied only leukocytes. It was later discovered, however, that the gametocytes of this parasite developed in erythrocytes as well (Cook 1954; Desser 1967).

Leucocytozoids were first identified as agents of disease by Tartakovsky (1913) who worked with domestic and wild anseriforms in northeastern Russia. Tartakovsky's work was apparently overlooked by his contemporaries in Canada (Wickware 1915) and Germany (Knuth and Magdeburg 1922) who subsequently described the disease without reference to it (Valkiūnas 2005). Later work by O'Roke (1934), Karstad (1965), Fallis and Bennett (1966), Khan and Fallis (1968), Kocan (1968), Herman et al. (1975),

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Desser and Ryckman (1976), Mörner and Wahlström (1983), and others defined *L. simondi* as an important disease agent. *Leucocytozoon marchouxi* was suspected to be pathogenic to columbiforms by Oosthuizen and Markus (1968) and Peirce (1984), but this was not confirmed until the 1990s by Peirce et al. (1997). Initial evidence of the pathogenicity of *Leucocytozoon toddi* to raptors was reported by Olsen and Gaunt (1985) and Korpimäki et al. (1995), while conclusive data were published later by Raidal and Jaensch (2000).

The first discovery of arthropods as vectors of leucocytozoids occurred in the early 1930s when O'Roke (1930) and Skidmore (1931) simultaneously and independently showed that simuliid black flies transmitted *L. simondi* to ducks and *L. smithi* to turkeys. One species of *Leucocytozoon* (*Leucocytozoon caulleryi*) was found subsequently by Akiba (1960) to be transmitted by biting midges of the family Ceratopogonidae.

It was not until the 1940s that megalomeronts (=megaloschizonts; exoerythrocytic stages that develop in macrophages and other cells of the reticuloendothelial system) were discovered (Huff 1942; Wingstrand 1947). These were significant contributions that eventually led to an understanding of the pathogenicity of several species of leucocytozoids (Valkiūnas 2005).

During the 1960s and 1970s, considerable progress was made in understanding the morphology, development, and transmission of species of *Leucocytozoon*. Much of this was due to the efforts of Canadian researchers who were working with *L. simondi* in waterfowl, including A. M. Fallis, S. S. Desser, and R. A. Khan.

From the early 1900s to the present, there have been a number of publications on the taxonomy and systematics of the *Leucocytozoon* fauna of birds (Valkiūnas 2005). Among these were papers by Mathis and Léger (1909–1913), de Mello (1916–1937), Coatney (1937–1938), Herman (1938–1976), Bennett and colleagues (1965–1995), Ashford (1971–1990), Nandi (1977–1986), Peirce (1977–present), and Valkiūnas and colleagues (1983–present).

The formation of the International Reference Centre for Avian Malaria Parasites in 1967 in St. John's, Newfoundland, Canada, was an important milestone in the development of our knowledge of blood parasites, including species of *Leucocytozoon* (Bennett and Laird 1973). In 1975, the center was renamed the International Reference Centre for Avian Haematozoa, and in 1995, it was moved to the Queensland Museum in Brisbane, Australia. Laird and Bennett were active in initiating the formation of the center, which contains a large collection of the literature on blood parasites of birds and also a vast collection of over 64,000 preparations (mostly stained blood films) from throughout the world. Type material for many species of *Leucocytozoon* is contained in the collection of the center (Bennett et al. 1980). Bennett and many colleagues along with visiting scientists at the center produced a large number of publications describing new species and providing redescriptions of a number of known species of *Leucocytozoon*. In addition, they produced several publications containing bibliographies of pertinent literature (Herman et al. 1976; Bennett et al. 1981a; Bishop and Bennett 1992), a list of species names considered valid at the time (Bennett et al. 1994), and avian host–parasite checklists (Bennett et al. 1982b; Bishop and Bennett 1992). All this resulted in a new understanding, appreciation, and awareness of *Leucocytozoon* and other blood protozoans, and has provided a framework for subsequent work on this genus.

This foundation of knowledge made a key theoretical paper possible on the influence of blood parasites, including leucocytozoids, on the development of bright plumage, peculiarities of song, choice of mating partners, and the resultant effects on populations (Hamilton and Zuk 1982). Publication of this paper led to a renaissance in the use of avian blood parasites to test a wide range of ecological hypotheses on the impacts of parasites on sexual selection and host fitness (Møller 1997), but not without some controversy. Among the criticisms that have been made of the research are that it has been conducted by nonparasitologists who do not fully understand and appreciate the complexity of the host–parasite systems in question and that the research has been based on identifications of the blood parasites only to the generic level (using morphological and molecular techniques) rather than identifying the parasites to species (Cox 1989; McLennan and Brooks 1991; Poulin and Vickery 1993; Poulin 1995; John 1997; Valkiūnas 2005). Regardless, publication of the paper by Hamilton and Zuk (1982) has helped to open this field up to interdisciplinary studies by a wide range of parasitologists, ecologists, and avian biologists.

Recently, molecular techniques using polymerase chain reaction-based and restriction enzyme-based assays were developed to allow diagnosis of leucocytozoid infections in a time-efficient manner (Hellgren et al. 2004; Beadell and Fleischer 2005). These techniques should provide reliable and inexpensive methods for detecting *Leucocytozoon* infections, although not yet at the species level. At the present time, it seems wise to use both molecular and traditional morphometric (microscopic) techniques to diagnose leucocytozoid species and investigate their phylogenetic relationships such as has been done by Sehgal et al. (2006a) and Martinsen et al. (2006).

Table 4.1. Prevalence of species of *Leucocytozoon* in birds in various zoogeographic regions throughout the world.

Zoogeographic region	Number of birds examined	Number of birds infected	Prevalence of infection (%)
Holarctic	102,590	16,619	16.2
Ethiopian	11,507	529	4.6
Oriental	45,091	1,327	2.9
Neotropical	54,116	79	0.1
Australian	—*	—*	—*
Antarctic	0	0	0

Source: Modified from and includes data from Greiner et al. (1975), McClure et al. (1978), White et al. (1978), Peirce (1981), Valkiūnas (1987, 1996), Bennett et al. (1992a), and Forrester et al. (2001a).

* There are no appropriate data on prevalence available for the Australian region.

DISTRIBUTION

Leucocytozoids are distributed worldwide except in the Antarctic (Valkiūnas 1996, 2005). All the currently known species are found in the Holarctic, Ethiopian, and Oriental zoogeographic regions with a few species being also found in the Neotropical and Australian regions (Table 4.1). The highest prevalence of leucocytozoids, the highest species diversity, and the greatest number of species specific to a particular zoogeographic region occur in the Holarctic. The fauna of the Neotropical region is the poorest (White et al. 1978), especially in comparison to the Nearctic region where in certain areas leucocytozoids are the dominant hematozoan (Greiner et al. 1975). The increase in the general prevalence of leucocytozoids on a gradient from the south (e.g., Neotropical region) to the north (e.g., Nearctic region) has been attributed to increased densities of host populations in the north and to changes in the dynamics of transmission (White et al. 1978; Valkiūnas 2005). Some species of *Leucocytozoon* are found even above the Arctic Circle and are transmitted there (Valkiūnas et al. 1990). These observations may be somewhat skewed because birds in the Holarctic region have been studied fairly extensively, whereas the avifaunas in other regions such as the Neotropic and Australian regions have received less attention (Valkiūnas 2005).

The geographic distributions of eight species of *Leucocytozoon* that have been reported to cause leucocytozoonosis in domestic and wild birds are presented in Table 4.2. The three species of most concern in wild populations include *L. simondi* in waterfowl, *L. marchouxi* in pigeons and doves, and *L. toddi* in raptors. All three are found commonly in the Holarctic region, whereas *L. marchouxi* and *L. toddi* are common there as well as in the Oriental and Ethiopian regions. *Leucocytozoon toddi* probably has the most widespread geographic distribution of the three species; in addition to the Holarctic, Oriental, and Ethiopian regions, it is found in the Neotropical region, although it seems to be less common in the latter three regions. The global distribution of *L. simondi* is given in Figure 4.1. There are no reports from Greenland, Iceland, South America, Australia, or Antarctica, and only one unconfirmed report from Africa. There are no reports of transmission of *L. simondi* in Africa, South Asia, and Mexico; the records from these regions were from overwintering Holarctic anatids (Valkiūnas 2007, Personal communications to D. J. Forrester, June 29, 2007, September 4, 2007, and October 22, 2007).

On a smaller scale, the distribution of leucocytozoids within different zoogeographic regions is influenced by the presence or absence of flowing streams and rivers in which the vectors develop (Desser and Bennett 1993; Valkiūnas 2005).

HOST RANGE

Species of *Leucocytozoon* have been reported from 22 of the 28 orders and 113 of the 204 avian families of birds recognized by Clements (2000) (Table 4.3). The highest numbers of species occur in Passeriformes (8 species), Galliformes (7), and Coraciiformes (4). Only 1, 2, or 3 species have been found in birds of the other avian orders. About 45% of the species of birds in the world have been investigated for blood parasites, and species of *Leucocytozoon* have been found in approximately 30% of these birds (Valkiūnas 2005).

No leucocytozoids have been reported from five orders of birds, that is, Tinamiformes (tinnamous), Podicipediformes (grebes), Procellariiformes (albatrosses and petrels), Phoenicopteriformes (flamingos), and Pterocliformes (sandgrouse). In some cases, the records for some orders might reflect sporadic or accidental infections in an abnormal host. One example is the report of *Leucocytozoon* sp. in a gaviiform (the Common Loon, *Gavia immer*). The loon in question was in captivity in an outdoor pen for over a month

Table 4.2. Geographic distribution of eight species of *Leucocytozoon* that are pathogenic to wild and domestic birds.

Species of *Leucocytozoon*	Main avian hosts	Common occurrence	Occasional records
		Pathogenic to wild birds	
Leucocytozoon marchouxi	Pigeons and doves	Holarctic and Ethiopian regions	Oriental region and Central America
Leucocytozoon simondi[*]	Ducks, geese, and swans	Holarctic region	Mexico and other areas outside of Holarctic region
Leucocytozoon toddi	Raptors	Holarctic and Ethiopian regions	Oriental and Neotropical regions
		Pathogenic mainly to domestic birds	
Leucocytozoon smithi	Turkeys	Nearctic region	Europe and South Africa (introduced)
Leucocytozoon macleani[†]	Chickens and pheasants	South and Southeast Asia	Central and southern Palearctic region and also Ethiopian and Oriental regions
Leucocytozoon struthionis	Ostriches	South Africa	None
Leucocytozoon schoutedeni[†]	Chickens	Ethiopian region	Southeast Asia and USA (possibly introduced)
Leucocytozoon caulleryi	Chickens	South Asia and Southeast Asia	None

Source: Prepared with data from Valkiūnas (2005, Personal communications to D. J. Forrester, June 29, 2007, September 4, 2007, and October 22, 2007).

[*] *Leucocytozoon simondi* is also pathogenic to domestic waterfowl.

[†] This species is mildly pathogenic.

(Forrester and Spalding 2003). During that time, it was not protected from arthropod vectors and was debilitated due to aspergillosis. The immunocompromised condition of the bird may have made it susceptible to infection by a leucocytozoid from other birds in the rehabilitation facility or from free-ranging birds in the immediate area. Similarly, infections with *Leucocytozoon struthionis* have been found only in chicks of ostriches (*Struthio camelus*) up to 7 weeks of age and never in adult birds. Both the low number and morphologic condition of the gametocytes were suggestive of abnormal infections (Walker 1913; Bennett et al. 1992d) that were "remarkably similar" in form and dimensions to *Leucocytozoon schoutedeni*, a common parasite of chickens in the area.

Leucocytozoonosis occurs primarily in members of the Anatidae (ducks, geese, and swans) (Table 4.4) and Columbidae (pigeons and doves) (Table 4.5), and less commonly in members of the Accipitridae (hawks, eagles, and kites) and Falconidae (falcons and caracaras) (Table 4.6). *Leucocytozoon simondi*, the most important pathogenic leucocytozoid in wild birds, particularly in the Holarctic, has been reported in 46 species of waterfowl from 17 countries (Table 4.4).

ETIOLOGY

Species of *Leucocytozoon* are parasitic protozoans and are classified within the phylum Apicomplexa (Levine, 1970), class Coccidea (Leuckart, 1879), subclass Coccidia (Leuckart, 1879), order Haemosporida (Danilewsky, 1885), and family Leucocytozoidae Fallis and Bennett, 1961. In birds, there is one genus in the family (*Leucocytozoon* Berestneff, 1904) that is divided into two subgenera (*Leucocytozoon* Berestneff, 1904 and *Akiba* Bennett, Garnham and Fallis, 1965).

Historically, the description of leucocytozoid species has been based mainly on the morphology of gametocytes in blood cells, although the examination of exoerythrocytic stages (i.e., meronts or schizonts) has been used to some extent. There are a number of problems associated with this practice (Valkiūnas 2005): (1) intensity of infection in wild birds is low, and prolonged microscopic searches of stained blood films are necessary to find adequate numbers of infected blood cells; (2) gametocytes are often deformed when blood films are prepared if appropriate precautions are not taken; (3) there are fewer morphologic characteristics available to use for leucocytozoids compared to other blood protozoa, and often the morphology of

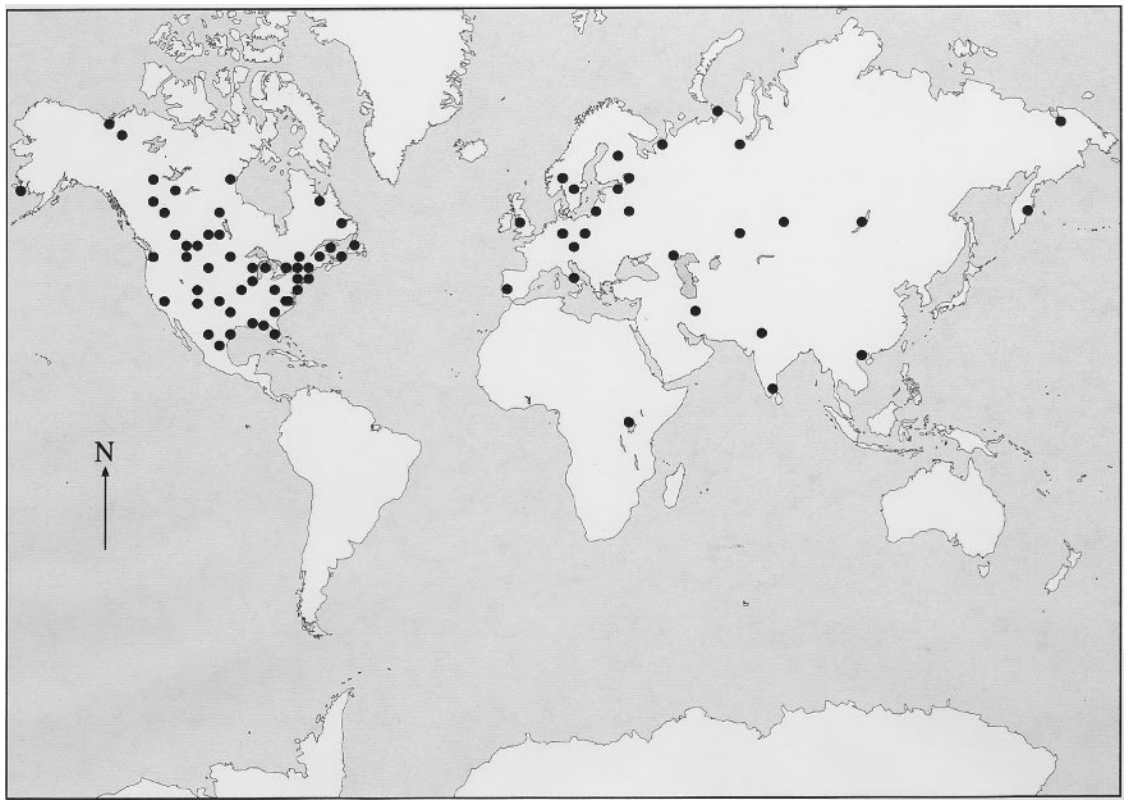


Figure 4.1. Distribution of *Leucocytozoon simondi* throughout the world. Solid circles indicate areas where infections were reported in either domestic, captive, or free-ranging wild waterfowl. It should be noted that no transmission has been reported from Africa, South Asia, and Mexico and that the records from these areas are from overwintering migratory Holarctic anatids. The data on which this figure is based are from Kučera (1981a), Valkiūnas (Personal communications to D. J. Forrester, June 29, 2007, September 4, 2007, and October 22, 2007), and the references given in Table 4.4.

the host cell is more important than the morphology of the parasite; (4) most morphologic features overlap among various species and must be used carefully in species descriptions; and (5) the use of the morphology of meronts for taxonomic purposes is not always valid since these are unknown for most species, and some species (*Leucocytozoon simondi*, for example) produce meronts that are quite different depending on the avian host.

Over time, several points of view on the host specificity of avian leucocytozoids have developed (Valkiūnas 2005). Early workers described new species on the basis of "a new host equals a new species," and this resulted in the proliferation of a large number of named species, many of which had either minor or no morphometric differences. A limited number of experimental attempts to transmit leucocytozoids to "abnormal" avian hosts (i.e., hosts of another avian family) via sporozoites from appropriate simuliid vectors eventually led to the idea that leucocytozoids are specific to host family. These attempts have been summarized by Bennett et al. (1991c). A more critical review of the literature indicates, however, that (1) three of the transmission attempts cited were not from published papers, but rather were "personal communications" and are of doubtful scientific value since details are not available; (2) one of the citations (Fallis and Bennett 1958) was incorrect; that is, it contained no information on the attempted transfer of *Leucocytozoon bonasae* from grouse to ducks and sparrows, rather they transferred *L. bonasae* from grouse to grouse; (3) the statement that Fallis et al. (1973) failed to transfer *L. schoutedeni* from chickens to guinea fowl and *Leucocytozoon neavei* from guinea fowl to chickens was

Table 4.3. Distribution of species of *Leucocytozoon* by orders and families of birds.

Avian order	Avian family	*Leucocytozoon* species
Struthioniformes	Struthionidae (Ostriches)	*Leucocytozoon struthionis*
Tinamiformes	—	—
Sphenisciformes	Spheniscidae (Penguins)	*Leucocytozoon tawaki*
Gaviiformes	Gaviidae (Loons)	*Leucocytozoon* sp*
Podicipediformes	—	—
Procellariiformes	—	—
Pelecaniformes	Anhingidae (Anhingas)	*Leucocytozoon vandenbrandeni*
	Phalacrocoracidae (Cormorants)	
Ciconiiformes	Ardeidae (Herons, Egrets, and Bitterns)	*Leucocytozoon leboeufi*
	Ciconiidae (Storks)	
	Threskiornithidae (Ibises and Spoonbills)	
	Ardeidae (Herons, Egrets, and Bitterns)	*Leucocytozoon nycticoraxi*
	Balaenicipitidae (Shoebills)	*Leucocytozoon* sp.
Phoenicopteriformes	—	—
Anseriformes	Anatidae (Ducks, Geese, and Swans)	*Leucocytozoon simondi*
Falconiformes	Accipitridae (Hawks, Eagles, and Kites)	*Leucocytozoon toddi*
	Cathartidae (New World Vultures)	
	Falconidae (Falcons and Caracaras)	
	Pandionidae (Ospreys)	
	Sagittariidae (Secretary birds)	
Galliformes	Meleagrididae (Turkeys)	*Leucocytozoon smithi*
	Cracidae (Guans, Chachalacas, and Curassows)	*Leucocytozoon* sp.
	Numididae (Guineafowl)	*Leucocytozoon neavei*
	Tetraonidae (Grouse, Ptarmigans, and Prairie Chickens)	*Leucocytozoon lovati*
	Phasianidae (Pheasants and Partridges)	*Leucocytozoon cheissini*
		Leucocytozoon macleani
		Leucocytozoon caulleryi
		Leucocytozoon schoutedeni
Opisthocomiformes	—	—
Gruiformes	Gruidae (Cranes)	*Leucocytozoon grusi*
	Otididae (Bustards)	*Leucocytozoon* sp.
	Rallidae (Rails, Gallinules, and Coots)	
Charadriiformes	Charadriidae (Plovers and Lapwings)	*Leucocytozoon legeri*
	Scolopacidae (Sandpipers)	
	Charadriidae (Plovers and Lapwings)	*Leucocytozoon sousadiasi*
	Jacanidae (Jacanas)	*Leucocytozoon* sp.
	Recurvirostridae (Avocets and Stilts)	
	Rostratulidae (Painted snipes)	
	Sternidae (Terns)	
Pterocliformes	—	—
Columbiformes	Columbidae (Pigeons and Doves)	*Leucocytozoon marchouxi*
Psittaciformes	Psittacidae (Parrots)	*Leucocytozoon* sp.
	Cacatuidae (Cockatoos)	
Musophagiformes	Musophagidae (Turacos)	*Leucocytozoon dizini*
Cuculiformes	Cuculidae (Cuckoos)	*Leucocytozoon centropi*
Strigiformes	Strigidae (Owls)	*Leucocytozoon danilewskyi*
	Tytonidae (Barn Owls)	
Caprimulgiformes	Caprimulgidae (Nightjars)	*Leucocytozoon caprimulgi*
	Podargidae (Frogmouths)	
Apodiformes	Trochilidae (Hummingbirds)	*Leucocytozoon* sp.
Coliiformes	Coliidae (Mousebirds)	*Leucocytozoon colius*
Trogoniformes	Trogonidae (Trogons and Quetzals)	*Leucocytozoon* sp.

(*continues*)

Table 4.3. (*Continued*)

Avian order	Avian family	*Leucocytozoon* species
Coraciiformes	Coraciidae (Rollers)	*Leucocytozoon bennetti*
	Bucerotidae (Hornbills)	*Leucocytozoon communis*
	Upupidae (Hoopoes)[†]	
	Coraciidae (Rollers)	*Leucocytozoon eurystomi*
	Momotidae (Motmots)	*Leucocytozoon* sp.
	Alcedinidae (Kingfishers)	*Leucocytozoon communis*
		Leucocytozoon eurystomi
		Leucocytozoon nyctyornis
	Meropidae (Bee-eaters)	*Leucocytozoon eurystomi*
		Leucocytozoon nyctyornis
Piciformes	Capitonidae (Barbets)	*Leucocytozoon squamatus*
	Picidae (Woodpeckers)	
Passeriformes	Laniidae (Shrikes)	*Leucocytozoon balmorali*
	Malaconotidae (Bushshrikes)	
	Corvidae (Crows, Jays, and Magpies)	*Leucocytozoon berestneffi*
		Leucocytozoon sakharoffi
	Nectariniidae (Sunbirds and Spiderhunters)	*Leucocytozoon dubreuili*
	Zosteropidae (White-eyes)	
	Certhiidae (Creepers)	*Leucocytozoon fringillinarum*
	Emberizidae (Buntings and Sparrows)	
	Estrildidae (Waxbills)	
	Hirundinidae (Swallows)	
	Icteridae (Troupials)	
	Parulidae (New World Warblers)	
	Ploceidae (Weavers)	
	Prionopidae (Helmetshrikes)	
	Prunellidae (Accentors)	
	Thraupidae (Tanagers)	
	Tyrannidae (Tyrant Flycatchers)	
	Viduidae (Indigobirds)	
	Oriolidae (Old World Orioles)	*Leucocytozoon majoris*
	Paradoxornithidae (Parrotbills)	
	Passeridae (Old World Sparrows)	
	Pittidae (Pittas)	
	Alaudidae (Larks)	*Leucocytozoon fringillinarum*
		Leucocytozoon majoris
	Bombycillidae (Waxwings)	*Leucocytozoon fringillinarum*
		Leucocytozoon majoris
	Cardinalidae (Saltators and Cardinals)	*Leucocytozoon fringillinarum*
		Leucocytozoon majoris
	Chloropseidae (Leafbirds)	*Leucocytozoon dubreuili*
		Leucocytozoon fringillinarum
	Fringillidae (Siskins and Crossbills)	*Leucocytozoon dubreuili*
		Leucocytozoon fringillinarum
	Mimidae (Mockingbirds and Thrashers)	*Leucocytozoon dubreuili*
		Leucocytozoon fringillinarum
		Leucocytozoon majoris
	Motacillidae (Wagtails and Pipits)	*Leucocytozoon fringillinarum*
		Leucocytozoon majoris
	Muscicapidae (Old World Flycatchers)	*Leucocytozoon dubreuili*
		Leucocytozoon majoris

(*continues*)

Table 4.3. (*Continued*)

Avian order	Avian family	*Leucocytozoon* species
	Paridae (Chickadees and Tits)	*Leucocytozoon hamiltoni*
		Leucocytozoon majoris
	Pynonotidae (Bulbuls)	*Leucocytozoon fringillinarum*
		Leucocytozoon majoris
	Sturnidae (Starlings)	*Leucocytozoon fringillinarum*
		Leucocytozoon majoris
	Sylviidae (Old World Warblers)	*Leucocytozoon balmorali*
		Leucocytozoon dubreuili
		Leucocytozoon fringillinarum
		Leucocytozoon majoris
	Timaliidae (Babblers)	*Leucocytozoon fringillinarum*
		Leucocytozoon majoris
	Turdidae (Thrushes)	*Leucocytozoon dubreuili*
		Leucocytozoon fringillinarum
		Leucocytozoon maccluri
		Leucocytozoon majoris
	Vireonidae (Vireos)	*Leucocytozoon dubreuili*
		Leucocytozoon fringillinarum
		Leucocytozoon majoris
	Aegithinidae (Ioras)	*Leucocytozoon* sp.
	Bucconidae (Puffbirds)	
	Campephagidae (Cuckoo-shrikes)	
	Cinclidae (Dippers)	
	Climactreridae (Australian Treecreepers)	
	Corcoracidae (Choughs and Apostlebirds)	
	Cracticidae (Bellmagpies)	
	Dicaeidae (Flowerpickers)	
	Dicruridae (Drongos)	
	Eurylaimidae (Broadbills)	
	Furnariidae (Ovenbirds)	
	Grallinidae (Mudnest Builders)	
	Indicatoridae (Honeyguides)	
	Irenidae (Fairy-bluebirds)	
	Meliphagidae (Honeyeaters)	
	Paradisaeidae (birds of paradise)	
	Philepittidae (Asities)	
	Picathartidae (Rockfowl)	
	Pipridae (Manakins)	
	Ptilogonatidae (Silky-flycatchers)	
	Ptilonorhynchidae (Bowerbirds)	
	Regulidae (Kinglets)	
	Sittidae (Nuthatches)	
	Troglodytidae (Wrens)	
	Vangidae (Vangas)	

Source: Prepared from data presented by Bennett and Campbell (1975), Greiner and Kocan (1977), Bennett et al. (1982, 1992a), Bishop and Bennett (1992), Forrester et al. (1994), Super and van Riper (1995), Rintamäki et al. (1999), Adlard et al. (2002, 2004), Valkiūnas et al. (2002), Savage (2004), Savage et al. (2004, 2006a, b), Jones et al. (2005), Peirce et al. (2005), and Valkiūnas (2005).

*Identity of species uncertain.

†Some ornithologists consider the family Upupidae to be in another order (Upupiformes), but we follow Clements (2000) for the purposes of this analysis.

Table 4.4. Reports of *Leucocytozoon simondi* in wild waterfowl.

Common name	Species	Location	Number of examinations	Prevalence (%)	Literature source
Mallard	*Anas platyrhynchos*	Belarus	U	U	Dyl'ko (1966)
		Canada (Alberta)	51	27	Williams et al. (1977)
		Canada (Alberta and Saskatchewan)	2,667	19	Bennett et al. (1982a)
		Canada (Labrador)	33	18	Bennett et al. (1991b)
		Canada (Manitoba and Saskatchewan)	85	<1	Burgess (1957)
		Canada (Maritime Provinces)	23	13	Bennett et al. (1975)
		Canada (Newfoundland)	—*	—*	IRCAH records
		Canada (Northwest Territories)	—	—	Williams et al. (1977)
		Canada (Nova Scotia)	—	—	IRCAH records
		Canada (Ontario)	—	—	Karstad (1965)
		Canada (Quebec)	—	—	IRCAH records
		Czechoslovakia	—	—	IRCAH records
		Germany	~50	NG	Böing (1925)
		Lithuania	19	26	Valkiūnas (1985)
		Lithuania	46	15	Valkiūnas et al. (1990)
		Mexico (Coahuila)	10	20	Bennett et al. (1991a)
		Norway (Rendalen)	—	—	Eide et al. (1969)
		Portugal	U	U	França (1912)
		Kazakhstan	27	4	Yakunin and Zhazyltaev (1977)
		Russia (Salechard)	—	—	Valkiūnas et al. (1990)
		Russia (Tomsk)	37	57	Valkiūnas†
		Russia (Volga River Delta)	17	41	Valkiūnas†
		USA (California)	15	13	Wood and Herman (1943)
		USA (California)	368	<1	Herman (1951)
		USA (Colorado)	110	10	Stabler et al. (1975)
		USA (Maryland)	59	2	Wetmore (1941)
		USA (Massachusetts)	624	15	Bennett et al. (1974a)
		USA (Michigan)	220	4	DeJong and Muzzall (2000)
		USA (Minnesota)	—	—	Green et al. (1938)
		USA (Ohio)	—	—	Al-Dabagh (1964)
		USA (Oklahoma)	402	9	Kocan et al. (1979)
		USA (South Dakota)	169	28	Polcyn and Johnson (1968)
		USA (Washington)	837	24	Clark (1980)
		USA (Wisconsin)	174	62	Trainer et al. (1962)
		USA (Wisconsin)	208	1	Bradshaw and Trainer (1966)

Northern Pintail	*Anas acuta*	Canada (Alberta)	49	18	Williams et al. (1977)
		Canada (Alberta & Saskatchewan)	505	11	Bennett et al. (1982a)
		Canada (Labrador)	14	35	Bennett et al. (1991b)
		Canada (Manitoba & Saskatchewan)	138	<1	Burgess (1957)
		Canada (Maritime Provinces)	228	18	Bennett et al. (1975)
		Canada (New Brunswick)	—	—	IRCAH records
		Canada (Northwest Territories)	18	4	Williams et al. (1977)
		Canada (Nova Scotia)	—	—	IRCAH records
		Canada (Prince Edward Island)	—	—	IRCAH records
		Canada (Quebec)	—	—	Laird and Bennett (1970)
		India (Rajasthan)	66	6	McClure et al. (1978)
		Russia (Chaun River Delta)	26	58	Valkiūnas et al. (1990)
		Russia (Salechard)	48	83	Valkiūnas et al. (1990)
		Russia (Kliuchi, Kamchatka)	22	45	Valkiūnas et al. (1990)
		Russia (Ust-Kara)	—	—	Valkiūnas et al. (1990)
		USA (California)	24	29	Wood and Herman (1943)
		USA (California)	263	1	Herman (1951)
		USA (Colorado)	68	16	Stabler et al. (1975)
		USA (Louisiana)	—	—	O'Roke (1934)
		USA (Maryland)	—	—	Wetmore (1941)
		USA (Wisconsin)	—	—	Trainer et al. (1962)
Garganey	*Anas querquedula*	Germany	NG	—	Böing (1925)
		India (Rajasthan)	34	3	McClure et al. (1978)
		Iran (Dashti Arjan)	—	—	IRCAH records
		Italy	—	—	Peirce (1981)
		Kazakhstan	10	20	Yakunin and Zhazyltaev (1977)
		Russia (Volga River Delta)	—	—	Valkiūnas[†]
		Russia (Salechard)	60	87	Valkiūnas et al. (1990)
Northern Shoveler	*Anas clypeata*	Canada (Alberta and Saskatchewan)	10	10	Bennett et al. (1982a)
		Russia (Kliuchi, Kamchatka)	—	—	Valkiūnas et al. (1990)
		Russia (Salechard)	—	—	Valkiūnas et al. (1990)
		USA (California)	55	11	Herman (1951)
		USA (Colorado)	—	—	Stabler et al. (1975)
		USA (Florida)	—	—	Forrester and Spalding (2003)

(*continues*)

Table 4.4. (*Continued*)

Common name	Species	Location	Number of examinations	Prevalence (%)	Literature source
Blue-winged Teal	*Anas discors*	Canada (Alberta)	—	—	IRCAH records
		Canada (Labrador)	—	—	Bennett et al. (1991b)
		Canada (Manitoba)	—	—	IRCAH records
		Canada (Maritime Provinces)	1,286	4	Bennett et al. (1975)
		Canada (New Brunswick)	—	—	IRCAH records
		Canada (Nova Scotia)	—	—	IRCAH records
		Canada (Prince Edward Island)	—	—	IRCAH records
		Canada (Alberta and Saskatchewan)	446	6	Bennett et al. (1982a)
		USA (Colorado)	39	18	Stabler et al. (1975)
		USA (Massachusetts)	87	14	Bennett et al. (1974)
		USA (Oklahoma)	58	5	Kocan et al. (1979)
		USA (Texas)	314	4	Loven et al. (1980)
Eurasian Teal	*Anas crecca*	Canada (Alberta and Saskatchewan)	119	38	Bennett et al. (1982a)
		Canada (Labrador)	73	82	Bennett et al. (1991b)
		Canada (Manitoba and Saskatchewan)	25	8	Burgess (1957)
		Canada (Maritime Provinces)	387	17	Bennett et al. (1975)
		Canada (New Brunswick)	—	—	IRCAH records
		Canada (Newfoundland)	—	—	IRCAH records
		Canada (Nova Scotia)	—	—	IRCAH records
		Canada (Quebec)	—	—	Laird and Bennett (1970)
		India (Rajasthan)	75	5	McClure et al. (1978)
		Iran	13	8	McClure et al. (1978)
		Norway (Rendalen)	—	—	Eide et al. (1969)
		Russia (Chaun River Delta)	—	—	Valkiūnas et al. (1990)
		Russia (Klyuchi, Kamchatka)	34	47	Valkiūnas et al. (1990)
		Russia (Salechard)	—	—	Valkiūnas et al. (1990)
		Russia (Volga River Delta)	—	—	Valkiūnas[†]
		USA (Colorado)	35	54	Stabler et al. (1975)
		USA (Maine)	—	—	Nelson and Gashwiler (1941)
		USA (Massachusetts)	87	23	Bennett et al. (1974a)
		USA (Oklahoma)	49	18	Kocan et al. (1979)
		USA (Texas)	89	7	Fedynich et al. (1993)

American Wigeon	*Anas americana*	Canada (Alberta and Saskatchewan)	28	18	Bennett et al. (1982a)
		Canada (Maritime Provinces)	180	2	Bennett et al. (1975)
		Canada (New Brunswick)	—	—	IRCAH records
		USA (California)	43	5	Herman (1951)
		USA (Colorado)	40	23	Stabler et al. (1975)
		USA (Maryland)	—	—	Wetmore (1941)
		USA (Oklahoma)	104	11	Kocan et al. (1979)
		USA (Texas)	64	5	Fedynich et al. (1993)
Gadwall	*Anas strepera*	Canada (Alberta and Saskatchewan)	24	4	Bennett et al. (1982a)
		USA (Colorado)	16	25	Stabler et al. (1975)
		USA (Florida)	—	—	Forrester and Spalding (2003)
American Black Duck	*Anas rubripes*	Canada (Labrador)	20	25	Bennett (1972)
		Canada (Labrador)	382	71	Bennett et al. (1991b)
		Canada (Maritime Provinces)	1,750	23	Bennett et al. (1975)
		Canada (New Brunswick)	—	—	IRCAH records
		Canada (Newfoundland)	—	—	IRCAH records
		Canada (Nova Scotia)	—	—	IRCAH records
		Canada (Ontario)	—	—	Clarke (1946)
		Canada (Prince Edward Island)	—	—	IRCAH records
		Canada (Quebec)	—	—	Laird and Bennett (1970)
		USA (Maine)	408	75	O'Meara (1956)
		USA (Maine)	29	89	Nelson and Gashwiler (1941)
		USA (Maryland)	89	7	Williams and Bennett (1978)
		USA (Massachusetts)	85	13	Herman (1938)
		USA (Massachusetts)	203	28	Bennett et al. (1974a)
		USA (Michigan)	—	—	DeJong and Muzzall (2000)
		USA (New York)	—	—	Reilly (1956)
		USA (Nebraska)	—	—	IRCAH records
		USA (Ohio)	13	38	Al-Dabagh (1964)
		USA (Washington, DC)	—	—	IRCAH records
		USA (Wisconsin)	—	—	Trainer et al. (1962)
Falcated Duck	*Anas falcata*	Russia (Kliuchi, Kamchatka)	23	48	Valkiūnas et al. (1990)
Baikal Teal	*Anas formosa*	Russia (Kliuchi, Kamchatka)	—	—	Valkiūnas et al. (1990)
Eurasian Wigeon	*Anas penelope*	India (Rajasthan and Tamil Nadu)	44	11	McClure et al. (1978)

(*continues*)

Table 4.4. (*Continued*)

Common name	Species	Location	Number of examinations	Prevalence (%)	Literature source
		Russia (Kliuchi, Kamchatka)	—	—	Valkiūnas et al. (1990)
		Russia (Salechard)	51	92	Valkiūnas et al. (1990)
Greater Scaup	*Aythya marila*	Canada (Labrador)	—	—	Bennett et al. (1991b)
		Russia (Kliuchi, Kamchatka)	63	35	Valkiūnas et al. (1990)
		Russia (Salechard)	—	—	Valkiūnas et al. (1990)
		Russia (Ust-Kara)	—	—	Valkiūnas et al. (1990)
Lesser Scaup	*Aythya affinis*	Canada (Northwest Territories)	—	—	IRCAH records
		USA (Colorado)	39	8	Stabler et al. (1975)
		USA (Texas)	180	36	Loven et al. (1980)
Redhead	*Aythya americana*	Canada (Alberta and Saskatchewan)	23	7	Bennett et al. (1982a)
		USA (California)	17	12	Wood and Herman (1943)
		USA (Colorado)	13	23	Stabler et al. (1975)
Canvasback	*Aythya valisineria*	USA (Louisiana)	—	—	O'Roke (1934)
		USA (Maryland)	88	6	Kocan and Knisley (1970)
		USA (Washington, DC)	—	—	IRCAH records
Ring-necked Duck	*Aythya collaris*	Canada (Maritime Provinces)	178	4	Bennett et al. (1975)
		Canada (New Brunswick)	—	—	IRCAH records
		Canada (Prince Edward Island)	—	—	IRCAH records
		USA (Florida)	283	9	Forrester et al. (2001b)
		USA (Louisiana)	—	—	O'Roke (1934)
Common Pochard	*Aythya ferina*	Kazakhstan	—	—	Yakunin and Zhazyltaev (1977)
Tufted Duck	*Aythya fuligula*	India (Rajasthan)	26	8	McClure et al. (1978)
		Macedonia	—	—	Wülker (1919)
		Russia (Kliuchi, Kamchatka)	—	—	Valkiūnas et al. (1990)
		Russia (Salechard)	40	88	Valkiūnas et al. (1990)
Red-crested Pochard	*Netta rufina*	Kazakhstan	—	—	Yakunin and Zhazyltaev (1977)
Long-tailed Duck	*Clangula hyemalis*	North America	—	—	Herman (1963)
		Russia (Chaun River Delta)	15	53	Valkiūnas et al. (1990)
Common Merganser	*Mergus merganser*	Canada (Labrador)	—	—	Bennett et al. (1991b)
		Canada (Ontario)	—	—	Fallis et al. (1954)
		USA (Colorado)	21	5	Stabler et al. (1975)
		USA (Maine)	—	—	Nelson and Gashwiler (1941)
		USA (Michigan)	55	47	DeJong et al. (2001)

Red-breasted Merganser	*Mergus serrator*	Canada (Quebec)	—	—	Laird and Bennett (1970)
		USA (California)	—	—	Wood and Herman (1943)
		USA (Massachusetts)	—	—	IRCAH records
Smew	*Mergellus albellus*	Russia (Salechard)	10	20	Valkiūnas et al. (1990)
Hooded Merganser	*Lophodytes cucullatus*	Canada (Quebec)	—	—	Laird and Bennett (1970)
		USA (Maine)	—	—	Nelson and Gashwiler (1941)
		USA (Massachusetts)	11	9	Bennett et al. (1974a)
		USA (Texas)	20	10	Loven et al. (1980)
Surf Scoter	*Melanitta perspicillata*	Canada (Labrador)	—	—	Bennett (1972)
Black Scoter	*Melanitta nigra*	Canada (Labrador)	—	—	IRCAH records
		USA (North Carolina)	—	—	NWHC records
Common Goldeneye	*Bucephala clangula*	Canada (Maritime Provinces)	—	—	Bennett et al. (1975)
		Canada (New Brunswick)	—	—	IRCAH records
		USA (Maine)	—	—	Nelson and Gashwiler (1941)
Spectacled Eider	*Somateria fischeri*	Russia (Chaun River Delta)	16	44	Valkiūnas et al. (1990)
Common Eider	*Somateria mollissima*	Canada (Labrador)	18	<1	Bennett (1972)
		Canada (Newfoundland)	126	<1	Bennett and Inder (1972)
		Russia (Ust-Kara)	—	—	Valkiūnas et al. (1990)
Wood Duck	*Aix sponsa*	Canada (Maritime Provinces)	51	14	Bennett et al. (1975)
		Canada (New Brunswick)	—	—	IRCAH records
		Canada (Nova Scotia)	—	—	IRCAH records
		Canada (Ontario)	66	16	Thul and O'Brien (1990)
		USA (Connecticut)	79	4	Thul and O'Brien (1990)
		USA (Florida)	2,143	2	Thul and O'Brien (1990)
		USA (Georgia)	729	2	Thul and O'Brien (1990)
		USA (Maine)	77	59	Nelson and Gashwiler (1941)
		USA (Maine)	322	51	O'Meara (1956)
		USA (Maine)	13	69	Herman et al. (1971)
		USA (Maine)	24	83	Thul et al. (1980)
		USA (Maine)	944	70	Thul and O'Brien (1990)
		USA (Maryland)	11	9	Thul et al. (1980)
		USA (Maryland)	93	1	Thul and O'Brien (1990)
		USA (Missouri)	371	1	Odell and Robbins (1994)

(*continues*)

Table 4.4. (*Continued*)

Common name	Species	Location	Number of examinations	Prevalence (%)	Literature source
		USA (Massachusetts)	230	<1	Herman et al. (1971)
		USA (Massachusetts)	730	33	Bennett et al. (1974a)
		USA (Massachusetts)	1,066	5	Thul and O'Brien (1990)
		USA (New Hampshire)	46	26	Thul and O'Brien (1990)
		USA (New York)	721	4	Thul and O'Brien (1990)
		USA (North Carolina)	912	2	Thul and O'Brien (1990)
		USA (Ohio)	35	3	Herman et al. (1971)
		USA (Pennsylvania)	10	10	Thul et al. (1980)
		USA (Pennsylvania)	840	1	Thul and O'Brien (1990)
		USA (Rhode Island)	26	8	Thul and O'Brien (1990)
		USA (South Carolina)	608	3	Thul and O'Brien (1990)
		USA (Vermont)	225	5	Thul and O'Brien (1990)
		USA (Virginia)	1,418	2	Thul and O'Brien (1990)
		USA (West Virginia)	217	5	Thul and O'Brien (1990)
		USA (Wisconsin)	40	8	Trainer et al. (1962)
Mandarin Duck	*Aix galericulata*	Northeastern Asia	U	U	Valkiūnas (2005)
Ruddy Duck	*Oxyura jamaicensis*	USA (Maryland)	—	—	Williams and Bennett (1978)
Trumpeter Swan	*Cygnus buccinator*	Canada (Alberta)	75	1	Bennett et al. (1981b)
		Canada (Northwest Territories)	38	21	Bennett et al. (1992b)
Tundra Swan	*Cygnus columbianus*	Canada (Northwest Territories)	—	—	IRCAH records
		USA (Alaska)	—	—	IRCAH records
		USA (Michigan)	—	—	IRCAH records
		USA (Utah)	—	—	IRCAH records
Mute Swan	*Cygnus olor*	Sweden	62	16	Mörner and Wahlström (1983)
		USA (New Hampshire)	—	—	IRCAH records
Canada Goose	*Branta canadensis*	Canada (Labrador)	—	—	Bennett (1972)
		Canada (Maritime Provinces)	66	2	Bennett et al. (1975)
		Canada (New Brunswick)	—	—	IRCAH records

		Canada (Quebec)	—	—	Laird and Bennett (1970)
		USA (Illinois)	353	9	Levine and Hanson (1953)
		USA (Maine)	—	—	IRCAH records
		USA (Massachusetts)	31	3	Herman (1938)
		USA (Michigan)	77	4	DeJong and Muzzall (2000)
		USA (Oklahoma)	98	1	Kocan et al. (1979)
		USA (Wisconsin)	—	—	Trainer et al. (1962)
		USA (Wisconsin)	175	2	Bradshaw and Trainer (1966)
Cackling Goose	*Branta hutchinsii*	USA (California)	—	—	Wood and Herman (1943)
Brant	*Branta bernicla*	Canada (Northwest Territories)	—	—	IRCAH records
Egyptian Goose	*Alopochen aegyptiaca*	Uganda	—	—	Minchin (1910)
Snow Goose	*Chen caerulescens*	Canada (Northwest Territories)	570	4	Bennett and MacInnes (1972)
		Canada (Alberta and Saskatchewan)	—	—	Bennett et al. (1982a)
Emperor Goose	*Chen canagica*	USA (Alaska)	134	1	Hollmen et al. (1998)
Greater White-fronted Goose	*Anser albifrons*	Canada (Northwest Territories)	—	—	IRCAH records
		USA (California)	—	—	Wood and Herman (1943)
		USA (Texas)	46	4	Kloss et al. (2003)
Graylag Goose	*Anser anser*	Canada (New Brunswick)	—	—	IRCAH records
		Canada (Quebec)	—	—	IRCAH records
		Germany	~ 200	NG	Böing (1925)
		Kazakhstan	42	7	Yakunin and Zhazyltaev (1977)
		Portugal	U	U	França (1912)
Swan Goose	*Anser cygnoides*	North Central Asia	U	U	Valkiūnas (2005)
Taiga Bean-Goose	*Anser fabalis*	Russia (Salechard)	—	—	Valkiūnas et al. (1990)

IRCAH, International Reference Centre for Avian Haematozoa, Queensland Museum, South Brisbane, Queensland, Australia; NWHC, National Wildlife Health Center, U.S. Geological Survey, Madison, WI, USA; NG, not given by author; U, values not known: either not given by author or we were unable to obtain the reference.

*Number of birds examined was less than 10 and therefore the prevalence was not calculated.

†Personal communications to D. J. Forrester, June 29, 2007, September 4, 2007, and October 22, 2007.

Table 4.5. Reports of *Leucocytozoon marchouxi* in wild pigeons and doves.

Common name	Species	Location	Number of examinations	Prevalence (%)	Literature source
Rock Pigeon	*Columba livia*	Australia	27	4	Adlard et al. (2004)
		Azerbaijan	U	U	Zeiniev (1975)
		Belarus	U	U	Dyl'ko (1966)
		England	—	—	Coles (1914)
		Georgia	U	U	Burtikashvili (1978)
		Iraq	12	8	Shamsuddin and Mohammad (1980)
		Kazakhstan	—*	—*	Yakunin and Zhazyltaev (1977)
		South Africa	33	3	Earlé and Little (1993)
		Tajikistan	75	1	Shakhmatov et al. (1972)
		Turkey	16	31	Ozmen et al. (2005)
		Turkmenistan	—	—	Berdyev (1979)
		USA (Colorado)	86	1	Stabler and Holt (1963)
Speckled Pigeon	*Columba guinea*	Nigeria	—	—	Cowper (1969)
		South Africa	11	9	Thomas and Dobson (1975)
		South Africa	50	2	Earlé and Little (1993)
Band-tailed Pigeon	*Patagioenas fasciata*	USA (California)	105	30	Stabler et al. (1977)
		USA (Colorado)	109	36	Stabler and Holt (1963)
		USA (Colorado)	364	65	Stabler et al. (1977)
		Mexico	—	—	Stabler et al. (1977)
Common Wood-Pigeon	*Columba palumbus*	England	109	15	Baker (1974)
		England	22	5	Peirce (1980)
		Germany	128	30	Böing (1925)
		Kazakhstan	19	32	Yakunin and Zhazyltaev (1977)
		Morocco	10	30	Gaud and Petitot (1945)
		Poland	—	—	Ramisz (1962)
Rameron Pigeon	*Columba arquatrix*	Uganda	—	—	Valkiūnas et al. (2005)
Stock Dove	*Columba oenas*	Kazakhstan	337	12	Yakunin and Zhazyltaev (1977)
Pale-backed Pigeon	*Columba eversmanni*	Kazakhstan	301	5	Yakunin and Zhazyltaev (1977)
		Kazakhstan (Tyan-Shan)	—	—	Kairullaev and Yakunin (1982)
Lemon Dove	*Columba larvata*	Ethiopia	18	11	Ashford et al. (1976)
Metallic Pigeon	*Columba vitiensis*	NG	U	U	Berson (1964)
Pink Pigeon	*Nesoenas mayeri*	Mauritius	313	29	Swinnerton et al. (2005)
			328	18	Bunbury et al. (2007)
Crested Pigeon	*Geophaps lophotes*	Australia	127	1	Adlard et al. (2004)

White-eared Dove	*Phapitreron leucotis*	Philippines	79	1	McClure et al. (1978)
Wedge-tailed Pigeon	*Treron sphenurus*	India	—	—	Ray (1952)
White-bellied Pigeon	*Treron sieboldii*	Japan	—	—	Murata (2002)
Madagascar Green-Pigeon	*Treron australis*	Nigeria	—	—	Cowper (1969)
Pink-necked Pigeon	*Treron vernans*	Philippines	15	7	McClure et al. (1978)
Wedge-tailed Pigeon	*Treron sphenurus*	NG	U	U	Berson (1964)
Spotted Imperial-Pigeon	*Ducula carola*	Philippines	—	—	McClure et al. (1978)
Mourning Dove	*Zenaida macroura*	USA (Arizona)	—	—	Wood and Herman (1943)
		USA (District of Columbia)	—	—	Wetmore (1941)
		USA (California)	—	—	Wood and Herman (1943)
		USA (Colorado)	269	14	Stabler and Holt (1963)
		USA (Florida)	918	<1	Shamis and Forrester (1977)
		USA (Georgia)	—	—	Thompson (1943)
		USA (Illinois)	464	2	Hanson et al. (1957)
		USA (Iowa)	41	15	Farmer (1960)
		USA (Maryland)	227	1	Williams and Bennett (1978)
		USA (New Jersey)	119	4	Huffman and Cali (1983)
		USA (New Mexico)	339	15	Gutiérrez (1973)
		USA (Ohio)	102	3	Al-Dabagh (1964)
		USA (Vermont)	27	19	Barnard and Bair (1986)
White-winged Dove	*Zenaida asiatica*	Mexico	72	3	Saunders (1959)
Ruddy Ground-Dove	*Columbina talpacoti*	NG	U	U	Berson (1964)
Red-eyed Dove	*Streptopelia semitorquata*	Ethiopia	23	9	Ashford et al. (1976)
		Kenya	—	—	Bennett and Herman (1976)
		Mauritius	60	8	Swinnerton et al. (2005)
		Uganda	—	—	Minchin (1910)
Spotted Dove	*Streptopelia chinensis*	Japan	111	5	Ogawa (1912)
		Mauritius	—	—	Swinnerton et al. (2005)
		Philippines	30	7	McClure et al. (1978)
		USA (California)[†]	25	4	Wood and Herman (1943)

(*continues*)

Table 4.5. (*Continued*)

Common name	Species	Location	Number of examinations	Prevalence (%)	Literature source
Laughing Dove	*Streptopelia senegalensis*	Ethiopia	22	5	Ashford et al. (1976)
		India	—	—	McClure et al. (1978)
		Kenya	—	—	Peirce et al. (1977a)
		Senegal	—	—	Leger and Leger (1914)
		South Africa	40	38	Earlé et al. (1991)
Eurasian Turtle-Dove	*Streptopelia turtur*	Corsica	—	—	Leger (1913)
		Germany	—	—	Böing (1925)
		Georgia	U	U	Burtikashvili (1978)
		Iraq	—	—	Shamsuddin and Mohammad (1980)
		Italy	U	U	Franchini (1924)
		Kazakhstan	194	9	Yakunin and Zhazyltaev (1977)
		Kazakhstan (Tyan-Shan)	33	19	Kairullaev and Yakunin (1982)
		Kuwait	19	11	Mohammed and Al-Taqi (1975)
		Morocco	25	12	Gaud and Petitot (1945)
		Spain	U	U	Covaleda Ortega and Gállego Berenguer (1946)
		Uzbekistan	U	U	Ulugzadaev and Abidzhanov (1975)
Oriental Turtle-Dove	*Streptopelia orientalis*	Kazakhstan	458	14	Yakunin and Zhazyltaev (1977)
		Kazakhstan (Tyan-Shan)	22	5	Kairullaev and Yakunin (1982)
		India	—	—	McClure et al. (1978)
		Japan	U	U	Ogawa (1912)
		Korea	—	—	McClure et al. (1978)

Ring-necked Dove	*Streptopelia capicola*	South Africa	—	—	Jansen (1952)
African Mourning Dove	*Streptopelia decipiens*	Ethiopia	51	4	Ashford et al. (1976)
Dusky Turtle-Dove	*Streptopelia lugens*	Kenya	—	—	Bennett and Herman (1976)
Red Collared-Dove	*Streptopelia tranquebarica*	Vietnam	U	U	Mathis and Leger (1910)
Island Collared-Dove	*Streptopelia bitorquata*	Philippines	235	<1	McClure et al. (1978)
Eurasian Collared-Dove	*Streptopelia decaocto*	India	15	7	McClure et al. (1978)
Little Cuckoo-Dove	*Macropygia ruficeps*	Malaysia	—	—	McClure et al. (1978)
Namaqua Dove	*Oena capensis*	South Africa	—	—	Jansen (1952)
Bar-shouldered Dove	*Geopelia humeralis*	Australia	—	—	Reece et al. (1992)
Zebra Dove	*Geopelia striata*	Mauritius	13	8	Swinnerton et al. (2005)
		Mauritius	17	12	Peirce et al. (1977b)
		Philippines	178	<1	McClure et al. (1978)
Emerald-spotted Wood-Dove	*Turtur chalcospilos*	Kenya	—	—	Peirce et al. (1977a)
		Zambia	—	—	Peirce (1984)
Tambourine Dove	*Turtur tympanistria*	Ethiopia	22	5	Ashford et al. (1976)
		Tanzania	—	—	Bennett and Herman (1976)
		Uganda	15	7	Bennett et al. (1974b)
Blue-spotted Wood-Dove	*Turtur afer*	Ethiopia	44	2	Ashford et al. (1976)
Black-billed Wood-Dove	*Turtur abyssinicus*	Ethiopia	—	—	Ashford et al. (1976)

NG, not given by author; U, values not known: either not given by author or we were unable to obtain the reference.
*Number of birds examined was less than 10 and therefore the prevalence was not calculated.
†Nonendemic; introduced into California where it now breeds.

Table 4.6. Reports of *Leucocytozoon toddi* in wild falconiforms.

Common name	Species	Location	Number of examinations	Prevalence (%)	Literature source
		Accipitridae			
Hooded Vulture	*Necrosyrtes monachus*	Gambia	34	6	Todd and Wolbach (1912)
White-backed Vulture	*Gyps africanus*	South Africa	35	3	Greiner and Mundy (1979)
		Zimbabwe	330	<1	Greiner and Mundy (1979)
Lappet-faced Vulture	*Torgos tracheliotus*	Zimbabwe	—*	—*	Greiner and Mundy (1979)
Short-toed Eagle	*Circaetus gallicus*	Algeria	—	—	Sergent and Fabiani (1922)
Black-shouldered Kite	*Elanus caeruleus*	Ethiopia	—	—	Ashford et al. (1976)
		Philippines	—	—	McClure et al. (1978)
		India	—	—	Nandi and Mandal (1984)
		South Africa	U	U	Enigk (1942)
Eurasian Buzzard	*Buteo buteo*	Czech Republic	99	38	Svobodová and Votýpka (1998)
		England	—	—	Simpson (1991)
		France	26	46	Mikaelian and Bayol (1991)
		Germany	189	31	Krone et al. (2001)
		Israel	—	—	Bishop and Bennett (1992)
		Italy	39	23	Sacchi and Prigioni (1984)
		Kazakhstan (Tyan-Shan)	19	84	Kairullaev and Yakunin (1982)
		Kazakhstan	13	23	Yakunin and Zhazyltaev (1977)
		Scotland	—	—	Peirce and Marquiss (1983)
		Spain	—	—	Peirce et al. (1983)
		Sweden	—	—	Wingstrand (1947)
		South Africa	18	33	Bennett et al. (1992c)
		Ukraine	—	—	Glushchenko (1962)
Madagascar Buzzard	*Buteo brachypterus*	Madagascar	—	—	Bennett and Blancou (1974)
Jackal Buzzard	*Buteo rufofuscus*	Kenya	—	—	Peirce and Cooper (1977a)
Long-legged Buzzard	*Buteo rufinus*	Iraq	—	—	Shamsuddin and Mohammad (1980)
		Kazakhstan	U	U	Yakunin (1972)
		Turkmenistan	—	—	Berdyev (1979)
Black Eagle	*Ictinaetus malayensis*	Bhutan	—	—	McClure et al. (1978)
Lizard Buzzard	*Kaupifalco monogrammicus*	Sub-Saharan Africa	11	64	Bennett et al. (1992c)
		DR of the Congo	—	—	Todd (1907)
		"French Congo"	U	U	Aubert and Heckenroth (1911)
		Guinea-Bissau	U	U	Tendeiro (1947)
		Nigeria	—	—	Cox and Vickerman (1965)

Brown Snake-Eagle	*Circaetus cinereus*	DR of the Congo	—	—	Rodhain et al. (1913)
Red-tailed Hawk	*Buteo jamaicensis*	USA (California)	458	26†	Sehgal et al. (2006b)
		USA (Colorado)	10	20	Stabler and Holt (1965)
		USA (Florida)	14	7	Forrester et al. (1994)
		USA (Louisiana)	—	—	Olsen and Gaunt (1985)
		USA (Maryland)	16	19	Williams and Bennett (1978)
		USA (Minnesota)	—	—	Taft et al. (1996)
		USA (Nebraska)	—	—	Coatney and Roudabush (1937)
		USA (New Jersey)	10	30	Kirkpatrick and Lauer (1985)
		USA (Oklahoma)	34	35	Kocan et al. (1977)
		USA (Washington)	—	—	Clark et al. (1968)
		USA (Wyoming)	—	—	Smith et al.(1998)
Red-shouldered Hawk	*Buteo lineatus*	USA (California)	40	38†	Sehgal et al. (2006b)
		USA (Florida)	22	5	Forrester et al. (1994)
		USA (Oklahoma)	—	—	Kocan et al. (1977)
Rough-legged Hawk	*Buteo lagopus*	Germany	—	—	Krone et al. (2001)
		Kazakhstan	—	—	Valkiūnas (1989)
		USA (Colorado)	—	—	Stabler and Holt (1965)
		USA (Oklahoma)	—	—	Kocan et al. (1977)
		Ukraine	U	U	Glushchenko (1963)
Ferruginous Hawk	*Buteo regalis*	USA (California)	—	—†	Sehgal et al. (2006b)
		USA (Colorado)	11	55	Stabler and Holt (1965)
		USA (Oklahoma)	—	—	Kocan et al. (1977)
Broad-winged Hawk	*Buteo platypterus*	Canada (Quebec)	—	—	CCWHC records
		USA (Minnesota)	10	80	Taft et al. (1996)
Swainson's Hawk	*Buteo swainsoni*	USA (Colorado)	14	43	Stabler and Holt (1965)
		USA (Montana)	—	—	Coatney and Jellison (1940)
Bald Eagle	*Haliaeetus leucocephalus*	Canada (British Columbia)	U	U	Tucker and Stewart (1988)
		USA (Florida)	23	17	Forrester et al. (1994)
		USA (Michigan)	12	100	Stuht et al. (1999)
		USA (Minnesota)	—	—	Stuht et al. (1999)
African Fish-Eagle	*Haliaeetus vocifer*	DR of the Congo	—	—	Laveran and Nattan-Larrier (1911)
White-tailed Eagle	*Haliaeetus albicilla*	Germany	15	<1	Krone et al. (2001)

(*continues*)

Table 4.6. (*Continued*)

Common name	Species	Location	Number of examinations	Prevalence (%)	Literature source
Golden Eagle	*Aquila chrysaetos*	USA (Colorado)	—	—	Stabler and Holt (1965)
		USA (Mississippi)	—	—	NWHC records
		USA (Ohio)	—	—	Al-Dabagh (1964)
Lesser Spotted Eagle	*Aquila pomarina*	Germany	20	5	Krone et al. (2001)
Tawny Eagle	*Aquila rapax*	Russia (Volgograd)	—	—	Kobyshev and Chashchina (1972)
		South Africa	12	8	Bennett et al. (1992c)
Wahlberg's Eagle	*Aquila wahlbergi*	South Africa	—	—	Bennett et al. (1992c)
Greater Spotted Eagle	*Aquila clanga*	Kazakhstan	—	—	Valkiūnas (1989)
Long-crested Eagle	*Lophaetus occipitalis*	DR of the Congo	—	—	Schwetz (1935)
Changeable Hawk-Eagle	*Spizaetus cirrhatus*	India	—	—	Nandi and Mandal (1984)
Red Kite	*Milvus milvus*	England	—	—	Bishop and Bennett (1992)
		Germany	24	8	Krone et al. (2001)
Black Kite	*Milvus migrans*	DR of the Congo	—	—	Rodhain et al. (1913)
		Sub-Saharan Africa	69	3	Bennett et al. (1992c)
		Kazakhstan	—	—	Yakunin and Zhazyltaev (1977)
		Russia (Volgograd)	—	—	Kobyshev and Chashchina (1972)
		DR of the Congo	—	—	Neave (1909)
Cooper's Hawk	*Accipiter cooperii*	USA (Arizona)	62	8	Boal et al. (1998)
		USA (California)	82	48[†]	Sehgal et al. (2006b)
		USA (Colorado)	11	64	Stabler and Holt (1965)
		USA (Florida)	—	—	Forrester et al. (1994)
		USA (Michigan)	—	—	Hartman (1927)
		USA (Minnesota)	27	78	Taft et al. (1996)
		USA (New Jersey)	—	—	Kirkpatrick and Lauer (1985)
		USA (Oklahoma)	—	—	Kocan et al. (1977)
		USA (Wisconsin)	80	59	Taft et al. (1994)
Northern Goshawk	*Accipiter gentilis*	Canada (Ontario)	—	—	Bennett and Fallis (1960)
		England	10	30	Peirce and Cooper (1977b)
		Germany	227	9	Krone et al. (2001)
		Spain	—	—	Peirce et al. (1983)
		USA (Colorado)	29	62	Stabler and Holt (1965)
		USA (Minnesota)	48	50	Taft et al. (1996)
		Wales	48	19	Toyne and Ashford (1997)

Sharp-shinned Hawk	*Accipiter striatus*	Canada (Ontario)	—	—	Clarke (1946)
		USA (California)	—	—	Wood and Herman (1943)
		USA (Colorado)	—	—	Stabler and Holt (1965)
		USA (Florida)	11	9	Forrester et al. (1994)
		USA (Louisiana)	—	—	Olsen and Gaunt (1985)
		USA (Minnesota)	55	73	Taft et al. (1996)
		USA (New Jersey)	166	60	Kirkpatrick and Lauer (1985)
		USA (New Mexico)	75	24	Smith et al. (2004)
		USA (Pennsylvania)	83	17	Powers et al. (1994)
Eurasian Sparrowhawk	*Accipiter nisus*	Azerbaijan	U	U	Zeiniev (1975)
		Czech Republic	308	29	Svobodová and Votýpka (1998)
		England	307	23	Ashford et al. (1991)
		Germany	132	5	Krone et al. (2001)
		India	—	—	McClure et al. (1978)
		Iraq	—	—	Shamsuddin and Mohammad (1980)
		Italy	—	—	Sacchi and Prigioni (1984)
		Kazakhstan	146	17	Yakunin and Zhazyltaev (1977)
		Kazakhstan (Tyan-Shan)	—	—	Kairullaev and Yakunin (1982)
		Kazakhstan	536	94	Valkiūnas (1989)
		Kazakhstan	11	45†	Sehgal et al. (2006b)
		Lithuania	21	48	Valkiūnas (1985)
		Portugal	—	—	França (1912)
		Russia	U	U	Valkiūnas (1985)
		Scotland	195	67	Peirce and Marquiss (1983)
		Sweden	—	—	Wingstrand (1947)
		Switzerland	—	—	Geigy et al. (1962)
Shikra	*Accipiter badius*	Ethiopia	—	—	Ashford et al. (1976)
		Guinea-Bissau	U	U	Tendeiro (1947)
		India	12	33	McClure et al. (1978)
		Kazakhstan	11	27	Valkiūnas (1989)
		Mali	—	—	Commes (1918)
		Sub-Saharan Africa	15	80	Bennett et al. (1992c)
		Zambia	—	—	Peirce (1984)

(*continues*)

Table 4.6. (*Continued*)

Common name	Species	Location	Number of examinations	Prevalence (%)	Literature source
Little Sparrowhawk	*Accipiter minullus*	Ethiopia	—	—	Ashford et al. (1976)
		South Africa	—	—	Earlé et al. (1991)
Japanese Sparrowhawk	*Accipiter gularis*	Malaysia	—	—	McClure et al. (1978)
		Philippines	—	—	McClure et al. (1978)
African Goshawk	*Accipiter tachiro*	Ethiopia	—	—	Ashford et al. (1976)
		South Africa	—	—	Bishop and Bennett (1992)
Chinese Goshawk	*Accipiter soloensis*	Philippines	10	10	McClure et al. (1978)
Besra	*Accipiter virgatus*	India	—	—	McClure et al. (1978)
		Malaysia	—	—	McClure et al. (1978)
Levant Sparrowhawk	*Accipiter brevipes*	Kazakhstan	—	—†	Sehgal et al. (2006b)
		Thailand	—	—	McClure et al. (1978)
Dark Chanting Goshawk	*Melierax metabates*	Mali	—	—	Rousselot (1953)
		South Africa	—	—	Bennett et al. (1992c)
Northern Harrier	*Circus cyaneus*	Italy	—	—	Sacchi and Prigioni (1984)
		Kazakhstan	10	30	Yakunin and Zhazyltaev (1977)
		USA (California)	—	—†	Sehgal et al. (2006b)
		USA (Colorado)	—	—	Stabler and Holt (1965)
		USA (Minnesota)	—	—	Taft et al. (1996)
		USA (New Jersey)	—	—	Kirkpatrick and Lauer (1985)
Pallid Harrier	*Circus macrourus*	Italy	—	—	Franchini (1923)
		Kazakhstan	20	20	Yakunin and Zhazyltaev (1977)
		Kazakhstan (Tyan-Shan)	—	—	Kairullaev and Yakunin (1982)
		Uzbekistan	—	—	Abidzhanov (1967)
Montagu's Harrier	*Circus pygargus*	Kazakhstan	32	22	Yakunin and Zhazyltaev (1977)
		Kazakhstan	27	78	Valkiūnas (1989)
		Italy	—	—	Sacchi and Prigioni (1984)
Lizard Buzzard	*Kaupifalco monogrammicus*	Guinea-Bissau	U	U	Tendeiro (1947)
		South Africa	—	—	Bishop and Bennett (1992)

Western Marsh-Harrier	*Circus aeruginosus*	Germany	—	—	Böing (1925)
		Iraq	U	U	Shamsuddin and Mohammad (1980)
		Italy	U	U	Franchini (1924)
		Kazakhstan	12	67	Valkiūnas (1989)
		Lithuania	—	—	Valkiūnas (1985)
		Russia (Volgograd)	—	—	Kobyshev and Chashchina (1972)
		Tadzhikistan	—	—	Subkhanov (1980)
Gabar Goshawk	*Micronisus gabar*	Ethiopia	—	—	Ashford et al. (1976)
European Honey-Buzzard	*Pernis apivorus*	Egypt	U	U	Mohammed (1958)
		Kazakhstan (Tyan-Shan)	—	—	Kairullaev and Yakunin (1982)
		Spain	—	—	Peirce et al. (1983)
		Cathartidae			
Black Vulture	*Coragyps atratus*	USA (Florida)	211	<1	Forrester and Spalding (2003)
Turkey Vulture	*Cathartes aura*	USA (Maryland)	79	3	Wetmore (1941)
		Falconidae			
Chimango Caracara	*Milvago chimango*	Chile	15	87	Forrester et al. (2001a)
Peregrine Falcon	*Falco peregrinus*	Australia	—	—	Raidal et al. (1999)
		England	—	—	Peirce and Cooper (1977b)
		Japan	—	—	Ogawa (1912)
		Kuwait	—	—	Tarello (2006)
		Malaysia	—	—	McClure et al. (1978)
Merlin	*Falco columbarius*	England	—	—	Peirce (1980)
		Kazakhstan	—	—	Yakunin and Zhazyltaev (1977)
		Russia (Volgograd)	—	—	Kobyshev et al. (1975)
Eurasian Kestrel	*Falco tinnunculus*	Finland	227	<1	Korpimäki et al. (1995)
		Germany	136	<1	Krone et al. (2001)
		Italy	20	5	Sacchi and Prigioni (1984)
		Kazakhstan	26	27	Yakunin and Zhazyltaev (1977)
		Kazakhstan	16	6	Valkiūnas (1989)
		South Africa	—	—	Bennett et al. (1992c)

(*continues*)

Table 4.6. (*Continued*)

Common name	Species	Location	Number of examinations	Prevalence (%)	Literature source
American Kestrel	*Falco sparverius*	Canada (Saskatchewan)	442	<1	Dawson and Bortolotti (1999)
		Mexico	U	U	Beltrán and Pardinas (1953)
		USA (Colorado)	58	22	Stabler and Holt (1965)
		USA (Oklahoma)	—	—	Kocan et al. (1977)
Saker Falcon	*Falco cherrug*	Kuwait	—	—	Tarello (2006)
Eleonora's Falcon	*Falco eleonorae*	Greece (Ägäis)	16	13	Wink et al. (1979)
Greater Kestrel	*Falco rupicoloides*	South Africa	19	5	Bennett et al. (1992c)
Lesser Kestrel	*Falco naumanni*	Kazakhstan	13	38	Yakunin and Zhazyltaev (1977)
Australian Kestrel	*Falco cenchroides*	Australia	—	—	Raidal and Jaensch (2000)
Eurasian Hobby	*Falco subbuteo*	Kazakhstan	11	9	Yakunin and Zhazyltaev (1977)
		Kazakhstan	36	6	Valkiūnas (1989)
		Pandionidae			
Osprey	*Pandion haliaetus*	Georgia	U	U	Burtikashvili (1978)
		Sagittariidae			
Secretary-bird	*Sagittarius serpentarius*	Mozambique	—	—	Travassos Santos Dias (1954)

Note: Unless otherwise indicated prevalences were determined by blood film analysis.
CCWHC, Canadian Cooperative Wildlife Health Centre, University of Saskatchewan, Saskatoon, Saskatchewan, Canada; NWHC, National Wildlife Health Center, U.S. Geological Survey, Madison, WI, USA; U, values not known: either not given by author or we were unable to obtain the reference.
*Number of birds examined was less than 10 and therefore the prevalence was not calculated.
†Prevalence determined by polymerase chain reaction technique.

incorrect—they did not attempt those cross family transfers, but instead successfully transferred the guinea fowl leucocytozoid to other guinea fowl and the chicken leucocytozoid to other chickens; (4) the statement that Khan and Fallis (1970a) were unable to transmit *Leucocytozoon dubreuili* from thrushes (Turdidae) to emberizids, icterids, and ducks was incorrect—they did not report attempts to make such transfers, but only transferred *L. dubreuili* from thrushes to thrushes, and (5) the claim that Skidmore (1931) was unable to transfer *L. smithi* from turkeys to other galliforms was incorrect—he experimentally transferred *L. smithi* from turkey to turkey, but did not attempt to transfer it to other galliforms. Regarding this last point, Skidmore (1931) did mention, however, that there were chickens, ducks, and geese on a farm where infected turkeys existed and that these other birds remained free of *L. smithi*. It should be noted here that in another paper (Solis 1973) that was not mentioned by Bennett et al. (1991c), unsuccessful attempts to experimentally transfer *L. smithi* from turkeys to quail, partridges, domestic ducks, and pheasants were reported. Also, not mentioned in Bennett et al. (1991c) were unsuccessful attempts by Fallis and Bennett (1966) and Fallis et al. (1954) to transmit *Leucocytozoon lovati* from grouse to geese and a sparrow, *Leucocytozoon danilewskyi* from an owl to ducks and a sparrow, *L. simondi* from ducks to grouse, chickens, turkeys, and pheasants as well as reported failures to transmit *L. caulleryi* from chickens to nine other galliform species (Morii and Kitaoka 1971). In conclusion, there is only limited experimental evidence to support the hypothesis that leucocytozoids are all specific at the avian family or subfamily level. Rather, there is substantial information that would lead us to conclude that they are specific at least to the ordinal level. Some leucocytozoids such as *L. smithi* of turkeys and *L. caulleryi* of chickens are host species specific, some are host genera specific such as *Leucocytozoon sakharoffi* of crows and ravens and *Leucocytozoon berestneffi* of jays, while some like *L. simondi* are family specific, and others such as *L. toddi*, *Leucocytozoon fringillinarum*, *Leucocytozoon majoris*, and *L. dubreuili* are found in numerous families, but all within the same order (Table 4.3). Valkiūnas (2005) reviewed the citations mentioned above and other literature on experimental infections and concluded that "... there are no scientific facts available which confirm the possibility that the same species of *Leucocytozoon* infect birds belonging to different orders." There are possible exceptions to this as illustrated by the leucocytozoid infections described earlier in a Common Loon and in young Ostriches. However, these infections might have originated from birds of other orders, although this was not proven. This is unlikely to be a common phenomenon.

Using the family host specificity approach, the number of described species of *Leucocytozoon* is around 143, whereas the ordinal host specificity and distinct morphological approach results in numerous synonymies and a list of only 36 valid species (Table 4.3). There is some evidence that cryptic species of *Leucocytozoon* exist (Sehgal et al. 2006b) and future research combining techniques of traditional parasitology and molecular biology will most certainly result in the determination of additional synonyms and rethinking of the systematics of the leucocytozoids.

Leucocytozoids have a number of stages that occur in the simuliid vector and in the blood and other tissues of the avian host. Two morphological forms of gametocytes, either round or elongate, occur in the blood of the avian host. These various stages will be discussed further in the next section in relation to the life cycle. The ultrastructure of the various stages of *L. simondi* has been studied by S. S. Desser and associates (Desser 1970a–c, 1972, 1973; Desser et al. 1970;), and for meronts of *L. toddi* by Raidal and Jaensch (2000).

Strains with varying degrees of pathogenicity have been recognized for *L. caulleryi* in chickens and *L. simondi* in waterfowl (Desser et al. 1978; Morii et al. 1986) and may exist for other species. In the case of *L. simondi*, several strains were recognized in Canada Geese (*Branta canadensis*) from Michigan, USA, and Ontario, Canada. One strain from geese in the Seney National Wildlife Refuge (NWR) in the upper peninsula of Michigan underwent complete development with primary development in the liver, resulting in round gametocytes in the blood and secondary development in reticuloendothelial cells, resulting in the production of many elongate gametocytes and was associated with mortality of goslings. Other strains from geese in Cusino Wildlife Research Station (40 km west of Seney NWR), White Pine Copper Company (278 km west of Seney NWR in Michigan), and Algonquin Park in Ontario underwent development only in the liver, producing only round gametocytes, and did not result in mortality (Desser et al. 1978). A pathogenic Norwegian strain has also been recognized, in which merogony is completed more rapidly than is the case with strains in North America; other differences were recognized in the location and size of megalomeronts (Eide and Fallis 1972).

Recently, partial sequences of the cytochrome *b* gene have been used to characterize species and perhaps subspecies of *L. fringillinarum* from House Sparrows (*Passer domesticus*) in Israel (Martinsen et al. 2006), *L. schoutedeni* from domestic chickens in Uganda (Sehgal et al. 2006a), and *L. toddi* in diurnal raptors in California, Kazakhstan, and Lithuania (Sehgal et al. 2006b). This approach will eventually help to define

intraspecific genetic diversity and the taxonomic and phylogenetic relationships of the leucocytozoids.

EPIZOOTIOLOGY

Leucocytozoids have an indirect life cycle that involves biting flies of the order Diptera as vectors. All are species of Simuliidae (black flies), with the exception of *L. caulleryi,* which uses biting midges of the genus *Culicoides*.

The best-studied life cycle is that of *L. simondi* (Figure 4.2). The works of O'Roke (1934), Huff (1942), Chernin (1952a), Desser (1967), Desser et al. (1968), Khan et al. (1969), Aikawa et al. (1970), Yang et al. (1971), and Eide and Fallis (1972) were foundational to understanding the life cycle of this species and of leucocytozoids in general. The following is a summary of the life cycle of *L. simondi* based on the works listed above as presented by Valkiūnas (2005).

Sporozoites (stages infective to avian hosts) are injected into the blood stream by biting flies while they are taking a blood meal. These sporozoites penetrate hepatic cells where they develop into first-generation meronts (Figure 4.2, 1–3). Over a 4- or 5-day period these meronts increase in size, undergo multiple nuclear divisions, and form a number of separate sections called cytomeres. These further develop into uninuclear merozoites and syncytia containing several nuclei. Some of these merozoites and syncytia enter the blood stream. These merozoites then invade erythrocytes and develop into gametocytes (round forms) (Figure 4.2, 4–6). Syncytia are carried by the blood to many organs (spleen, lymph nodes, liver, brain, etc.), where they are engulfed by macrophages and form megalomeronts (Figure 4.2, 7–8). These megalomeronts contain thousands of merozoites that rupture from the megalomeront and in turn penetrate lymphocytes and other leukocytes and develop into gametocytes (fusiform or elongate forms) (Figure 4.2, 9–11; Figure 4.3). The dynamics of parasitemia is illustrated in Figure 4.4. The round or fusiform gametocytes (male gametocytes or microgametocytes and female gametocytes or macrogametocytes) are infective for the dipteran vector. Once the gametocytes are ingested by a blood-feeding vector, they undergo sexual reproduction and form a zygote that becomes an ookinete (Figure 4.2, 12–14). The ookinete penetrates the midgut of the vector, undergoes sporogony, and produces sporozoites (Figure 4.2, 15–18). These sporozoites then migrate to the salivary glands of the insect vector (Figure 4.2, 19) and then can be injected into the next bird when the vector takes a blood meal.

While development of all leucocytozoid species that have been studied in vectors is similar, it varies depending on species and avian host. First-generation meronts develop in the parenchymal cells of the liver in all leucocytozoids except *L. caulleryi*, which develops in the endothelial cells of the capillaries of many organs (Valkiūnas 2005). First-generation meronts of *L. dubreuili* develop in liver cells and also in endothelial cells of the kidney (Khan and Fallis 1970a), but in *L. smithi*, they develop only in hepatocytes (Steele and Noblet 1992). Little is known about details of the

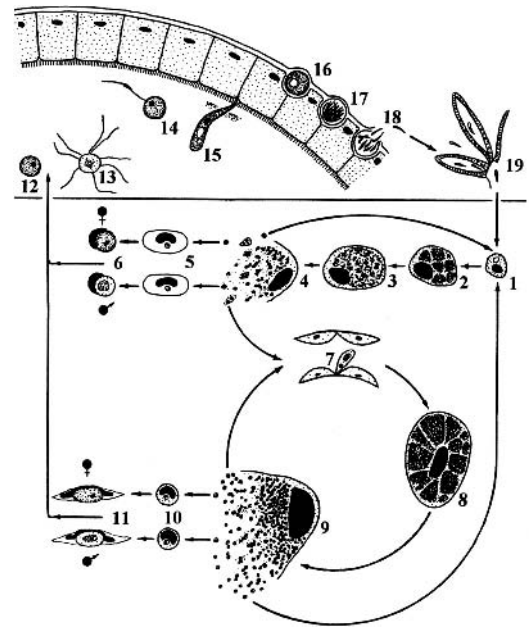


Figure 4.2. Diagrammatic illustration of the life cycle of *Leucocytozoon simondi*. Upper section of the illustration represents events that occur in the vector and lower section represents events that occur in the bird. 1, sporozoite or merozoite in hepatocyte; 2–4, hepatic meronts; 5. merozoites in erythrocytes; 6, gametocytes in round host cells; 7, syncytia of merozoites in reticuloendothelial cells; 8 and 9, megalomeronts; 10, merozoites in mononuclear leukocytes; 11, gametocytes in fusiform host cells; 12, macrogamete; 13, microgamete that is exflagellating; 14, fertilization of macrogamete; 15, ookinete penetrating the peritrophic membrane of the vector's gut wall; 16, young oocyst; 17 and 18, sprogony; 19, sporozoites in the salivary glands of the vector. From Valkiūnas (2005), with permission of the author and *CRC Press.*

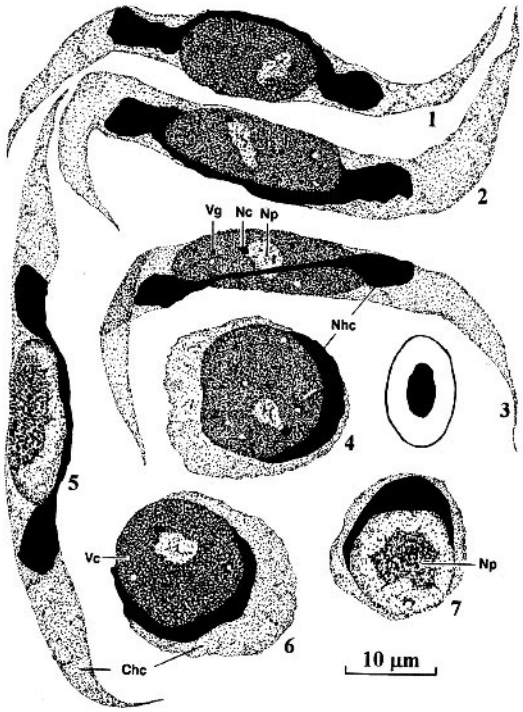


Figure 4.3. Illustrations of the round and elongate forms of the gametocytes of *Leucocytozoon simondi* based on a blood smear from a Eurasian Wigeon (*Anas penelope*). 1–4 and 6, macrogametocytes; and 5 and 7, microgametocytes. Uninfected erythrocyte is between 3 and 4. Chc, cytoplasm of host cell; Nc, nucleolus; Nhc, nucleus of host cell; Np, nucleus of parasite; Vc, vacuole; Vg, valutin granule. From Valkiūnas et al. (1990), with permission of the author and *Parazitologiya* (St. Petersburg).

development and morphology of meronts or megalomeronts in the avian hosts of *L. marchouxi* and *L. toddi*, although they are known to occur. Peirce et al. (1997) described and provided photomicrographs of megalomeronts of *L. marchouxi* in a Pink Pigeon (*Nesoenas mayeri*), as did Simpson (1991) for *L. toddi* in a Eurasian Buzzard (*Buteo buteo*).

Vectors are known for 14 of the 36 leucocytozoid species (Table 4.7). Studies on transmission of species of *Leucocytozoon* include Skidmore (1932), Fallis et al. (1956), Anderson et al. (1962), Barrow et al. (1968), Baker (1970), Noblet et al. (1975), Allison et al. (1978), Greiner and Forrester (1979), Pinkovsky et al. (1981), and Kiszewski and Cupp (1986).

Some species of *Leucocytozoon* can be transmitted by more than one species of black fly, and some species of black fly can transmit more than one species of *Leucocytozoon*. The geographic range of the parasite being transmitted is restricted to the range of the susceptible vector(s) as well as other ecological and behavioral factors. For example, *L. simondi* is absent in nonmigratory Mottled Ducks (*Anas fulvigula*), Fulvous Whistling-Ducks (*Dendrocygna bicolor*), Canada Geese, and Wood Ducks (*Aix sponsa*) in Florida (Thul and O'Brien 1990; Forrester and Spalding 2003) because of a number of biotic and abiotic factors, including behavioral and physiological chacteristics of both hosts and vectors. *Cnephia ornithophilia* is a capable vector of *L. simondi* (Tarshis 1972) and has been found in 14 counties in northern Florida from October through May (Pinkovsky 1976; Pinkovsky and Butler 1978), yet there is no evidence that transmission of *L. simondi* between infected migratory birds and uninfected resident species takes place. *Cnephia ornithophilia* may be either spatially separated from nonmigratory ducks and geese in Florida, or does not feed on these birds. However, this does not preclude migratory species from carrying the parasite with them when they fly south to their wintering grounds from the northern breeding range where transmission occurs. Patent infections have been reported for migratory Wood Ducks and Ring-necked Ducks (*Aythya collaris*; Thul and O'Brien 1990; Forrester et al. 2001b); but as these birds recover from breeding and migrate, their parasitemias may be reduced to a point where they may be too low to infect vectors.

As with other vector-borne diseases and parasites, transmission of *Leucocytozoon* is dependent on availability of appropriate vectors and presence of a sufficient number of gametocytes in the peripheral circulation of the avian host to infect those vectors. In areas with temperate climates, this is achieved through "spring relapse" (Desser et al. 1968; Khan and Fallis 1970b). While the avian host is preparing either for spring migration to the breeding ground or for breeding, it undergoes hormonal changes that induce an increase in the number of circulating blood parasites. Day length and interspecific stress also play a role (Chernin 1952b; Barrow 1963). This raises the parasitemia to a level that facilitates infection of vectors just prior to the production of naïve young of the year. Parasitemias during spring relapse vary among different species of waterfowl. In one study in Ontario, Canada, parasitemias were higher in American Black Ducks (*Anas rubripes*) than in Mallards (*Anas platyrhynchos*) or domestic ducks (Khan and Fallis 1968).

Parasitemias typically decrease after the breeding season and are maintained at a low level. This undoubtedly conserves parasite resources by not having

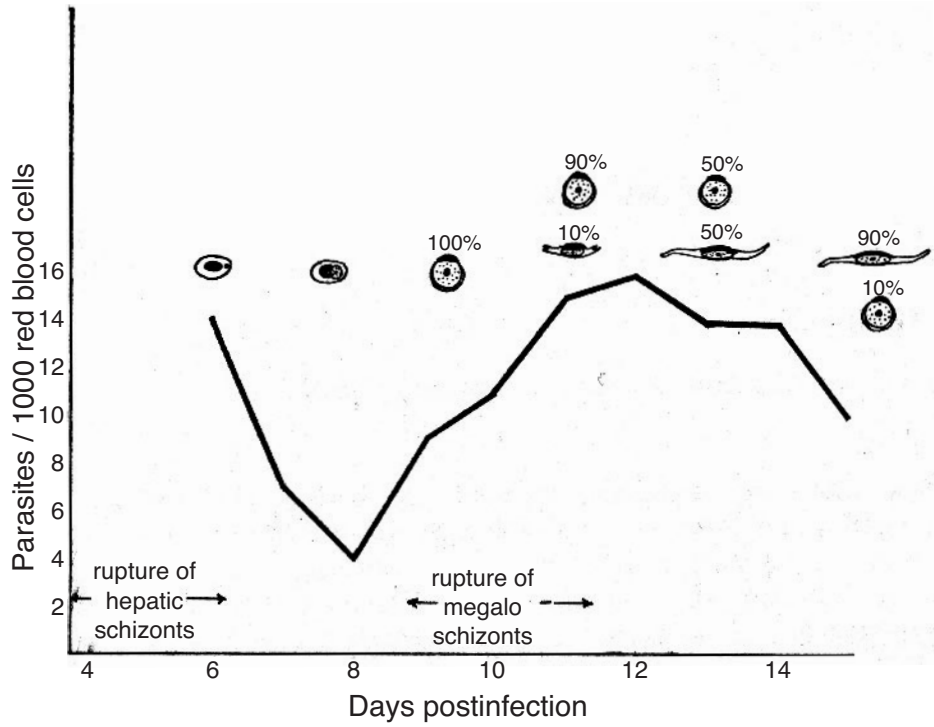


Figure 4.4. Dynamics of parasitemia of *Leucocytozoon simondi* in experimentally infected ducklings and the ratio of round and fusiform host cells during the development and rupture of hepatic meronts and megalomeronts. (a) Period of rupture of hepatic meronts; (b) period of rupture of megalomeronts. Ordinate indicates mean parasitemia from eight ducklings expressed as number of parasites per 1,000 erythrocytes; abscissa indicates days post inoculation of sporozoites. Adapted from Desser 1967, with permission of the author and the *Journal of Protozoology*.

parasites being produced when there are no vectors available for transmission and causes less damage to the avian host. Relapses have been observed in birds infected with *L. danilewskyi*, *L. dubreuili*, *L. lovati*, *L. simondi*, *L. smithi*, *L. toddi*, and some other leucocytozoids (Ashford et al. 1990; Valkiūnas 2005).

The behavior of avian hosts is an important feature of the epizootiology of leucocytozoid infections. This includes nesting and roosting habits (Figure 4.5) as well as migratory behavior. Some colonial-nesting birds have a higher diversity of haemosporidian parasites, including leucocytozoids, and higher prevalances of infection than do solitary-nesting birds (Tella 2002). This is most likely related to greater efficiency of transmission where host density is high. There are exceptions to this, however; some colonial nesting birds have very low prevalences of blood parasites and some have none at all, probably due to factors that may either hinder or limit numbers of vectors (Valkiūnas 2005). In England, *L. toddi* is transmitted to adult and nestling Eurasian Sparrowhawks (*Accipiter nisus*) primarily at the nest site prior to dispersal of nestlings at about 2 months of age, resulting in a 33% prevalence of infection (Ashford et al. 1990, 1991).

Migratory waterfowl (and other avian species as well) are exposed to a more diverse community of parasites than are nonmigratory species, and therefore have a higher risk of infection (Figuerola and Green 2000). One example of this is *L. simondi* infections in Wood Ducks in the Atlantic Flyway in North America. Wood Ducks were sampled from 82 sites in 19 states and provinces throughout the flyway from Florida, USA, to New Brunswick, Canada, during the nesting season and before migration began (Thul et al. 1980; Thul and O'Brien 1990). Infections with *L. simondi* were found in ducks from Ontario, Canada, and several northern

Table 4.7. Species of dipterans known to serve as vectors of species of *Leucocytozoon*.

	Species of *Leucocytozoon*	Species of vector
1	*Leucocytozoon balmorali*	Unknown
2	*Leucocytozoon bennetti*	Unknown
3	*Leucocytozoon berestneffi*	*Prosimulium decemarticulatum*
		Simulium aureum
4	*Leucocytozoon caprimulgi*	Unknown
5	*Leucocytozoon caulleryi*	*Culicoides arakawae*
		Culicoides circumscriptus
		Culicoides odibilis
		Culicoides schultzei
6	*Leucocytozoon centropi*	Unknown
7	*Leucocytozoon cheissini*	Unknown
8	*Leucocytozoon colius*	Unknown
9	*Leucocytozoon communis*	Unknown
10	*Leucocytozoon danilewskyi*	*Prosimulium decemarticulatum*
		Simulium aureum
		Simulium latipes
11	*Leucocytozoon dizini*	Unknown
12	*Leucocytozoon dubreuili*	*Cnephia ornithophilia*
		Prosimulium decemarticulatum
		Simulium aureum
		Simulium croxtoni
		Simulium latipes
		Simulium quebecense
13	*Leucocytozoon eurystomi*	Unknown
14	*Leucocytozoon fringillinarum*	*Cnephia ornithophilia*
		Prosimulium decemarticulatum
		Simulium aureum
		Simulium croxtoni
		Simulium latipes
		Simulium quebecense
15	*Leucocytozoon grusi*	Unknown
16	*Leucocytozoon hamiltoni*	Unknown
17	*Leucocytozoon leboeufi*	Unknown
18	*Leucocytozoon legeri*	Unknown
19	*Leucocytozoon lovati*	*Simulium aureum*
		Simulium croxtoni
		Simulium latipes
		Simulium minus
		Simulium quebecense
20	*Leucocytozoon maccluri*	Unknown
21	*Leucocytozoon macleani*	*Eusimulium geneculare*
		Simulium metatarsale
22	*Leucocytozoon majoris*	Unknown
23	*Leucocytozoon marchouxi*	Unknown
24	*Leucocytozoon neavei*	*Simulium adersi*
		Simulium impukane
		Simulium nyasalandicum
25	*Leucocytozoon nycticoraxi*	Unknown
26	*Leucocytozoon nyctyornis*	Unknown
27	*Leucocytozoon sakharoffi*	*Prosimulium decemarticulatum*
		Simulium angustitarse
		Simulium aureum
		Simulium latipes
		Simulium quebecense
28	*Leucocytozoon schoutedeni*	*Simulium adersi*
		*Simulium impukane**
		Simulium nyasalandicum
		Simulium vorax
29	*Leucocytozoon simondi*	*Cnephia ornithophilia*
		Simulium anatinum
		Simulium fallisi
		Simulium innocens
		Simulium latipes
		Simulium parnassum
		Simulium rendalense
		Simulium rugglesi
		Simulium venustum
		Simulium vittatum
30	*Leucocytozoon smithi*	*Prosimulium hirtipes*
		Simulium aureum
		Simulium congareenarum
		Simulium jenningsi
		Simulium meridionale
		Simulium pictipes
		Simulium slossanae
		Simulium venustum
		Simulium vittatum
31	*Leucocytozoon sousadiasi*	Unknown
32	*Leucocytozoon squamatus*	Unknown
33	*Leucocytozoon struthionis*	Unknown
34	*Leucocytozoon tawaki*	*Austrosimulium australense*
		Austrosimulium dumbletoni
		Austrosimulium ungulatum
35	*Leucocytozoon toddi*	*Prosimulium decemarticulatum*†
		Simulium aureum†
		Simulium quebecense†
36	*Leucocytozoon vandenbrandeni*	Unknown

Sources: Chang (1975), Greiner (1991), and Valkiūnas (2005).

*Identification uncertain (Fallis et al. 1973).

†Sporogony occurs in these vectors, but transmission to birds has not been proven (Bennett et al. 1993a).

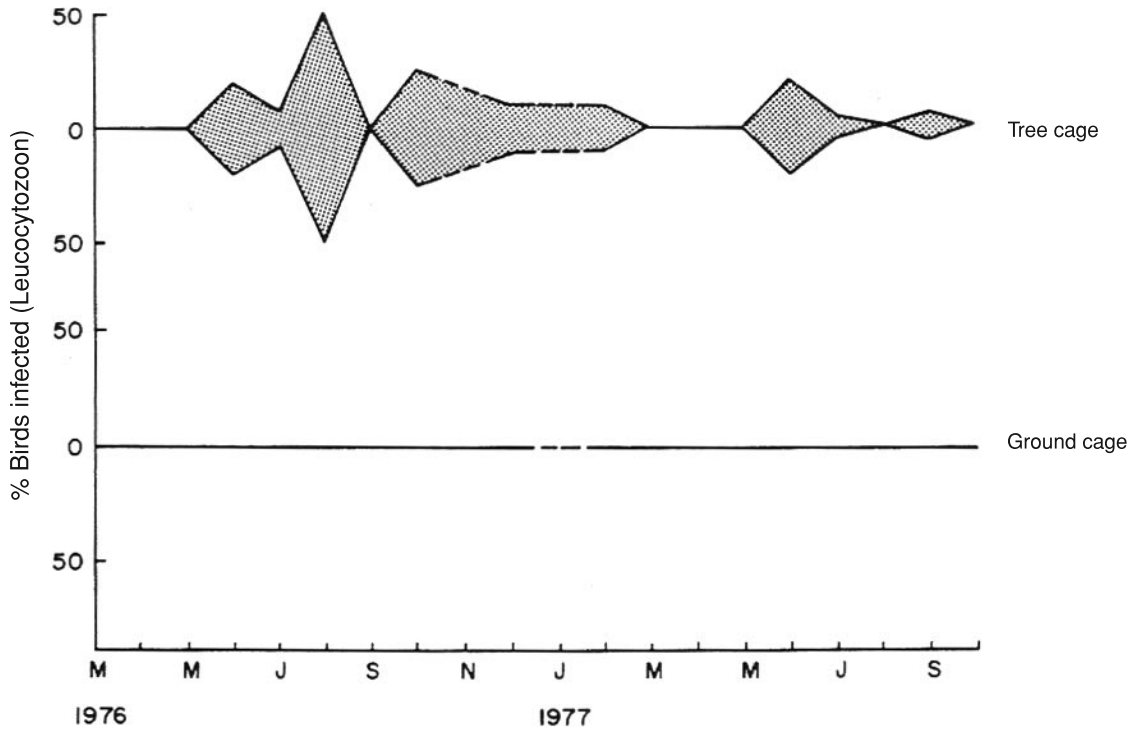


Figure 4.5. Transmission of *Leucocytozoon smithi* to sentinel domestic turkeys maintained in cages in Wild Turkey (*Meleagris gallopavo*) habitat at Fisheating Creek Wildlife Management Area in southern Florida during 1976 and 1977. Tree cages were located in the canopy where turkeys roost at night; ground cages were in ground-level habitat where they feed, rest, and nest. Broken lines indicate missing data due to deaths of sentinel birds unrelated to disease. From Forrester and Spalding (2003).

states (Maine, Massachusetts, New York, Maryland, and Pennsylvania), but not in any of the states south of Maryland. After migration began in the fall, infected ducks were found in southern states; they had acquired their infections in the north prior to flying south for the winter. Another example is the presence of leucocytozoids in several species of passeriforms in the Curonian Spit, which is located partly in Lithuania and partly in Russia and projects into the Baltic Sea (Valkiūnas 1993). Through a long-term banding study, it was determined that leucocytozoids were not transmitted to the migratory population of passeriforms that were hatched on the spit, but were present in the same species of passeriforms that had migrated south and had overwintered in southern Europe and Africa where they acquired the parasites and then returned to the Curonian Spit. It was determined that there were no simuliid vectors on the Curonian Spit due to the lack of flowing, well-oxygenated fresh water needed for the flies to breed.

Transmission of leucocytozoids is also dependent on a number of abiotic factors including favorable environmental conditions, particularly temperature, rainfall, humidity, and the presence or absence of running water. Running water is necessary for black fly vectors to reproduce (Adler et al. 2004), and this requirement in turn influences the transmission of leucocytozoids. For example, the prevalence of *L. simondi* in waterfowl is lower in years of drought than it is in normal years in western Canada (Bennett et al. 1982a). In southern Florida, there is a significant correlation between the prevalence of *L. smithi* in Wild Turkeys (*Meleagris gallopavo*) and the depth of nearby creeks. Prevalence of infection is higher during periods when water levels in streams and adjacent cypress swamps increase and available habitat for black flies expands (Figure 4.6).

As a result of variation in abiotic factors, transmission of leucocytozoids occurs during restricted periods of time in northern climates or throughout the year in warmer climates. Sentinel birds have been used

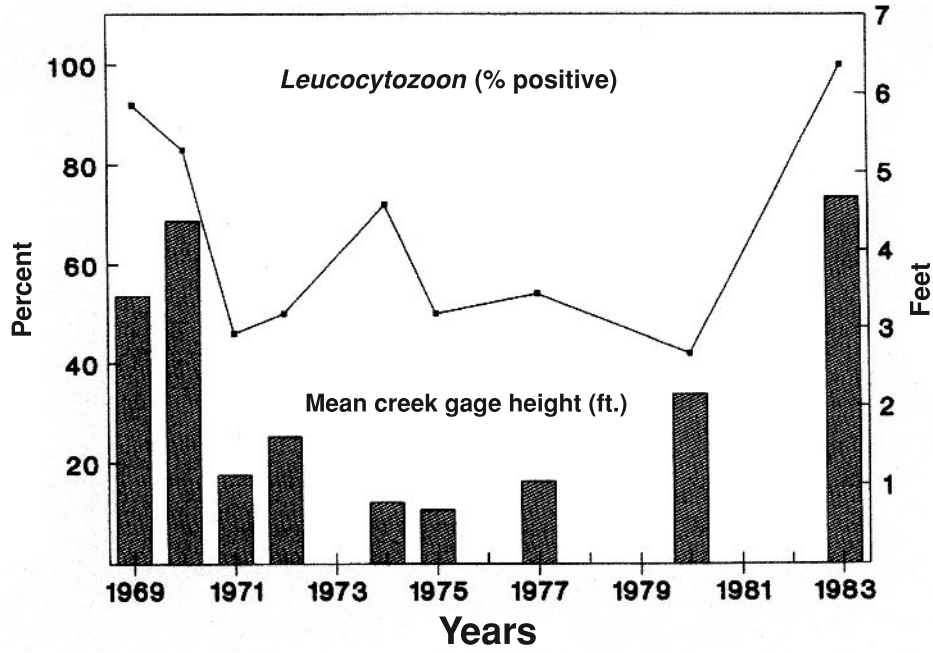


Figure 4.6. Comparison of prevalences of *Leucocytozoon smithi* in Wild Turkeys (*Meleagris gallopavo*) at Fisheating Creek Wildlife Management Area in southern Florida during the months of July, August, September, and October, with the depth of water in Fisheating Creek during the preceding March and April over a 15-year period, 1969–1983. From Forrester and Spalding (2003).

to study the dynamics of transmission of a number of leucocytozoids including *L. simondi* in waterfowl (Herman and Bennett 1976) and *L. smithi* in Wild Turkeys (Forrester and Spalding 2003). In eastern Canada, transmission of *L. simondi* to sentinel ducks occurs in June and July. Transmission of *L. smithi* to sentinel turkeys takes place over a more extended period of time in the subtropical climate of southern Florida (Figure 4.5), but occurs only during March, April, and May in northern Florida, possibly because of weather or the absence of the primary vector (*Simulium slossonae*) during the rest of the year (Atkinson and van Riper 1991).

CLINICAL SIGNS

Clinical signs of leucocytozoonosis are usually nonspecific and may not be apparent (Wobeser 1997). Young ducks and geese are most susceptible to leucocytozoonosis and may die within a short time after infection. Ducklings may be active and normal in the morning, ill and with no interest in eating by midafternoon, and dead by the following morning (O'Roke 1934). Older birds may be listless and lose their wariness of humans, but rarely die of the disease. Anemia is the most important clinical sign (Maley and Desser 1977) and packed cell volumes may be only 20% of normal (Fallis et al. 1951). Other signs are anorexia, lethargy, labored breathing, and diarrhea (Wobeser 1997). Some birds exhibit nervous signs such as marked excitement (O'Roke 1934) and convulsions (Khan and Fallis 1968).

Doves infected with *L. marchouxi* have been reported to exhibit listlessness, ruffled feathers, anemia, and below average body weights (Oosthuizen and Markus 1968; Peirce 1984). Clinical signs in raptors infected with *L. toddi* may range from erratic flight, reduced flight speeds, lack of coordination, depression, blindness, spontaneous erratic vocalization, and seizures to anorexia, weight loss, vomiting, weakness, labored breathing, and ruffled feathers (Raidal and Jaensch 2000; Tarello 2006).

PATHOGENESIS

Three species of *Leucocytozoon* are reported to be pathogenic to wild birds, *L. simondi*, *L. marchouxi*, and *L. toddi* (Table 4.2). There is some evidence that *L. danilewskyi* may be pathogenic to owls, but definitive

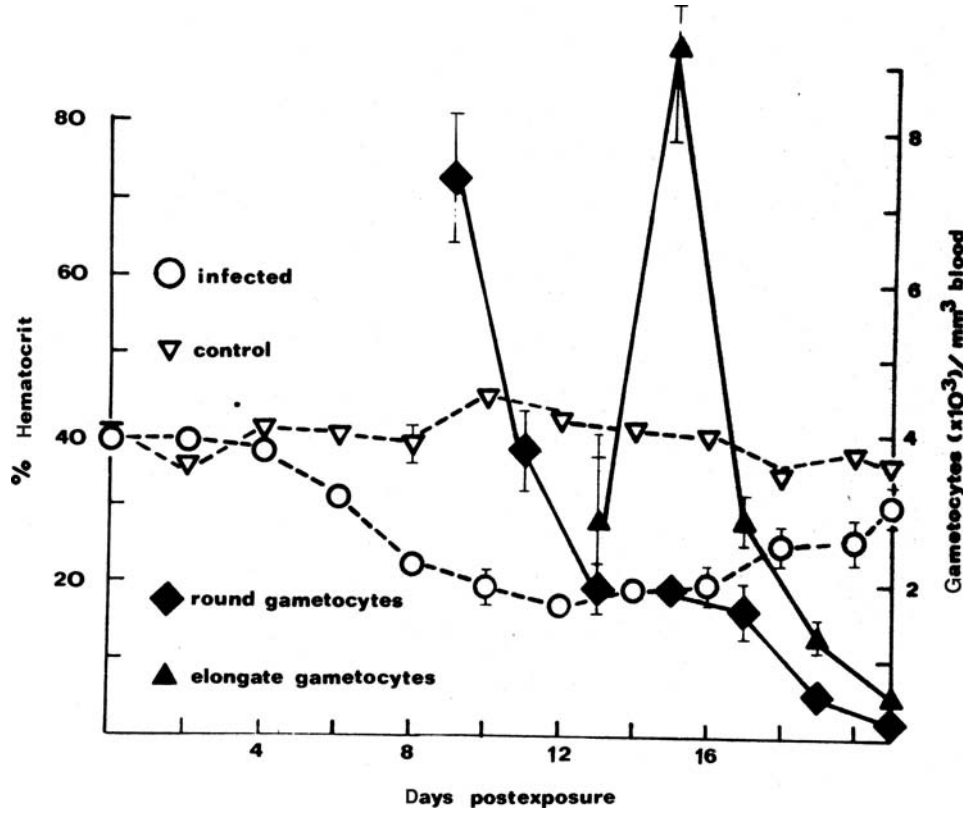


Figure 4.7. Mean percentage hematocrits and numbers of gametocytes for ducklings infected with *Leucocytozoon simondi* compared with uninfected controls. Bars surrounding data points indicate standard error of the mean. From Maley and Desser (1977), with permission of the authors and the *Canadian Journal of Zoology*.

data are lacking. There is one report that *L. danilewskyi* infection may have caused a reduction in egg production (Korpimäki et al. 1993), but necropsies as well as clinical and histopathologic studies were not performed to verify this. In another study, mortality of fledgling owls was attributed to severe black fly feeding in concert with *L. danilewskyi* infections (Hunter et al. 1997), but not to leucocytozoonosis alone. The best-studied species is *L. simondi* (see reviews in Wobeser (1997) and Valkiūnas (2005)).

The pathogenesis of leucocytozoonosis in waterfowl due to *L. simondi* can best be understood with reference to the life cycle and development of gametocytes and exoerythrocytic stages in the tissues of infected birds over time (see Figure 4.2). It begins with the injection of sporozoites into the blood stream and their subsequent invasion of hepatic cells. Here, they undergo further development into meronts over a period of 5 days (Desser 1967). Beginning on days 4–6 postinfection (PI), erythrocytes become fragile and birds become anemic as numbers of erythrocytes begin to drop (Figure 4.7). Anemia is associated with the rupture of meronts and the release of merozoites and syncytia into the circulation. Merozoites invade erythrocytes and develop into round gametocytes; syncytia are carried via blood to various organs including spleen, lymph nodes, liver, and brain, and are engulfed by macrophages and form numerous megalomeronts containing thousands of merozoites. Megalomeronts are quite large, some reaching 60–189 μm in diameter (Desser 1967). It has been estimated that in some infections megalomeronts can make up to three-fourths of the mass of spleen and lymphoid tissue by day 9 PI (Desser 1967). Rupture of megalomeronts and release of merozoites occurs 9–12 days PI and coincides with the peak of erythrocyte fragility and anemia. This anemia is thought to be due to an "anti-erythrocyte factor" released from the meronts or their host cells rather than destruction of the erythrocytes by gametocytes, since the peak of anemia precedes the peak of

parasitemia (Kocan and Clark 1966; Desser and Ryckman 1976). The highest mortality occurs in ducklings at day 12 PI when anemia reaches its peak and most of the megalomeronts have ruptured (Kocan and Clark 1966; Maley and Desser 1977; Valkiūnas 2005).

No detailed studies have been conducted on the pathogenesis of *L. marchouxi* in pigeons and doves and *L. toddi* in raptors, although megalomeronts have been described in numerous internal organs in Pink Pigeons (Peirce et al. 1997) and in Eurasian Buzzards (Simpson 1991).

Pathogenic and nonpathogenic strains of *L. simondi* have been recognized (Eide and Fallis 1972; Desser et al. 1978). The pathogenic strains (i.e., the Norway strain and the Seney strain) undergo primary merogony in the liver and secondary merogony and formation of megalomeronts in various additional organs. Nonpathogenic strains such as Cusino, White Pine, and Algonquin undergo only primary merogony in the liver and do not produce megalomeronts. Pathogenicity of some leucocytozoids seems to be related to the development of megalomeronts (Valkiūnas 2005). However, of the eight species of *Leucocytozoon* that are pathogenic to domestic and wild birds (Table 4.2), three (*Leucocytozoon simondi, L. marchouxi*, and *L. caulleryi*) produce megalomeronts, whereas two other species (*Leucocytozoon macleani* and *L. smithi*) do not. It is not known if the other two species (*Leucocytozoon struthionis* and *L. schoutedeni*) produce megalomeronts. Megalomeronts of *L. toddi* have been described (Simpson 1991), but lack cytomeres that are characteristic of megalomeronts in other species and may actually be large primary meronts (Peirce et al. 1997).

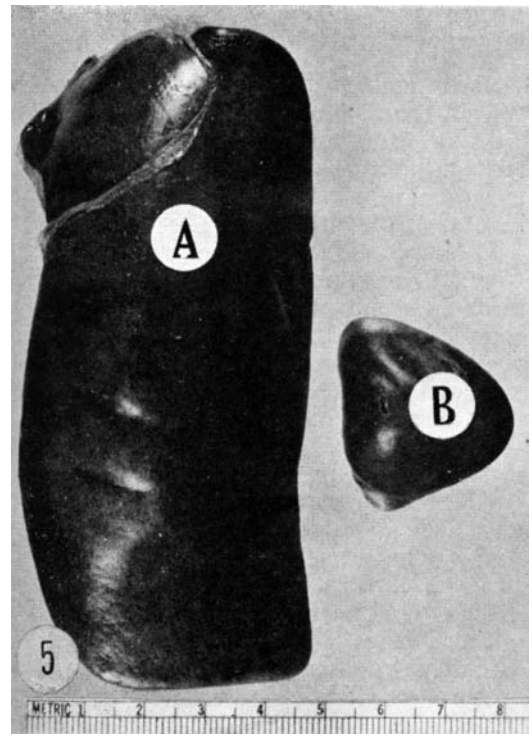


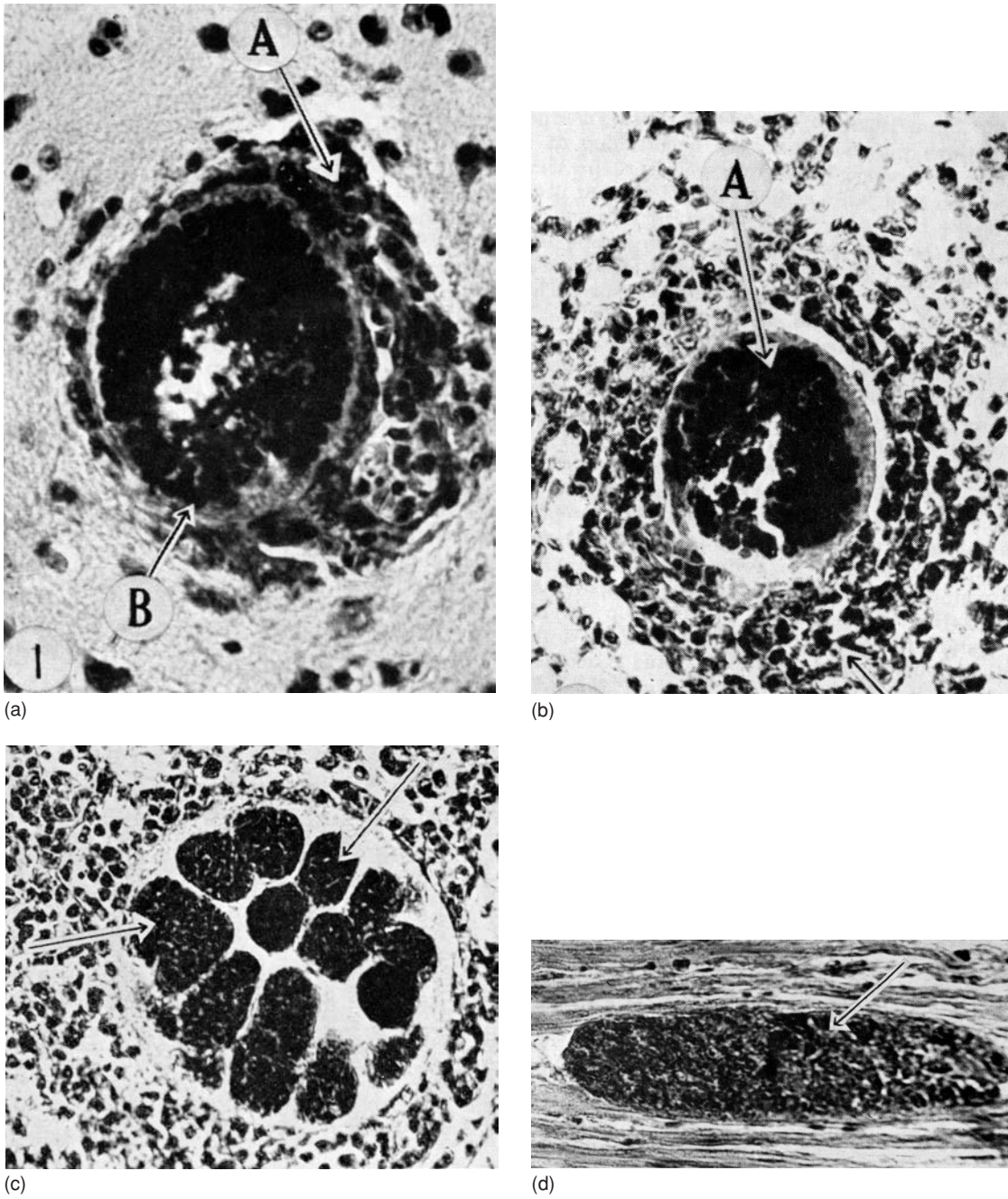
Figure 4.8. Gross view of a spleen from a duck infected with *Leucocytozoon simondi* (a) compared with one from an uninfected control (b). From Newberne (1957), with permission of the *American Journal of Veterinary Research*.

PATHOLOGY

Gross lesions in waterfowl with fatal leucocytozoonosis include enlargement of the spleen (Figure 4.8) and liver, paleness of tissues, and thin watery blood (Wobeser 1997).

Histological studies of infections of *L. simondi* have been reported by several authors (see Wobeser 1997), but the most complete and detailed description was that of Newberne (1957). Capillaries of lungs, liver, and spleen were distended by the presence of many gametocytes, but local host tissue reaction was not evident. Megalomeronts in the brain had moderate to marked cellular reactions (Figure 4.9a), but meronts elicited moderate, slight, or no such reaction. Megalomeronts were located in close association with small blood vessels, and the host reaction was characterized by the proliferation of large mononuclear cells. Megalomeronts that had ruptured and contained no merozoites were filled with an eosinophilic coagulum and large mononuclear cells. In some cases, there were scattered scars that were believed to be the remnants of depleted megalomeronts. Some megalomeronts in the lung had marked host reaction consisting of several layers of lymphoid cells, plasma cells, large mononuclear cells, and fibroblasts (Figure 4.9b), whereas others had less severe reactions or none at all. Megalomeronts in other organs such as spleen (Figure 4.9c) and cardiac muscle (Figure 4.9d) had no local host tissue reactions.

A variety of microscopic changes were noted in various organs. In liver, severe central necrosis was so widespread in some cases that the necrotic areas were confluent. In these areas, there were also marked periportal and diffuse lymphocytic infiltration, prominent Kupffer cells that contained hemosiderin, and macrophages containing pigment. Enlarged spleens were congested and contained many macrophages that were swollen and contained large amounts of pigment and cellular debris. The normal splenic architecture was almost completely obliterated in most birds. Some birds had pulmonary congestion and some had infiltrates of histiocyte-type cells containing cellular



(a) (b)

(c) (d)

Figure 4.9. Megalomeronts of *Leucocytozoon simondi* in various tissues of an infected duck. Hematoxylin and eosin. From Newberne (1957), with permission of the *American Journal of Veterinary Research*. (a) Megalomeront in the brain, showing cellular reaction (A) and round cytomeres (B). ×233. (b) Megalomeront (A) in the lung, showing a prominent cellular reaction (bottom arrow). ×233. (c) Megalomeront in the spleen, showing islands of cytoplasmic masses (arrows). ×300. (d) Elongated megalomeront (arrow) in the cardiac muscle. ×300.

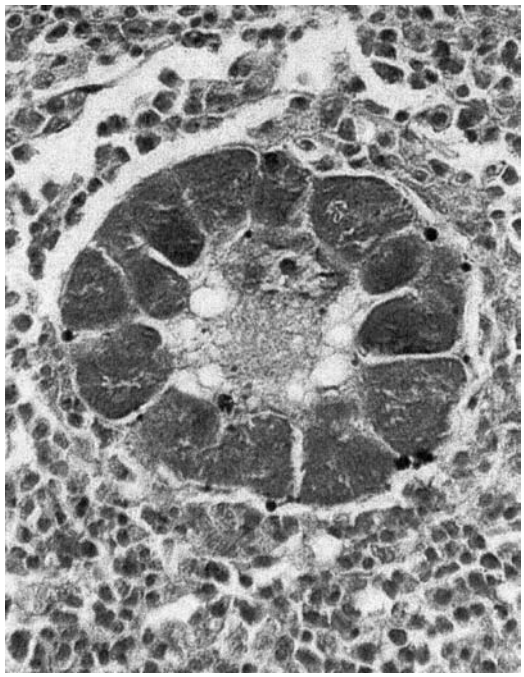


Figure 4.10. Megalomeront of *Leucocytozoon marchouxi* in the spleen of an infected Pink Pigeon (*Nesoenas mayeri*) from Mauritius. Hematoxylin and eosin. ×1140. From Peirce et al. (1997), with permission of *Veterinary Record*.

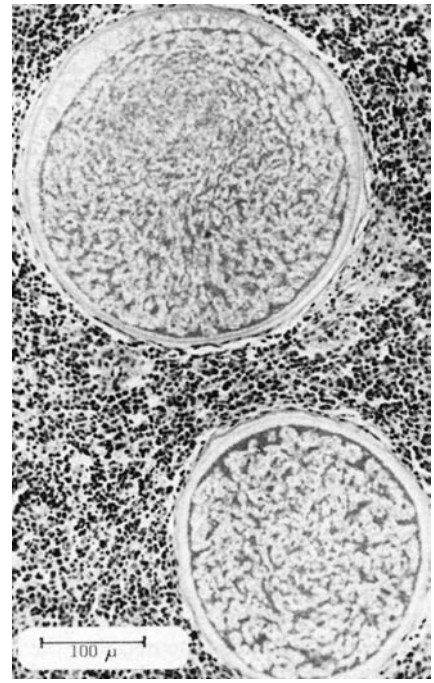


Figure 4.11. Structures identified as megalomeronts of *Leucocytozoon toddi* in the spleen of a Eurasian Buzzard (*Buteo buteo*) from England. Hematoxylin and eosin. From Simpson (1991), with permission of *Veterinary Record*.

debris and traces of pigment in septa of air spaces. Lymphocytic infiltration of the myocardium was seen in some birds. There was moderate to marked hyperplasia and replacement of fat by proliferating cells in bone marrow.

Little is known about gross and histologic lesions in doves and pigeons infected with *L. marchouxi*. A 7-week-old Pink Pigeon squab from Mauritius that died of leucocytozoonosis had megalomeronts in various stages of development in the liver, pancreas, heart, kidney, intestine, and spleen (Peirce et al. 1997). Megalomeronts measuring up to 210 μm in diameter were very numerous in the spleen (Figure 4.10). There was also liver and renal tubular necrosis and hemorrhage in the myocardium.

Splenomegaly has been reported as a gross lesion of leucocytozoonosis in a Eurasian Buzzard infected with *L. toddi* (Simpson 1991). Histopathological features have been described in connection with central nervous disease and blindness in Peregrine Falcons (*Falco peregrinus*) and Australian Kestrels (*Falco cenchroides*) in Australia (Raidal et al. 1999; Raidal and Jaensch 2000). Lesions included severe endarteritis, pectenitis, and meningoencephalomyelitis. The arterioles of the meninges, brain, optic papillae, optic nerve, and spinal cord had marked proliferation of endothelial cells and numerous meronts measuring from 40 to 60 μm in diameter. Meronts were also present in smaller numbers in lung, liver, heart, and intestines. Megalomeronts were not reported in the falcons and kestrels, but Simpson (1991) reported these stages in the spleen, pectoral muscle, and heart of a Eurasian Buzzard in England (Figure 4.11). No hemorrhage or myodegeneration was associated with the megalomeronts. However, these may not be megalomeronts, but actually very large meronts, since cytomeres were not present (Peirce et al. 1997).

DIAGNOSIS

Diagnosis of *Leucocytozoon* infections (i.e., leucocytozooniasis) and diagnosis of the disease caused by *Leucocytozoon* spp. (i.e., leucocytozoonosis) must be

considered separately. Infections by *Leucocytozoon* spp. can be diagnosed readily by examining stained thin films made from peripheral blood and finding the characteristic gametocytes. Valkiūnas (2005, pp. 213–216) has provided an excellent description of the methods of making and staining thin blood smears. By noting the morphologic and metric characteristics of the gametocytes, the host involved, and by using appropriate descriptive literature, the species can be determined. The recent monograph by Valkiūnas (2005) contains pertinent information gathered from the world literature on blood protozoans, including measurements, illustrations, and keys, and is a significant resource for identifying these organisms.

Diagnosis of leucocytozoonosis should include observation of appropriate clinical signs (especially anemia), the presence of typical gross and histologic lesions, and the identification of gametocytes of *Leucocytozoon* spp. in the blood (Wobeser 1997). It must be remembered, however, that mortality of young birds may occur in the absence of parasitemia, and birds may be parasitemic without having leucocytozoonosis (Herman et al. 1975; Wobeser 1997).

Since the early 1970s, a number of serological tests have been developed to detect antibodies against *L. caulleryi* in chickens. These include agar gel precipitation (Morii 1972), counter-immunoelectrophoresis (Fujisaki et al. 1980), immunofluorescence (Fujisaki et al. 1981; Isobe and Akiba 1982), an enzyme-linked immunosorbent assay (ELISA) (Isobe and Suzuki 1986, 1987a, b), immunoblot analysis (Isobe et al. 1998), and a latex agglutination test using recombinant R7 antigen (Ito and Gotanda 2005). Similar tests have not been developed for other species of *Leucocytozoon.*

Over the past 12 years, a number of molecular genetic tests have been developed for screening birds for the presence of blood parasites. Most of the tests are polymerase chain reaction-based assays that target fragments of small unit (18S) ribosomal RNA (Feldman et al. 1995; Jarvi et al. 2002) or mitochondrial cytochrome *b* genes (Bensch et al. 2000; Fallon et al. 2003; Waldenström et al. 2004) to identify *Plasmodium* and *Haemoproteus* at the generic level. Several recent modifications of these assays allow *Leucocytozoon* to be distinguished from *Plasmodium* and *Haemoproteus* (Hellgren et al. 2004; Beadell and Fleischer 2005; Cosgrove et al. 2006), but none of these tests allow identification of leucocytozoids below the level of genus. Recent efforts to link molecular tests with traditional morphological species that are recognized as valid will lead to development of important diagnostic tools for investigating the ecology of these organisms and for recognizing cryptic species or subspecies (Martinsen et al. 2006; Sehgal et al. 2006a).

NATURAL RESISTANCE AND IMMUNITY

Information on natural or innate resistance to leucocytozoids is sparse, although some data are available on experimental infections of *L. simondi* in domestic ducks, American Black Ducks, and Mallards (Khan and Fallis 1968). Primary infections using ducklings and adults of all three species resulted in higher mortality in domestic ducks (white Pekins) than in American Black Ducks and Mallards given the same doses of sporozoites. Relapse parasitemias were higher in American Black Ducks than in either domestic ducks or Mallards. Overall, domestic ducks were more susceptible to *L. simondi* than the endemic wild species. These observations are similar to those made by several earlier investigators (Anderson et al. 1962; Trainer et al. 1962; Fallis and Bennett 1966).

With the exception of *L. caulleryi* infections in chickens and *L. simondi* infections in waterfowl, very little is known about acquired immunity to leucocytozoids. Chickens that have recovered from primary infections of *L. caulleryi* are resistant to reinfection (Morii et al. 1986, 1989), although young chickens are less resistant than older ones (Morii and Kitaoka 1970). IgM and IgG antibodies are involved in immunity (Isobe and Suzuki 1987b) as well as cell-mediated responses (Nakata et al. 2003; Ito and Gotanda 2005). Complete protection against *L. caulleryi* is achieved by immunization with a recombinant R7 vaccine which is expressed against second-generation meronts (Ito and Gotanda 2005).

It is not known if the immune responses in chickens infected with *L. caulleryi* also occur in primary infections of wild birds with other species of *Leucocytozoon*. In waterfowl infected with *L. simondi*, this does not seem to be the case. Domestic ducks exposed to a single infection and not challenged until 6 weeks later are not resistant to reinfection (Fallis et al. 1951). However, ducks exposed to primary infections of *L. simondi* and then repeatedly exposed to infected vectors over a 3-week period have persistently lower parasitemias than do uninfected control ducks that are exposed to infected vectors at the same time (Fallis et al. 1951). Fallis et al. (1974) referred to this as a state of premunition. However, chronically infected birds that are exposed to infection a year later develop high parasitemias and die.

Immunological factors such as concentrations of white blood cells (Ortego and Espada 2007) and immunoglobulins (Tomás et al. 2007) have been used to

assess disease risk and the impact of blood parasites (including *Leucocytozoon* spp.) on breeding populations of wild birds. Unfortunately, these studies have involved birds infected by multiple species of parasites (protozoans and arthropods), and the role of the species of *Leucocytozoon* in the process is not clear.

PUBLIC HEALTH CONCERNS

There are no public health concerns since leucocytozoids infect only birds.

DOMESTIC ANIMAL HEALTH CONCERNS

Six species of *Leucocytozoon* cause significant disease in domestic birds (Table 4.2). These include *L. simondi* in domestic waterfowl in the US, Canada, and Europe; *L. smithi* in domestic turkeys in the US and Canada; *L. macleani* in chickens in Southeast Asia; *L. struthionis* in captive ostriches in South Africa; *L. schoutedeni* in chickens in sub-Saharan Africa and Southeast and Southern Asia; and *L. caulleryi* in chickens of many Southeast and Southern Asian countries (Fallis et al. 1974; Springer 1978; Valkiūnas 2005). In an analysis of 237 published reports of mortality or pathogenicity cause by avian blood protozoans, 95% were reports on leucocytozoonosis caused by *L. caulleryi* ($n = 90$), *L. simondi* ($n = 72$), and *L. smithi* ($n = 37$) in domestic chickens, ducks, and turkeys (Bennett et al. 1993b). *L. simondi* and *L. smithi* are also found in wild waterfowl and Wild Turkeys, respectively, and these hosts may serve as reservoirs of the parasite for their domestic counterparts. *Leucocytozoon smithi* and *L. caulleryi* cause significant economic losses in poultry in certain areas of the US and Asia (Fallis et al. 1974; Morii 1992; Bennett et al. 1993b), whereas the other species are of lesser concern. *Leucocytozoon smithi* causes mortality of poults and adult turkeys (Stoddard et al. 1952), but also causes a decrease in production and hatchability of eggs in those that survive (Jones et al. 1972). *Leucocytozoon caulleryi* also causes mortality and retarded growth in young chicks and reduction of egg production in adult chickens (Morii 1992).

WILDLIFE POPULATION IMPACTS

The impact of leucocytozoonosis on populations of wild birds is not clear. As mentioned earlier, three species (*Leucocytozoon simondi* in waterfowl, *L. marchouxi* in doves and pigeons, and *L. toddi* in raptors) are of concern, and there may be other pathogenic species that are not recognized as such at this time.

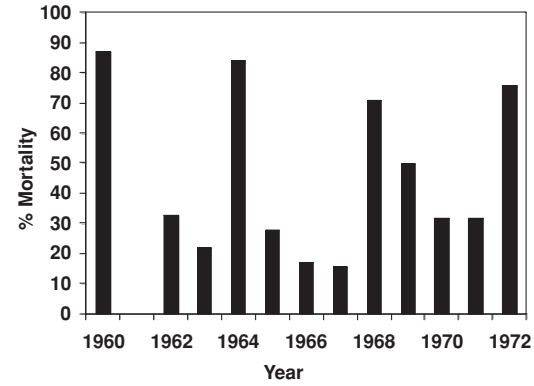


Figure 4.12. Annual mortality of Canada Geese (*Branta canadensis*) goslings at Seney National Wildlife Refuge in northern Michigan from 1960 to 1972. Figure was prepared with data published by Herman et al. (1975).

Leucocytozoonosis caused by *L. simondi* mainly occurs in the northern Holarctic and has caused localized mortality in wild waterfowl in the northern US (O'Roke 1931, 1934; Herman et al. 1975), Canada (Karstad 1965; Leighton and Riddell 1979), and Sweden (Mörner and Wahlström 1983). The best example is the documented annual mortality of Canada Goose goslings at Seney NWR in northern Michigan (Herman et al. 1975). Mortality of goslings was noted in the refuge since its inception in 1935. The best records were from 1960 to 1972 when mortality of goslings reached over 70% every 4 years (Figure 4.12). Correlations between these cyclic fluctuations of mortality and weather, hunting, predation, and other parasites and diseases have not been found. Other reports of mortality are more anecdotal in nature. Mortality as high as 90% was observed in Mallards and American Black Ducks in some areas of Michigan in the early 1930s (O'Roke 1931, 1934). Death of a juvenile Mallard in 1963 due to leucocytozoonosis in Ontario, Canada, was described by Karstad (1965). No information was given on the number of Mallards at risk in the population from which the duck came or if there was additional mortality of Mallards in the area in question. There is another similar report of a wild duckling (species not identified) dying of leucocytozoonosis in Saskatchewan, Canada (Leighton and Riddell 1979). In southern Sweden, 10 of 62 young Mute Swans (*Cygnus olor*) examined at necropsy over a 10-year period were found to have died of leucocytozoonosis (Mörner and Wahlström 1983). In a 1993 analysis of the

literature on mortality caused by avian blood parasites, 199 reports on *Leucocytozoon* spp. were found and of those 79 were concerned with *L. simondi*. However, of those 79 reports, only 7 dealt with wild waterfowl, the other 72 were records of mortality in domestic ducks and geese (Bennett et al. 1993b). Since this analysis was published, two studies designed to test the effect of *L. simondi* on mortality (Shutler et al. 1996) and growth rates (Shutler et al. 1999) of Mallard and American Black Duck ducklings under conditions of natural exposure to *L. simondi* in Ontario, Canada, have been completed. No adverse effects were observed in either study, but the authors may have been dealing with nonpathogenic strains of the parasite. It is clear that although pathogenic strains of *L. simondi* cause some mortality among wild anseriforms, there is little or no evidence, with the possible exception of Canada Geese at Seney NWR, that the parasite is controlling population densities of waterfowl.

In 1993–1994, mortality of two Pink Pigeon squabs from Mauritius was attributed to leucocytozoonosis caused by *L. marchouxi* (Peirce et al. 1997). A subsequent study of the same population of Pink Pigeons was conducted in 2003 (Bunbury et al. 2007). Pigeons less than 1 year of age had higher prevalences of infection with *L. marchouxi* (∼45%) compared to older birds (∼10–20%). There was no measurable effect on body condition based on measurements of body mass, tarsus length, culmen-gape, culmen-skull, culmen-feathers, and wing length. However, mortality during the 12-month period after sampling was 21% for infected birds compared to 12% for uninfected birds. There was a statistically reduced likelihood of infected birds surviving to 90 days postsampling compared to uninfected birds.

Although *L. toddi* has been shown to cause leucocytozoonosis and mortality in falcons and kestrels in Australia (Raidal et al. 1999; Raidal and Jaensch 2000), observations on the effects on raptor populations are limited and indicate that there is no impact. Long-term studies have been conducted on Eurasian Sparrowhawks in England (Ashford et al. 1990, 1991; Ashford 1994). No statistical differences were found between the survival rate of infected and uninfected nestlings and adults. Reduction of the growth rate of nestlings, increased mortality in young fledged birds, and reduction in the fecundity of infected adults were only temporary and not statistically verified because of small sample sizes. Similarly, no difference was found in the survival of infected versus uninfected nestlings and fledglings of Northern Goshawks (*Accipiter gentilis*) in a 1994 study of 48 nestlings from 23 nests in Wales (Toyne and Ashford 1997).

On the other hand, it must be remembered that chronic infections may have harmful sublethal influences on avian populations that are not obvious or readily measured. Wild birds also commonly have concurrent infections with a wide variety of other infectious and noninfectious disease agents. Infections with *Leucocytozoon* might have additive effects or interact with these agents in a synergistic fashion and lead to compromised behavior or health. Leucocytozoid infections might not be the direct cause of death, but may elevate host susceptibility to predation or other disease agents or compromise host fitness for reproduction or migration. Little is known about the physiological and ecological costs of leucocytozoid infections and more research is needed.

PREVENTION, TREATMENT, AND CONTROL

A variety of techniques have been used to prevent and treat clinical disease caused by *L. caulleryi* and *L. smithi* in domestic poultry and have met with some success. Two vaccines have been developed against the megalomeronts of *L. caulleryi* to protect chickens against leucocytozoonosis. One is a formalin-killed vaccine containing second-generation megalomeronts (Morii et al. 1990), while the other is a recombinant vaccine based on a second-generation megalomeront protein (Ito and Gotanda 2004). Pyrimethamine and a combination of sufamonomethoxine and pyrimethamine are effective when administered in food (Akiba et al. 1963, 1964; Akiba 1970). Repellents such as DA-14-7 have been used effectively inside chicken houses and directly on feathers to decrease biting by the vectors of *L. caulleryi* (Hori et al. 1964; Kitaoka et al. 1965). Among domestic turkeys, clopidol is effective in reducing numbers of gametocytes of *L. smithi* in the blood, but does not eliminate infections completely (Siccardi et al. 1974). Vector control in areas where domestic turkey populations are at risk from infection with *L. smithi* has been tested by applying *Bacillus thuringiensis israelensis* (Bti) to flowing streams, the source of black fly vectors. In one study, all streams within 7 km of a turkey farm were treated with a wettable formulation of Bti, leading to a reduction in transmission, reduced parasitemias, and prevention of morbidity or mortality among domestic turkeys (Horosko and Noblet 1968). Temephos, an organophosphate larvicide, was also effective against the larvae of black flies when applied by air to running streams in an area that was endemic for *L. smithi.* No harmful effects to nontarget stream biota were detected (Kissam et al. 1973, 1975). When possible, vector-proof screening of pens may be of use, although this is not always feasible.

Prevention, treatment, and control of leucocytozoonosis in free-living populations of wild birds

are difficult. While leucocytozoonosis in captive waterfowl and raptors can sometimes be treated satisfactorily with quinine derivatives (O'Roke 1934), atebrine (Coatney and West 1937), trimethoprim and sulfamethoxazole (Remple 2004), and melarsomine (Tarello 2006), currently there are no effective treatments for *L. simondi* or *L. toddi* in wild birds (Bennett 1987; Bermudez 2003). In one field study of *L. majoris* in a small sample of Eurasian Blue Tits (*Cyanistes caeruleus*), prevalence was significantly lower (20%) in females captured in nest boxes and treated with primaquine than in untreated controls (62%) (Tomás et al. 2005).

MANAGEMENT IMPLICATIONS

For most free-ranging avian populations, *Leucocytozoon* infections are probably of little concern, although our knowledge of this topic is limited. However, as previously discussed, pathogenic strains of *L. simondi*, *L. marchouxi*, *L. toddi*, and perhaps other species exist and therefore some avian populations may be at risk.

Little can be done for most wild populations to mitigate the impact of leucocytozoonosis, especially on a large scale. In the cases of small populations, subpopulations of endangered species, or in rehabilitation settings, it may be advisable to try to manage infections. Since injured raptors with blood parasite infections (including *L. toddi*) have significantly longer rehabilitation times and higher mortality rates than do uninfected raptors (Olsen and Gaunt 1985), treatment of hematozoan infections may be beneficial and increase survival when birds are released back into the wild.

Treating (Tomás et al. 2005) or vaccinating (Plumb et al. 2007) a free-ranging population might be done in situations where significant numbers of individuals can be easily captured. Examples include cavity-nesting species, birds such as geese that are flightless for periods of time during molt, and social or colonial species that can be readily captured through use of baits or nets. This approach may be applicable to small populations of endangered or threatened birds. Judicious water management or the use of chemicals or biological control agents to treat streams to eliminate or reduce the black fly vectors may also be effective. If certain subpopulations of birds harbor a pathogenic strain of *Leucocytozoon* (*L. simondi* in waterfowl, for example), it might be advisable to selectively reduce or eliminate the subpopulation in an attempt to prevent its spread to other populations. The possible impact of leucocytozoonosis on avian populations, especially waterfowl, should be considered when changes in water flow patterns associated with hydroelectric dams or other types of river and stream management are planned.

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5
Isospora, *Atoxoplasma*, and *Sarcocystis*

Ellis C. Greiner

INTRODUCTION

The genera *Isospora*, *Atoxoplasma*, and *Sarcocystis* are coccidian parasites closely related to *Toxoplasma* and *Eimeria* (Chapters 8, 9, and 11). They produce oocysts with two sporocysts containing four sporozoites each. The life cycles are different for each of these genera, and location of the endogenous or tissue stages of the parasites in the avian host determines which genus is present. Confusion can occur as to which genus is involved depending on how much of the life cycle has been detected. There are species that are pathogenic to the avian host; there are species that are not; and for most species, we do not know their impact on birds. Host specificity varies from being species specific to being able to infect a variety of birds. Most information on these genera is from examination of captive birds.

ATOXOPLASMA AND *ISOSPORA*

Atoxoplasmosis is a disease of birds that is caused by unusual coccidian parasites in the genus *Atoxoplasma*. While this genus has recently been synonymized with *Isospora* (Barta et al. 2005), a great deal of confusion still surrounds it. Most avian coccidia with direct life cycles complete their development in the epithelial cells of the gut and occasionally in the bile ducts of the liver and the collecting tubules of the kidney. The species of *Isospora* that cause atoxoplasmosis have a phase that is extraintestinal in monocytes. The avian taxa that appear to be most at risk are the members of the avian families Fringillidae and Sturnidae.

Atoxoplasmosis is a disease of the recticuloendothelial system as well as the intestines, and a wide range of birds are infected with these organisms. Whereas most infections do not cause disease, they are fatal in some avian hosts. The stages of this parasite that inhabit the blood have been confused with species of *Lankesterella*, and undoubtedly also *Hepatozoon* and *Haemogregarina*. This is not to imply that these latter genera are synonymous with *Atoxoplasma*, but some of the reports of these other genera may actually have been *Atoxoplasma*. In fact, Levine (1982) felt the genus *Atoxoplasma* was valid and placed 19 species into this genus, including some species originally named in these other genera. We have still not addressed questions posed by Baker et al. (1972) in their review of avian blood coccidians: (1) "Are all atoxoplasms really *Isospora*?" (2) "If they are, are they one widespread species?" and (3) "If there are two different groups referred as atoxoplasms, are the two groups monospecific or not?" In this chapter, the recent synonomy of *Atoxoplasma* with the genus *Isospora* is followed, but the disease is continued to be referred to as atoxoplasmosis to distinguish it from disease caused by enteric species of *Isospora*.

Species of *Isospora* are monoxenous, with single host life cycles. There are numerous species of *Isospora* for which their entire life cycle is restricted to the intestinal epithelium of their avian hosts. Most species of *Isospora* are considered host species specific. Little is known about most of them other than the morphology of the oocysts. Thus, if they do have an impact on wild avian populations, it is not recognized.

SYNONYMS

Atoxoplasmosis is sometimes called "going light" as infected birds may stop eating and lose weight.

When associated with disease, infections with *Isospora* spp. will be referred to as coccidiosis, but this condition may be caused by other genera such as *Eimeria* and *Caryospora*. Infection with *Isospora* spp. in the absence of clinical disease is called coccidiasis.

HISTORY

The genus *Atoxoplasma* was defined by Garnham (1950) as those "parasites which inhabit the monocytes of birds from many parts of the world, are strictly host specific, non-pathogenic and possess a delicately granular cytoplasm not enclosed by a periplast, and a large diffuse nucleus with a tiny karyosome." He

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stated that the genus was created for the "sake of convenience" to assist in naming the parasites with this morphology. Parts of this description are not correct as some species are pathogenic to their hosts, and we have no proof of the host specificity of these parasites. There are 19 species listed as valid. Levine (1982) listed these and their presumed synonyms (species of *Lankesterella*, *Hepatozoon*, and *Haemogregarina*) and added a species name to the parasite responsible for an epizootic in Evening Grosbeaks (*Coccothraustes vespertinus*) that occurred in Algonquin Park, Canada (Khan and Desser 1971). *Atoxoplasma* is a synonym of *Lankesterella*, and the red mite (*Dermanyssus gallinae*) is the vector of the parasite in the House Sparrow (*Passer domesticus*) (Lainson 1959). A closely related parasite in canaries (*Serinus canaria*) was transmitted by fecal contamination with oocysts that were structurally similar to species of *Isospora* (Box 1970). Red mites were not present on the canaries, thus demonstrating that this was truly a coccidian parasite.

DISTRIBUTION

Infections of *Atoxoplasma* have been reported from all continents except Antarctica. Many of the reports are based on examinations of blood smears of free-ranging birds. Cases where the stages of the parasite that inhabit circulating monocytes have been clearly associated with fecal oocysts are usually in captive birds in aviaries or under laboratory conditions. Therefore, the true geographic distribution of atoxoplasmosis is probably best indicated by the presence of stages seen on smears of whole blood or white blood cells from the "buffy coat" from centrifuged whole blood, rather than in feces where the infections cannot be distinguished from species of *Isospora* that are restricted to the gut. Related to this point, atoxoplasmosis is a major problem in captive propagation of the Bali Myna (*Leucopsar rothschildi*; Partington et al. 1989) and is apparently caused by the only species of coccidian that has been reported from this host, *Isospora rothschildi* (Upton et al. 2001).

While many species of avian *Isospora* are recognized that are restricted to the gut, the geographic distribution of these has not been determined.

HOST RANGE

The species of *Isospora* that cause atoxoplasmosis have been reported as blood parasites in at least 58 avian families (Bennett et al. 1982; Bishop and Bennett 1992). Unfortunately, it is not clear whether species of *Lankesterella*, *Hepatozoon*, and *Haemogregarina* were correctly differentiated from *Isospora* when these identifications were made. Thus, many orders of birds from a variety of habitats are infected with these parasites. This does not imply that disease occurs in all these hosts, but that disease is possible if conditions are correct.

The level of host specificity for species of *Isospora* that cause atoxoplasmosis is unknown. Khan and Desser (1971) inoculated blood and tissue homogenates (not oocysts) from infected Evening Grosbeaks (*Coccothraustes vespertinus*) into ducks and four species of passeriforms. All but the ducks became positive within 8–14 days. This suggests that there is some host specificity, but at a high taxonomic level. Box (1970) was able to transmit *Isospora* from House Sparrow to House Sparrow, but not to canary. In a study conducted at the San Diego Zoological Gardens (McAloose et al. 2001), a variety of passerines were found to be infected with a species of *Isospora* that causes atoxoplasmosis. The birds ranged in age from several days to nearly 18 years. Most appeared to be infected with one species based on polymerase chain reaction (PCR) amplification of a portion of the small subunit rRNA, but a second potential species was also present with a more restricted host distribution (Schrenzel et al. 2001). Therefore, there is evidence for some host specificity and some indication that at least some of these parasites are transmitted among unrelated host species.

Approximately 140 species of enteric *Isospora* have been reported from a wide variety of avian families (Duszynski et al. 2000).

ETIOLOGY

Oocyst morphology is distinct among species and these can be distinguished by differences in size in some mixed infections (Figures 5.1–5.4). There is no way

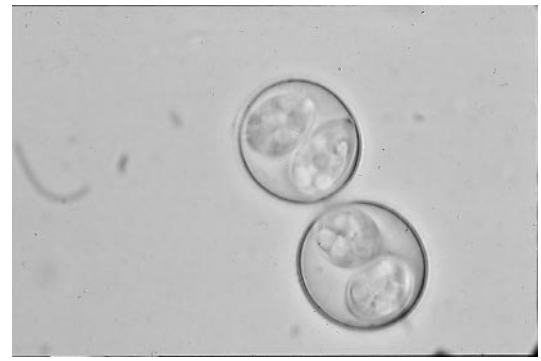


Figure 5.1. Oocysts of *Isospora rothschildi* from Bali Myna (*Leucopsar rothschildi*) (1,000×; 20 × 20 μm).

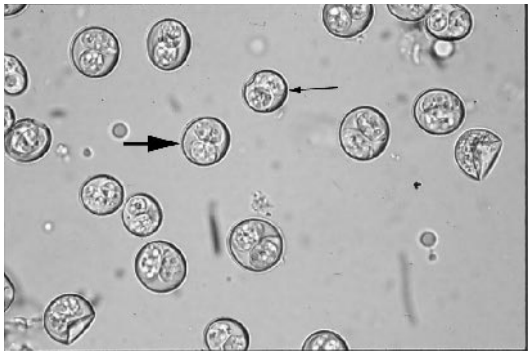


Figure 5.2. Oocysts of *Isospora canaria* (larger arrow; 22 × 21 μm) and *Isospora serini* (smaller arrow; 16 × 16 μm) from Island Canary (*Serinus canaria*) (400×).



Figure 5.4. Oocyst of *Isospora* sp. from Red-billed Leiothrix (*Leiothrix lutea*) (1,000×; 26 × 21 μm).

to distinguish oocysts of enteric species of *Isospora* from those that cause atoxoplasmosis.

The mononuclear cell stages or merozoites of species of *Isospora* that cause atoxoplasmosis have few distinctive morphological features (Figures 5.5–5.8). Thus, the named species of *Atoxoplasma* were distinguished primarily by host species.

EPIZOOTIOLOGY

Coccidia have a direct life cycle and are transmitted by a fecal–oral route that involves ingestion of infective oocysts. The oocysts of species of *Isospora* need to undergo asexual reproduction (=sporogony) in the abiotic environment before they become infective. When ingested by a suitable avian host, the oocysts excyst in the intestines and release sporozoites. Sporozoites invade epithelial cells that line the mucosa. These then undergo asexual reproduction (=merogony) and produce progeny called merozoites. These escape and kill their host cells and invade other host cells where they are genetically programmed to undergo a set number of additional generations of merogony. This process greatly increases the number of parasites in the host. Merozoites from the last generation of merogony invade epithelial cells and initiate the sexual phase of the life cycle (=gametogony). Gametes are produced that fuse to form zygotes. An oocyst wall forms around the zygote and unsporulated oocysts are released, killing their host cells in the process. Oocysts are shed in the feces and undergo sporogony within a few days to become infectious to the next avian host. Those species of *Isospora* that cause atoxoplasmosis undergo early merogony in mononuclear phagocytes in the gut mucosa. Some of these infected monocytes leave the gut and are found in the circulation, but final stages of



Figure 5.3. Oocyst of *Isospora* sp. from Fish Crow (*Corvus ossifragus*) (1,000×; 20 μm).

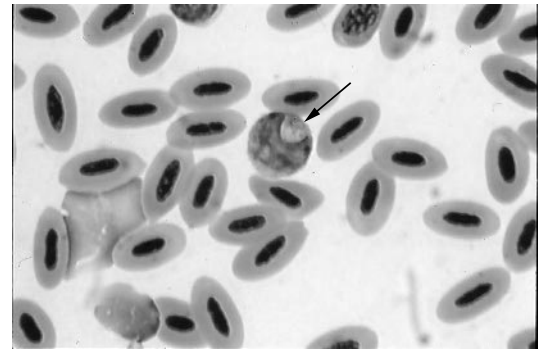


Figure 5.5. Merozoite in monocyte of Bali Myna (*Leucopsar rothschildi*).

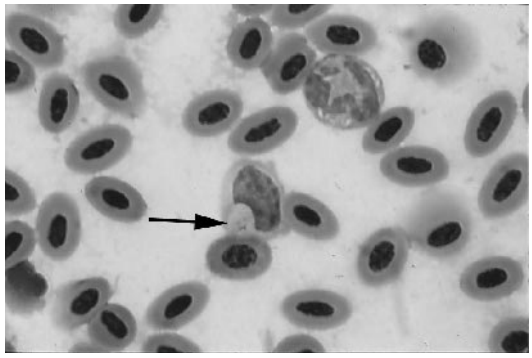


Figure 5.6. Merozoite in monocytes in Superb Starling (*Lamprotornis superbus*).

merogony and gametogony occur in intestinal epithelial cells like most enteric coccidia (Box 1977).

Regardless of whether infections develop only in epithelial cells of the intestines or also in monocytes, oocysts are the infective stage. Coccidiasis caused by the enteric species of *Isospora* develops only if hosts have no previous exposure to the parasites and if the dose of oocysts is sufficiently high. Most infections do not cause disease.

Normally, when coccidia cause disease there are factors that allow a large buildup of infectious oocysts that can be acquired by a susceptible host within a short period of time. In some birds, the number of oocysts shed by adults may increase during nesting. Transmission to nestlings may occur when these are shed during egg laying, brooding, or while the parents are feeding the young in the nest. The type of nest construction might also be important for collection of and buildup of oocysts.

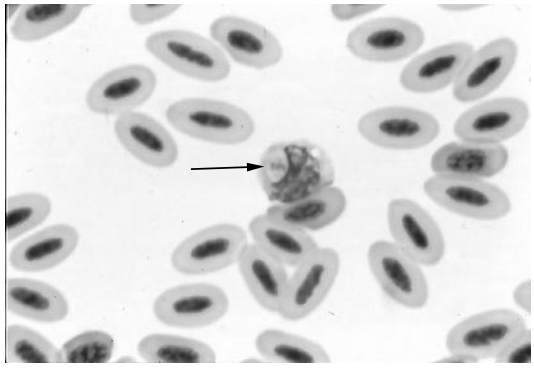


Figure 5.7. Merozoite in monocyte in Wattled Starling (*Creatophora cinerea*).

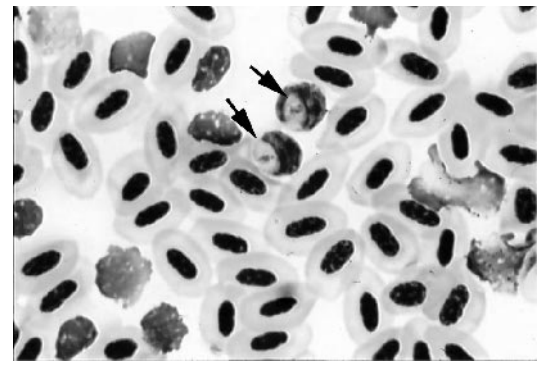


Figure 5.8. Merozoites in monocytes in Golden-crested Myna (*Ampeliceps coronatus*).

Prevalence of atoxoplasmosis in free-ranging birds is poorly understood. In a 4-year study in Ontario, Khan and Desser (1971) estimated that the annual prevalence in Evening Grosbeaks ranged from 29 to 68%. Prevalence in four species of birds from Hawaii ranged from 0.1% in Japanese White-eyes (*Zosterops japonicus*) to 2.9% in House Finches (*Carpodacus mexicanus*), to 8.6% in House Sparrows, and to 17.4% in Nutmeg Mannikins (*Lonchura punctulata*) (van Riper et al. 1987). Prevalence was 100% in 90 House Sparrows and Eurasian Tree Sparrows (*Passer montanus*) in Poland (Kruszewicz 1991). Ball et al. (1998) detected oocysts that they were able to correlate with monocyte infections in the feces of 136 of 922 (14.8%) European Greenfinches (*Carduelis chloris*) in Great Britain. Atoxoplasmosis was the primary cause of death in the loss of 95 of 98 Black Siskins (*Carduelis atrata*) when captive birds were imported from South America to Italy (Giacomo et al. 1997). PCR has recently been used to identify infections associated with atoxoplasmosis in feces, blood, and tissues of captive birds. Tissues from 19 of 32 dead tanagers, representing 15 species, were positive for *Isospora* by PCR in a zoo in the northern United States based on amplification of a fragment of 18s rRNA (Adkesson et al. 2005). These included Purple Honeycreeper (*Cyanerpes caeruleus*), Red-legged Honeycreeper (*Cyanerpes cyaneus*), Blue Dacnis (*Dacnis cayana*), Violaceous Euphonia (*Euphonia violacea*), Hawaii Amakihi (*Hemignathus virens*), Apapane (*Himatione sanguinea*), Silver-beaked Tanager (*Ramphocelus carbo*), Passerini's Tangara (*Ramphocelus passerinii*), White-lined Tanager (*Tachyphonus rufus*), Burnished-buff Tanager (*Tangara cayana*), Paradise Tanager (*Tangara chilensis*), Turquoise Tanager (*Tangara mexicana*), Green-and-gold Tanager (*Tangara schrankii*), Blue-gray Tanager (*Thraupis episcopus*), and Iiwi (*Vestiaria coccinea*).

Prevalence of infection has not been reported for other host species infected with *Isospora* spp. that cause atoxoplasmosis. These include American Goldfinches (*Carduelis tristis*) in Canada (Middleton and Julian 1983), Nashville Warblers (*Vermivora ruficapilla*) in Michigan, USA (Swayne et al. 1991), Eurasian Bullfinch (*Pyrrhula pyrrhula*) in Great Britain (McNamee et al. 1995), and Northern Cardinal (*Cardinalis cardinalis*) in Arizona, USA (Baker et al. 1996).

CLINICAL SIGNS

Clinical signs associated with atoxoplasmosis include loss of appetite, weight loss, diarrhea, lethargy, and ruffled feathers (Norton et al. 1993). Clinical signs are usually not evident in birds that pass oocysts of enteric species of *Isospora*, but clinical signs might mimic those of atoxoplasmosis when disease is present.

PATHOGENESIS AND PATHOLOGY

Much of the available information on pathology of atoxoplasmosis is from captive birds because they are readily available for observation before death and can be necropsied soon afterward. It is only in the most fortuitous occasions that free-ranging birds are observed in the early stages of disease and even more rarely that one can observe the disease as it progresses. Gross lesions associated with atoxoplasmosis include enlargement of the liver and spleen and presence of tiny white foci of necrosis through the parenchyma of both organs and sometimes on the surface of the heart. The pancreas may be hemorrhagic and edematous, the intestines will be filled with fluid, and the air sacs and the pericardium may be filled with a yellowish clear fluid. Enlargement of the liver and spleen is, in part, caused by an infiltrate of mononuclear cells including macrophages, lymphocytes, and plasma cells (Partington et al. 1989; Norton et al. 1993; S. Terrell, personal communication).

Host cells are eventually killed by developing parasites in infections with *Isospora*, but like many coccidia, there is a balance between loss of these cells and their replacement with new ones. When rate of loss is equivalent to rate of replacement, the parasites may be in harmony with their hosts with no evidence of clinical disease. Coccidiosis can develop in naïve individuals when large numbers of oocysts are ingested within a short period of time.

DIAGNOSIS

Based on personal observations of atoxoplasmosis in a large number of captive Bali Mynas, there is rarely any correlation between presence of fecal oocysts and presence of mononuclear merozoites in the same bird at the same time. Standard fecal flotation using centrifugation with Sheather's sugar is the best way to concentrate and cleanse the oocysts for visualization and measurement. This flotation medium is more viscous than saturated salt solutions and makes it possible to use oil immersion lenses and higher magnification to observe finer points of oocyst morphology without having the oocysts move out of the field of view when adjusting focus. Key morphological features of oocysts that are used to distinguish species include length and width of the oocyst and sporocyst, their shapes, presence or absence of a nipple-like Stieda body at one end of the sporocyst, presence of granular material in both the oocyst and the sporocyst called a residuum, presence of a thinning at one end of the oocyst called the micropyle, presence of a cap over the micropyle, presence of large refractile granules in the oocysts called polar bodies, and number of layers in the oocyst wall. Oocysts of *Isospora* need to be aerated for about a week to allow sporulation to occur and to produce infectious oocysts. Fully sporulated oocysts have all the morphological features that are used to identify species. Oocysts can be sporulated in a 3% potassium dichromate solution or a 1% sulfuric acid solution by gently bubbling air through the solution. This process will prevent bacterial overgrowth and reduce adverse odors.

Mature oocysts of *Isospora* will contain two sporocysts and each will contain four sporozoites (Figures 5.1–5.4). To confirm a diagnosis of atoxoplasmosis, both fecal oocysts and merozoites in the monocytes must be present. This may require collection of multiple blood samples over the course of 5–7 days to find merozoites in the monocytes. Mononuclear merozoites are most easily found in impression smears of spleen or liver, but smears prepared from the buffy coat will provide an adequate sample of monocytes when the host is still alive. This procedure will provide a higher number of monocytes for review than in a normal blood smear and thus enhance the potential of detecting the parasite. These are prepared by centrifuging whole blood in a microhematocrit centrifuge tube, breaking the tube at the top of the erythrocyte pellet where white blood cells are concentrated into a thin white band, smearing the top portion of the pellet containing the buffy coat onto a glass microscope slide, and rapidly drying. Both smears of the buffy coat and impression smears should be fixed in absolute methanol and then stained with Giemsa or Wrights/Giemsa. In the vast majority of cases, a single merozoite will be present in the monocyte causing an indentation in the host cell nucleus (Figures 5.5–5.8). It is possible to find meronts in these monocytes as well as single parasites. If one does not find infected monocytes, then it is possible that the bird is infected with an enteric species of

Isospora and thus will have only meronts and gametocytes in the gut epithelium and oocysts in the feces.

Fecal collections should ideally be spread over a 5-day interval and collected from the host at least three times during this period. At necropsy, gross signs of atoxoplasmosis include hepatomegaly, splenomegaly, white pinpoint foci on the surface and cut surfaces of spleen, liver, and occasionally heart, presence of an edematous pancreas, and presence of fluid in the intestinal tract. It is useful to collect the normal range of tissues for histopathology and to make impression smears of at least the spleen and liver.

IMMUNITY

No studies have been conducted on the immunology of avian species of *Isospora*. Atoxoplasmosis is usually a disease of young birds, particularly fledglings, and adults are usually not affected.

SARCOCYSTIS

In contrast to *Isospora*, species of *Sarcocystis* have indirect life cycles. Intermediate hosts containing the large, distinctive intramuscular tissue cysts need to be eaten by the definitive host to transmit the infection. The intermediate host is, in turn, infected by the fecal–oral route by ingesting sporocysts that are excreted by the definitive host. When disease occurs in the intermediate host, it is caused by meronts during early phases of infection and not the large obvious *Sarcocystis*.

SYNONYMS

Infections with *Sarcocystis* spp. are called sarcocystosis.

HOST RANGE AND DISTRIBUTION

Few studies of host range have been conducted for the species of *Sarcocystis* that infect birds. Twelve species of this genus use birds as definitive hosts and twenty-two species use birds as intermediate hosts. Two additional species can use birds for both definitive and intermediate hosts (Table 5.1; Odening 1998).

The most detailed studies of host range in avian hosts have been conducted with *Sarcocystis falcatula*. When sarcocysts from Brown-headed Cowbirds (*Molothrus ater*) and Boat-tailed Grackles (*Quiscalus major*) are fed to opossums, the opossums produce oocysts and sporocysts that are infective to canaries and House Sparrows (Box and Duszynski 1978). Budgerigars (*Melopsittacus undulatus*), Zebra Finches (*Taeniopygia guttata*), and Rock Pigeons (*Columba livia*) are susceptible to infection with sporocysts from opossums, but domestic chickens (*Gallus gallus*) and Helmeted Guineafowl (*Numida meleagris*) are not (Box and Smith 1982). Thus, four different orders of birds can serve as hosts for this species.

Species of *Sarcocystis* that infect birds are widely distributed, with reports from all continents with the exception of Antarctica (Table 5.1).

ETIOLOGY

Like their relatives in the genus *Isospora*, species of *Sarcocystis* are coccidian parasites and have both intestinal and extraintestinal tissue stages and produce infective oocysts that are passed in the feces of their definitive hosts. Meronts of *S. falcatula* occur in endothelial cells of the intermediate host and may be visualized with immunoperoxidase staining or hematoxylin and eosin (Figure 5.9). Sarcocysts are large spindle-shaped structures that occur in the muscle fibers of the intermediate host. They are round when seen in cross section and narrow and elongate when viewed in longitudinal sections (Figure 5.10). Sporocysts (Figure 5.11) typically rupture from their thin-walled oocysts when shed in the feces of the definitive host and are fully sporulated and infectious to the intermediate host as soon as they reach the external environment.

EPIZOOTIOLOGY

The life cycle of *Sarcocystis* is similar to *Isospora*, but requires two hosts. When intermediate hosts consume infective sporocysts, sporozoites are liberated in the intestine. These move through the intestinal wall into the arterial endothelium of the mesenteric lymph nodes where the initial round of asexual reproduction (merogony) occurs (Dubey et al. 1989). Subsequent cycles of merogony occur in endothelial cells in other organs and it is during this phase of development that most pathology occurs. At completion of merogony, merozoites are released that enter muscle cells and develop into septate cysts, which will undergo another form of asexual reproduction called endopolygony. This process leads to the formation of countless infective forms called bradyzoites. Mature sarcocysts will persist in muscle tissue until consumed by a carnivore. After ingestion by a suitable definitive host, bradyzoites will be released in the intestine, enter gut epithelial cells, and undergo sexual reproduction or gametogony. This process forms gametocytes, which mature to produce male (microgametes) and female (macrogametes) that unite to form fertile zygotes. A thin oocyst wall then forms around each zygote and these undergo a final round of asexual reproduction (sporogony) to generate two sporocysts that each contains four sporozoites. Unlike the oocysts of enteric species of *Isospora* that undergo

Table 5.1. Species of *Sarcocystis* that parasitize birds.

Sarcocystis spp.	Intermediate host	Definitive host	Geographic region
Sarcocystis accipitris	Island Canary (*Serinus canaria*)	Northern Goshawk (*Accipiter gentilis*)	Europe
Sarcocystis alectoributeonis	Chukar (*Alectoris chukar*)	Eurasian Buzzard (*Buteo buteo*)	Kazakhstan
Sarcocystis alectorivulpes	Chukar	Mammal (Canidae)	Kazakhstan
Sarcocystis ammodrami	Grassland Sparrow (*Ammodramus humeralis*)	Unknown	South America
Sarcocystis aramidis	Slaty-breasted Wood-Rail (*Aramides saracura*)	Unknown	South America
Sarcocystis buteonis	Mammal (Cricetidae, Muridae, Chinchillidae, Erethizotidae, Leporidae)	Eurasian Buzzard, Red-tailed Hawk (*Buteo jamaicensis*)	Holarctic
Sarcocystis cernae	Mammal (Cricetidae)	Eurasian Kestrel (*Falco tinnunculus*)	Europe
Sarcocystis cheeli	Unknown	Black Kite (*Milvus migrans*)	India
Sarcocystis citellibuteonis	Mammal (Sciuridae)	Eurasian Buzzard	Kazakhstan
Sarcocystis colii	Red-faced Mouse Bird (*Colius erythromelon*)	Unknown	Africa
Sarcocystis dispersa	Mammal (Muridae)	Northern Long-eared Owl (*Asio otus*), Barn Owl (*Tyto alba*)	Europe
Sarcocystis espinosai	Mammal (Cricetidae)	Northern Saw-whet Owl (*Aegolius acadicus*)	North America
Sarcocystis falcatula	*Passeriformes Cuculiformes Columbiformes Psittaciformes*	Opossums (Didelphis spp.)	America
Sarcocystis garzettae	Little Egret (*Egretta garzetta*)	Unknown	South Africa
Sarcocystis glareoli	Mammal (Cricetidae)	Eurasian Buzzard	Western Palearctic
Sarcocystis horvathi	Domestic chicken (*Gallus gallus*)	Unknown	Europe
Sarcocystis jacarinae[*]	Blue-black Grassquit (*Volatinia jacarina*)	Unknown	South America
Sarcocystis kaiserae	Laughing Dove (*Streptopelia senegalensis*)	Unknown	South Africa
Sarcocystis kirmsei	Siamese Fireback (*Lophura diardi*), Common Hill Myna (*Gracula religiosa*)	Unknown	Southeast Asia Central America
Sarcocystis nontenella	Eurasian Buzzard	Unknown	Europe
Sarcocystis oliverioi[*]	Green-rumped Parrotlet (*Forpus passerinus*)	Unknown	America
Sarcocystis peckai	Ring-necked Pheasant (*Phasianus colchicus*)	Mammal (Canidae)	Unknown
Sarcocystis phoeniconaii	Lesser Flamingo (*Phoenicopterus minor*)	Unknown	Africa
Sarcocystis ramphastosi	Keel-billed Toucan (*Ramphastos sulfuratus*)	Unknown	Unknown[†]

(*continues*)

Table 5.1. (*Continued*)

Sarcocystis spp.	Intermediate host	Definitive host	Geographic region
Sarcocystis rauschorum	Mammal (Cricetidae)	Snowy Owl (*Bubo scandiacus*)	North America
Sarcocystis rileyi	Dabbling ducks (*Anas* spp.), Goldeneye (*Bucephala* spp.), American Wigeon (*Anas americana*), White-winged Scoter (*Melanitta fusca*), Blue-winged Teal (*Anas discors*), Northern Shoveler (*Anas clypeata*)	Mammal (Mustelidae, Marsupialia)	North America
Sarcocystis scotti	Mammal (Muridae)	Tawny Owl (*Strix aluco*)	Europe
Sarcocystis sebeki	Mammal (Muridae, Leporidae, Mustelidae)	Tawny Owl	Europe
Sarcocystis setophagae[*]	American Redstart (*Setophaga ruticilla*)	Unknown	North America
Sarcocystis spaldingae	Great Blue Heron (*Ardea herodias*), Striated Heron (*Butorides striata*), Great Egret (*Ardea alba*), Little Blue Heron (*Egretta caerulea*), White Ibis (*Eudocimus albus*), Yellow-crowned Night-Heron (*Nyctanassa violacea*)	Unknown	North America
Sarcocystis sulfuratusi	Keel-billed Toucan	Unknown	Unknown
Sarcocystis wenzeli	Domestic Chicken	Mammal (Canidae)	Europe

Note: Information is summarized from Odening (1998) and Dubey et al. (2004).
[*]Considered by Odening (1998) to be possible synonyms of *Sarcocystis falcatula*.
[†]Described from captive bird; natural range unknown.

sporogony outside of the host, these complete sporogony prior to passing out of the gastrointestinal tract and are immediately infectious to intermediate hosts.

A classic problem in the distribution of avian sarcocystosis in North America developed when old world psittacines were brought into contact with Virginia opossums (*Didelphis virginianus*) and exposed to infection with thesporocysts of *S. falcatula*—a parasite of Brown-headed Cowbirds. Infections are often fatal

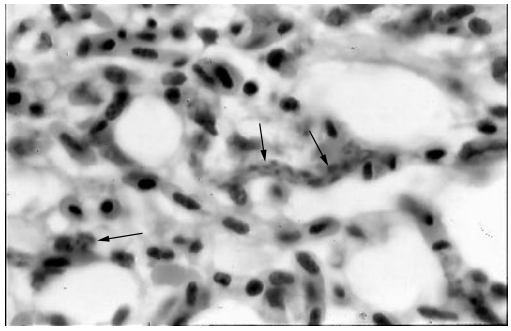


Figure 5.9. Elongate meront of *Sarcocystis falcatula* (arrows) in endothelial cells of a pulmonary capillary from a Lory (*Lorinus* sp.). Hematoxylin and eosin (1000×).

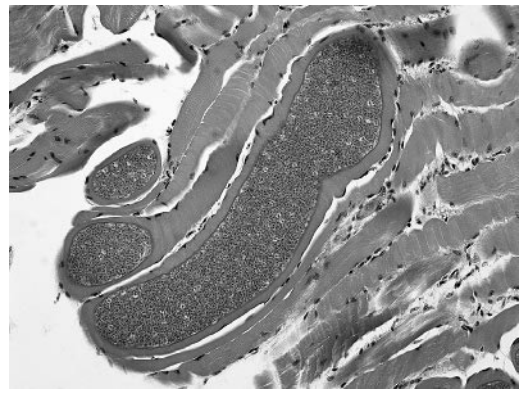


Figure 5.10. Sarcocysts of *Sarcocystis falcatula* in Brown-headed Cowbird (*Molothrus ater*). Muscles are cut in cross and longitudinal sections. Hematoxylin and eosin (100×; maximum width of sarcocysts is 92 μm).

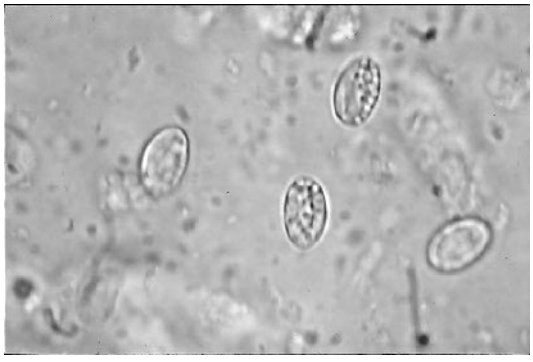


Figure 5.11. Sporocysts of *Sarcocystis falcatula* from feces of Virginia Opossum (*Didelphis virginiana*) (1000×; 11 × 7 μm).

(Hillyer et al. 1991) and may influence the success of captive propagation programs aimed at reintroduction of rare parrots into endemic sites. Infections in captivity result when opossums roam around aviaries and deposit their feces where food or water can be contaminated (Hillyer et al. 1991). Exposure may also occur when cockroaches feed on opossum feces and then enter cages and are eaten by the birds. The sporocysts are not destroyed in the gut of the roach and remain viable and infectious (Clubb and Frenkel 1992). This might occur in the wild where introduced species of parrots have become established in regions where this parasite is cycling. No one seems to know what happened to the massive population of budgerigars that became established in southeastern Florida, but the role that *S. falcatula* may have played in their disappearance is not known. One introduced psittacine pest species that is from the Neotropics, the Monk Parakeet (*Myiopsitta monachus*), is not susceptible to this parasite (E. Greiner, personal observations).

CLINICAL SIGNS

Many species of birds contain sarcocysts that often reach very high intensities, yet health problems are usually not apparent. Sarcocystosis in birds is best known from work on *S. falcatula* in old world psittacines. Susceptible parrots may be fine and active one day and dead the next without showing any outward signs of infection. Some will become anorexic, weak, have difficulty in breathing, have blood in the oral cavity and trachea, and exhibit neurologic signs (Hillyer et al. 1991).

PATHOGENESIS AND PATHOLOGY

As indicated earlier, developing meronts rather than sarcocysts are the primary cause of pathology in intermediate hosts. *Sarcocystis falcatula* is one of the best-studied species in birds, and detailed knowledge about pulmonary and hepatic pathology is derived from experimental infections using budgerigars. Early meronts develop in pulmonary endothelial cells and cause these cells to enlarge and obstruct the capillaries. Inflammatory infiltrates develop in response to tissue damage, and blockage of the blood vessels can lead to interstitial, air space edema, and pulmonary congestion (Smith et al. 1987). Most meronts develop in the lungs with a much smaller proportion in the liver and kidney. The earliest merogony begins about 12 h after ingestion of sporocysts and infection of the lamina propria of the intestines. Meronts are found by day 2 in the lungs and liver. The first sarcocysts develop in cardiac muscle by day 7, but these cysts degenerate by 30–40 days postinfection. Sarcocysts in skeletal muscle appear by day 8 in the pectoral and major leg muscles, but those in the pectoral muscles degenerate (Smith et al. 1989). This does not always happen as massive numbers of sarcocysts can sometimes be seen grossly at necropsy of psittacines (Bolon et al. 1989). While most deaths are attributed to pneumonitis, inflammation of the liver, muscles, kidneys, and brain may also be evident (Smith et al. 1989).

Sarcocystosis in captive old world psittacines can be very acute. The most frequent sign of infection is pulmonary edema that may sometimes be associated with hemorrhage (Hillyer et al. 1991). Parrots may also develop an enlarged spleen and liver with marked inflammation in a number of internal organs (Page et al. 1992). By contrast, infections with *Sarcocystis* are usually nonpathogenic in other avian hosts.

DIAGNOSIS

Sporulated oocysts of species of *Sarcocystis* usually rupture in the feces when they pass out of host, releasing infective sporocysts into the environment (Figure 5.11). The sporocysts may be concentrated from the feces of definitive hosts by the same fecal flotation methods with saturated salt or sugar solutions that are used to recover oocysts from other genera. In the intermediate host, one must inspect the muscles of freshly dead birds for grossly visible, elongate white, thread-like sarcocysts or find the sarcocysts in histologic sections (Figure 5.10). Meronts are less obvious and will require careful scrutiny of sections of host tissues such as the lung (Figure 5.9). Since nothing is shed from the intermediate hosts, necropsy or biopsy is necessary to detect infections with *Sarcocystis* in these hosts.

IMMUNITY

No studies have been done on immunity to *Sarcocystis* in birds. Species of *Sarcocystis* may cause problems in birds of any age, for example *S. falcatula*, but these are usually abnormal hosts for the parasite.

ATOXOPLASMA, ISOSPORA, AND SARCOCYSTIS

PUBLIC HEALTH CONCERNS

Species of *Isospora* will infect only birds. The avian species of *Sarcocystis* may use other vertebrate classes as either intermediate or definitive hosts. None are known to be infectious to humans.

DOMESTIC ANIMAL HEALTH CONCERNS

There are no records of infections of *Isospora* in poultry. There are species of *Isospora* that infect domestic mammals, but none of them infect birds. There are no known cases where dogs or cats serve as definitive hosts of species of *Sarcocystis* that infect birds. Captive wild birds may be at risk from other vertebrates since the complete life cycle and definitive hosts of many species *Sarcocystis* are unknown (Table 5.1).

WILDLIFE POPULATION IMPACTS

Khan and Desser (1971) reported the first outbreak of atoxoplasmosis (=*Lankesterella*) in wild populations, but there have been relatively few reports of atoxoplasmosis in free-ranging birds since then. These include reports of atoxoplasmosis in American Goldfinches and Nashville Warblers that were brought into captivity (Middleton and Julian 1983; Swayne et al. 1991) and a case of pneumonia in a Northern Cardinal that was attributed to this disease (Baker et al. 1996). Most reports of atoxoplasmosis originate from aviaries where this disease may have significant impacts on efforts to use captive propagation to reestablish threatened or endangered species in the wild. This disease has had a substantial impact on captive propagation of the Bali Myna (Partington et al. 1989; Norton et al. 1993), and possibly the largest influence these genera will have on wildlife populations will be their effects on captive populations of threatened or endangered species.

TREATMENT AND CONTROL

Species of *Isospora* are normally not treated in free-ranging birds, but in captive situations both hygiene and anticoccidial drugs have been used successfully to control atoxoplasmosis. Compounds that are effective in drinking water include sulfachlorpyrazine (ESB3) and toltrazuril (Baycox). Sulfachlorpyridazine (Vetisulid) may be substituted for sulfachlorpyrazine, but a vitamin B12 supplement should be used during the treatment (Norton et al. 2002).

The suggested treatment for infections with enteric species of *Isospora* is use of trimethoprim-sulphamethoxazole (Clyde and Patton 1996). Combinations of trimethoprim-sulphamethoxazole and pyrimethamine or trimethoprim-sulfadiazine have been used successfully to control *Sarcocystis* (Page et al. 1992; Clyde and Patton 1996).

Most problems with these genera will be in captivity rather than in the wild. Some of the main problems with atoxoplasmosis will be with captive propagation where the intention is to release individuals back into the wild such as is documented for the Species Survival Plan for the Bali Myna (Norton et al. 2002). Similar concerns might occur with species of *Sarcocystis*. In captivity, a means of reducing contact between psittacines and opossums is to use a hot wire about 10 cm above the ground and about 1 m from the aviary barrier (Susan Clubb, personal communication).

ACKNOWLEDGMENTS

I thank the many keepers and veterinarians in zoos and aviaries across the country for getting me involved with atoxoplasmosis and sarcocystosis. In particular, I thank Dr Terry Norton for his patience and continued prodding and Dr Carter Atkinson for his energetic, professional, and highly useful editorial assistance with this chapter.

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6
Trichomonosis

Donald J. Forrester and Garry W. Foster

INTRODUCTION

Trichomonosis is a protozoan disease caused by the flagellate *Trichomonas gallinae* (Rivolta 1878). It is primarily a disease of the upper digestive and respiratory tracts of columbiforms, raptors, psittaciforms, and a few other birds. Effects vary from subclinical infections (i.e., trichomoniasis) to significant disease (i.e., trichomonosis) that leads to severe organ necrosis, caseation, tissue invasion, and death (Kocan and Herman 1971). Although many instances of this disease relate to individual birds or siblings in the nest, epizootics of sizeable proportions are known, especially among free-ranging columbiforms (Haugen and Keeler 1952).

There is a sizeable body of literature on trichomonosis, only a part of which will be discussed here. Several general reviews have been published (Florent 1938; Stabler and Herman 1951; Stabler 1954; Kocan and Herman 1971; Conti 1993; BonDurant and Honigberg 1994; and Cole 1999). Pokras et al. (1993) summarized publications on the disease in owls.

SYNONYMS

Trichomoniasis, canker, roup (columbiforms, psittaciforms, and other birds), frounce (raptors). (Note: The authors recognize that the term trichomoniasis has been used commonly in the historical literature to refer to the disease caused by *T. gallinae* as well as infection without apparent disease, but we are following the convention of referring to the disease as trichomonosis and the infection without clinical signs as trichomoniasis. See Kassai (2006) for a discussion of this practice.)

HISTORY

Trichomonosis is probably the oldest known wildlife disease for which there are written records. Many years before the cause of the disease was discovered, there were reports of the lesions in birds of prey used for falconry. For example, a book was published in the 1500s which contained a description of the disease (Turbervile 1575). It was not until 300 years later that *T. gallinae* was identified as the etiologic agent (Rivolta 1878). Rivolta described the organism from the upper digestive tract and liver of a Rock Pigeon (*Columba livia*). Following Rivolta's work, there was a period of approximately 100 years when numerous papers were published concerning the nomenclature for this parasite as well as its morphology and its host and geographic distribution throughout the world (Stabler 1954). At that time, there was also considerable research conducted on the cultivation and nutritional requirements of the organism, particularly during the 1930s and 1940s by R. Cailleau, A. Bos, and others. Most of this work was done in Europe and North America. These studies on the cultivation of the parasite were foundational to research that followed on improved methods of diagnosis, treatment, and control and led to studies on virulence, pathogenicity, and immunity in the 1950s, 1960s, and 1970s. The contributions of R. M. Stabler, R. M. Kocan, B. M. Honigberg, and their colleagues were especially noteworthy during that period. From 1980 to the 2000s there were a number of significant findings related to immunity, pathology, ultrastructural morphology of the agent, and the use of molecular techniques to improve diagnosis and understand the phylogeny of trichomonads in general, including *T. gallinae*. Additional details on the history of *T. gallinae* and trichomonosis can be found in the reviews by Stabler (1954), Kocan and Herman (1971), BonDurant and Honigberg (1994), Knispel (2005), and Bunbury (2006).

DISTRIBUTION

Trichomonas gallinae is cosmopolitan and has been reported from every major land mass except Antarctica, Greenland, and the northern parts of North America, Europe, and Asia (Figure 6.1). Its distribution is correlated closely with that of the Rock Pigeon, one of its most important hosts.

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Figure 6.1. Distribution of *Trichomonas gallinae* throughout the world. Solid circles indicate areas where infections were reported either in captive or free-ranging wild birds. This figure is based on information from the references listed in Tables 6.1 and 6.2 and locality records from Babes and Puscariu (1890), Ratz (1913), Volkmar (1930), Bos (1932), Callender and Simmons (1937), Hees (1938), Bushnell and Twiehaus (1940) Russell (1951), Ahmed et al. (1970), Ballouh and Eisa (1980), Minowa et al. (1982), Zhang et al. (1982), Garner and Sturtevant (1992), Rosskopf and Woerpel (1996), and Silvanose et al. (1998).

HOST RANGE

Trichomonas gallinae is common especially in columbiforms, and one of these, the Rock Pigeon, is considered to be its primary host (Stabler 1954). Infections have been reported in Rock Pigeons from 31 countries representing every continent except Antarctica (Table 6.1). In addition to the Rock Pigeon, infections are known to occur in 18 other species of columbiforms (Table 6.1), 26 species of falconiforms (Table 6.2), and 9 species of strigiforms (Table 6.2). Other captive and experimental hosts include psittaciforms, passeriforms, galliforms, gruiforms, and anseriforms. There is also one report of trichomonosis (lesions and trichomonad identification) from a charadriiform (an unidentified species of gull) from the Shetland Isles off the coast of Scotland (Hees 1938). Although there are several reports of successful experimental infections, some accompanied by lesions, in several species of passeriforms (Callender and Simmons 1937; Levine et al. 1941; Stabler 1953), natural infections in these birds are not common. However, in 2002 there was an outbreak of trichomonosis in Kentucky that involved approximately 200 wild House Finches (*Carpodacus mexicanus*) and House Sparrows (*Passer domesticus*) (NWHC 2002). In addition, widespread mortality attributed to a trichomonosis-like disease was reported in several areas in England during 2005 and 2006 and involved large numbers of European Greenfinches (*Carduelis chloris*) and Chaffinches (*Fringilla coelebs*) (Pennycott et al. 2005; Lawson et al. 2006).

ETIOLOGY

Rivolta (1878) discovered the etiologic agent of trichomonosis in 1878 and named it *Cercomonas gallinae*. Rivolta also described another flagellate from

Table 6.1. Reports of *Trichomonas gallinae* in free-ranging and captive columbiforms.

Host	Location	Status	Number of birds		%	Literature source
			Examined	Infected		
Rock Pigeon (*Columba livia*)	Australia (Glenfield)	C*	1	1	—	Hart (1941)
	Australia (Perth)	C	31	22	71	McKeon et al. (1997)
		C*	NG	NG	—	Reece et al. (1985)
	Austria (Vienna)	C*	3	1	—	Krenn (1935)
	Brazil	W	68	18	27	Tasca and DeCarli (1999)
		W	167	104	62	De Carli et al. (1979)
	Canada (Alberta)	W*	NG	NG	—	Pybus and Onderka (2001)
	Canada (Ontario)	W*	NG	6	—	CCWHC (2007)
	Canada (Quebec)	W*	NG	2	—	CCWHC (2007)
	Chile (Santiago)	W	100	11	11	Toro et al. (1999)
	Croatia	W	285	148	52	Greguric et al. (1986)
		W	332	232	70	Bošnjak and Greguric (1989)
	Egypt	W	106A	60	57	Abd-El-Motelib et al. (1994)
		C*	87J	35	40	
		C*	95N	23	24	
	England	W*	NG	8	—	DEFRA (2003)
	Ecuador (Galapagos Islands)	W	10	10	100	Harmon et al. (1987)
		W*	16	6	38	
		W	18	8	44	Padilla et al. (2004)
	Germany	C*	NG	NG	60–100	Friedhoff (1982)
		C*	NG	32	—	Knispel (2005)
		C	NG	41	—	
	Greece	W*	110	14	13	Githkopoulos and Liakos (1987)
	Hungary	W	—	—	—	Ratz (1913)
	India (Hyderabad)	C*	2	2	—	Mohteda (1956)
	India (Calcutta)	C*	2	2	—	Bhattacharya et al. (1997)
	Iran	C*	~720	NG	—	Bozorgmehri-Fard and Moeinvaziri (1985)

Italy	W*	262	41	16	Delogu et al. (1997)
	NG	85	6	7	Tacconi et al. (1993)
	NG	109	35	32	Catelli et al. (1999)
Japan (Nakano)	C*	NG	NG	—	Oguma (1931)
Japan (Tokyo)	W*	NG	NG	—	Minowa et al. (1982)
Namibia	W*	NG	NG	—	Pepler and Oettlé (1992)
Netherlands	C*	NG	NG	—	Bos (1932)
Nigeria	W	NG	NG	—	Oyedapo et al. (2004)
"Persian Gulf States"	W	NG	NG	—	Samour et al. (1995)
Philippines	C*	NG	NG	—	Tongson et al. (1969)
Romania	W	—	—	—	Babes and Puscariu (1890)
Serbia (Belgrade)	W	57	NG	—	Kulišic et al. (1996)
	C	96	NG	—	
Slovenia (Ljubljana)	W	293	109	37	Dobeic (2003)
	W	139	11	8	Dovc et al. (2004)
	W, C	399	304	76	Zadravec et al. (2006)
South Africa (Cape Town)	C*	2	2	—	Jowett (1907)
South Africa (Wellington)	C*	10	5	50	Pepler and Oettlé (1992)
Spain	W	101	80	79	Martínez-Moreno et al. (1989)
Sudan	NG*	NG	NG	—	Ballouh and Eisa (1980)
Switzerland	C	NG	NG	—	Sporri (1938)
Trinidad	W	44	9	20	Kaminjolo et al. (1988)
Turkey	W	80	60	75	Gulegen et al. (2005)
United Arab Emirates	W*	60	21	35	Bailey et al. (2000)
	C*	150	102	68	
USA (Alabama)	W	1	1	—	Haugen and Keeler (1952)
USA (Arkansas)	W	10	8	—	Barrows (1975)
USA (California)	C*	>100	NG	30	Niemeyer (1939)
	W	11	11	100	Stabler and Herman (1951)
USA (Colorado)	W*	100	69	69	Stabler (1951)
USA (Connecticut)	C*	50	30	60	Stabler and Herman (1951)

(*continues*)

Table 6.1. (*Continued*)

Host	Location	Status	Number of birds		%	Literature source
			Examined	Infected		
	USA (Florida)	W	13	9	69	Shamis (1977)
		W	27	21	78	Forrester and Spalding (2003)
		C	7	6	—	Forrester and Foster (2001)
	USA (Hawaii)	C*	2	2	—	Yager and Gleiser (1946)
	USA (Illinois)	W	50	50	100	Jaskoski and Plank (1967)
	USA (Iowa)	W	NG	NG	—	Stiles (1939)
		C*	1	1	—	Kietzmann (1993)
	USA (Louisiana)	C*	515	102	20	Rosenwald (1944)
	USA (Maryland, New	C	55	54	98	Stabler (1941b)
	Jersey, and Pennsylvania)	W	187	102	55	
	USA (Maryland)	W*	148	109	74	Locke and Herman (1961)
	USA (Minnesota)	C*	16	16	100	Waller (1934)
	USA (Nebraska)	W	4	2	—	Greiner and Baxter (1974)
	USA (New Jersey)	W*	NG	NG	—	Cauthen (1934)
	USA (New York)	C*	62	17	27	Cauthen (1936)
	USA (North Carolina)	W*	1	1	—	McCulloch (1950)
	USA (South Carolina)	W	10	1	—	Barrows (1975)
	USA (Pennsylvania)	C	20	20	100	Stabler and Shelanski (1936)
	USA (Virginia)	W	20	12	65	Barrows (1975)
	USA (Texas)	W	153	19	12	Panigrahy et al. (1982)
	USA (Washington, DC)	W*	148	109	74	Stabler and Herman (1951)
Common Wood-Pigeon	England	W*	NG	"mass	—	Duff (2003)
(*Columba palumbus*)				mortality"		
		W*	1,026	79	<1	Cousquer (2003)
	Netherlands	W*	—	—	—	Jansen (1944)
	Scotland	W*	NG	1	—	SACVS (2006)
	Spain	W*	6A	2	—	Höfle et al. (2004)
		W	91	31	34	Villanua et al. (2006)

White-crowned Pigeon (*Patagioenas leucocephala*)	USA (Florida Keys)	W W*	41[†]12	36[‡] 12	88 100	Kocan and Sprunt (1971)
Band-tailed Pigeon (*Patagioenas fasciata*)	USA (Arizona)	W	156	8	5	Sileo and Fitzhugh (1969)
	USA (California)	W*	NG	NG	—	Stabler and Braun (1979)
		W*	NG	~2,000	—	NWHC (1995)
		W*	NG	~16,000	—	Cole (1999)
		W*	NG	~2,000	—	NWHC (2004)
		W*	NG	~300	—	NWHC (2006)
	USA (Colorado)	W	109	21	19	Stabler (1951)
Pink Pigeon (*Nesoenas mayeri*)	Mauritius	W	41	9	22	Swinnerton et al. (2005)
		W*	2,991	1,504	50[¶]	Bunbury (2006)
Seychelles Blue-Pigeon (*Alectroenas pulcherrima*)	Seychelles	W	3	1	—	Bunbury (personal communication, September 16, 2007)
Eurasian Collared-Dove (*Streptopelia decaocto*)	England	W*	NG	2	—	Cornelius (1972)
	England	W*	665	25	<1	Cousquer (2003)
	Germany	NG	NG	NG	—	Knispel (2005)
	Italy	W*	125	66	53	Delogu et al. (1997)
	Northern Ireland	W	NG	6	—	Beggs and Kennedy (2005)
	Scotland	W*	NG	"several"	—	SACVS (1994)
	USA (Florida)	W*	6	6	—	Forrester and Spalding (2003)
Ring Turtle-Dove (*Streptopelia* risoria)[§]	USA (California)	C*	11	11	100	Stabler and Herman (1951)
	USA (Iowa)	E*	14	14	100	Kietzmann (1993)
	USA (Iowa)	E*	36	16	44	Powell and Hollander (1982)
	USA (New York)	C*	288	136	47	Cauthen (1936)
Red-eyed Dove (*Streptopelia semitorquata*)	South Africa (Constantia)	W*	NG	NG	—	Pepler and Oettlé (1992)

(*continues*)

Table 6.1. (*Continued*)

Host	Location	Status	Number of birds		%	Literature source
			Examined	Infected		
Madagascar Turtle-Dove (*Streptopelia picturata*)	Mauritius	W	109	17	16	Swinnerton et al. (2005)
		W	247	115	47	Bunbury et al. (2007)
Spotted Dove (*Streptopelia chinensis*)	Australia (Sydney)	W*	1	1	—	Hart (1940)
	Malaysia	C*	8	3	—	Amin-Babjee et al. (1986)
	Mauritius	W	3	1	—	Swinnerton et al. (2005)
		W	32	6	19	Bunbury et al. (2007)
Laughing Dove (*Streptopelia senegalensis*)	Australia (Perth)	W	76	35	46	McKeon et al. (1997)
	Mauritius	W	4	2	—	Bunbury et al. (2007)
	South Africa (Constantia)	W*	NG	NG	—	Pepler and Oettlé (1992)
Zebra Dove (*Geopelia striata*)	Mauritius	W	9	3	—	Swinnerton et al. (2005)
		W	17	10	59	Bunbury et al. (2007)
	USA (Hawaii)	W*	2	2	—	Kocan and Banko (1974)
Mourning Dove (*Zenaida macroura*)	Canada (Ontario)	W*	NG	60	—	CCWHC (2007)
	USA (Alabama)	W*	204	5	2	Haugen and Keeler (1952)
		W*	NG	~50	—	NWHC (2003)
	USA (Arizona)	W*	NG	3	—	NWHC (1993)
		W*	NG	1	—	NWHC (1995)
		W*	NG	108	—	NWHC (2003)
		W	NG	NG	16	Hedlund (1998)
	USA (Arkansas)	W*	60	1	2	Stabler and Herman (1951)
		W	10	8	80	Barrows (1975)
	USA (California)	W*	450	200	45	Stabler and Herman (1951)
		W	55	21	38	Rupiper and Harmon (1988)
		W*	NG	~1,400	—	NWHC (2004)
	USA (Colorado)	W*	100	23	23	Stabler (1951)
	USA (Connecticut)	W*	40	28	70	Stabler and Herman (1951)
	USA (Florida)	W	142	10	7	Conti and Forrester (1981)

	W*	NG	5	—	NWHC (1989)
USA (Georgia)	W	10	1	10	Barrows (1975)
	W*	NG	5	—	NWHC (1998)
	W*	NG	~18	—	NWHC (2001)
	W*	NG	~40	—	NWHC (2002)
USA (Illinois)	C*	4	4	—	Stabler and Herman (1951)
	W*	NG	3	—	NWHC (1995)
USA (Indiana)	W*	NG	34	—	Barnes (1951)
USA (Iowa)	C*	NG	"several"	—	Stabler and Herman (1951)
USA (Kansas)	W*	NG	~15	—	NWHC (1993)
	W*	NG	13	—	NWHC (2003)
USA (Kentucky)	W	NG	3	—	Locke and Herman (1961)
USA (Maryland)	W*	32	1	3	Stabler and Herman (1951)
	W*	14N	2	14	Locke and Herman (1961)
		520J,A	13	3	
USA (Massachusetts)	W	80	2	3	Stabler and Herman (1951)
USA (Michigan)	W*	NG	~22	—	NWHC (1991)
USA (Missouri)	W*	NG	~10	—	NWHC (1998)
	W	4,052	226	6	Schulz et al. (2005)
	W*	1	1	—	Padilla et al. (2004)
USA (Nebraska)	W*	121A	57	47	Greiner and Baxter (1974)
		17J	7	41	
		7N	2	29	
USA (Nevada)	W*	NG	6	—	NWHC (2003)
USA (New Mexico)	W*	NG	~800	—	Cole (1999)
	W*	NG	~300	—	NWHC (1995)
USA (New York)	C*	5	4	—	Cauthen (1936)
	W*	NG	143	—	NWHC (1998)
USA (North Carolina)	W*	NG	~500	—	Cole (1999)
USA (Ohio)	W*	2N	1	—	Harwood (1946)
	W*	NG	NG	11	Stabler and Herman (1951)
	W*	NG	~195	—	NWHC (1983)

(*continues*)

Table 6.1. (*Continued*)

Host	Location	Status	Number of birds			Literature source
			Examined	Infected	%	
	USA (Oklahoma)	W	163A,J	21	13	Carpenter et al. (1972)
			20N	6	30	
	USA (Oregon)	W*	NG	~90	—	NWHC (1991)
	USA (Pennsylvania)	W*	NG	~24	—	NWHC (2003)
	USA (South Carolina)	W*	56A	9	16	Kocan and Amend (1972)
			252J	5	2	
		W*	NG	5	—	NWHC (2001)
		W*	NG	~12	—	NWHC (2002)
	USA (Tennessee)	W	NG	1	—	Locke and Herman (1961)
		W*	NG	~34	—	NWHC (2001)
	USA (Texas)	W	101N	53	52	Locke and Herman (1961)
	USA (Utah)	W	155	27	17	Ostrand et al. (1995)
		W*	230	1	<1	
	USA (Virginia)	W*	1	1	—	Stabler and Herman (1951)
		W*	6N	5	—	Sprunt (1957)
		W*	NG	~15	—	NWHC (2000)
		W	20	13	65	Barrows (1975)
	USA (West Virginia)	W*	NG	10	—	NWHC (1998)
		W*	NG	~15	—	NWHC (2000)
	USA (Wisconsin)	W*	2	2	—	Stabler and Herman (1951)
Galapagos Dove (*Zenaida galapagoensis*)	Ecuador (Galapagos Islands)	W	27	3	11	Harmon et al. (1987)
White-winged Dove (*Zenaida asiatica*)	USA (Arizona)	W	19A	11	58	Toepfer et al. (1966)
			23N	21	91	
		W	NG	NG	98	Hedlund (1998)
	USA (Florida)	W	25	25	100	Conti et al. (1985)

		W	67	65	97	Conti and Forrester (1981)
	USA (Texas)	W	17	6	35	Locke and Kiel (1960)
		W	51	51	100	Stabler (1961)
		W	97A	97	100	Glass et al. (2001)
		W	74J	73	99	
Common Ground-Dove	USA (Florida)	W	4	3	—	Stabler and Holt (1962)
(*Columbina passerina*)	USA (Texas)	W	4	3	—	Locke et al. (1961)
Inca Dove (*Columbina inca*)	USA (Arizona)	W	NG	NG	52	Hedlund (1998)
	USA (Texas)	W*	3	3	—	Locke and James (1962)
White-tipped Dove	Panama	E*	NG	NG	—	Callender and Simmons (1937)
(*Leptotila verreauxi*)						
	USA (Texas)	W	2	2	—	Hayse and James (1964)

A, adults; W, wild bird infected naturally; C, captive bird infected naturally; E, bird experimentally infected; J, juveniles; N, nestlings; NG, not given by authors.

*Lesions reported.

†Number of nests examined that contained squabs. The total number of squabs examined was not given.

‡Number of nests examined that contained at least one infected squab. The total number of infected squabs was not given.

§Clements (2000) did not list this as a valid species. Stevenson and Anderson (1994) stated that "According to Goodwin (1967) and other authorities, the turtle-dove, long kept in captivity, was derived from the African Collared-Dove (*Streptopelia roseogrisea*), native to North Africa and Arabia. There is some doubt as to whether it is now specifically distinct from that species."

¶This prevalence value is derived from multiple examinations of 426 Pink Pigeons over a 20-month period. The numbers examined here and the numbers positive for *Trichomonas gallinae* are actually the number of examinations rather than the numbers of birds examined. Many birds were examined multiple times.

Table 6.2. Reports of *Trichomonas gallinae* from free-ranging and captive falconiforms and strigiforms.

Host	Location	Status	Number of birds		%	Literature source
			Examined	Infected		
Falconiforms						
Black Kite (*Milvus migrans*)	South Africa	W*	1	1	—	Pepler and Oettlé (1992)
Bald Eagle (*Haliaeetus leucocephalus*)	USA (New York)	W*	1	1	—	Rettig (1978)
		W*	2	2	—	Stone and Nye (1981)
Egyptian Vulture (*Neophron percnopterus*)	England	C*	1	1	—	Keymer (1972)
Shikra (*Accipiter badius*)	England	C*	1	1	—	Keymer (1972)
African Goshawk (*Accipiter tachiro*)	South Africa	W*	2	2	—	Oettlé (1990); Pepler and Oettlé (1992)
Rufous-chested Sparrowhawk (*Accipiter rufiventris*)	South Africa	W*	1	1	—	Oettlé (1990); Pepler and Oettlé (1992)
Cooper's Hawk (*Accipiter cooperii*)	USA (Arizona)	W*	144N	115	79	Boal and Mannan (1999)
	USA (Pennsylvania)	E*	2	1	—	Stabler (1941b)
		W*	89A	1	<1	Boal et al. (1998)
			233N	140	60	
	Canada (British Columbia)	W	NG	3N	—	Rosenfield et al. (2002)
Western Marsh-Harrier (*Circus aeruginosus*)	Spain	W*	NG	1J	—	Knispel (2005)
Northern Goshawk (*Accipiter gentilis*)	England	W*	36	14	39	Cooper and Petty (1988)
	Germany	W*	269N	175	65	Krone et al. (2005)
	Poland	W*	39N	61	64	Wieliczko et al. (2003)

Gray Hawk (*Buteo nitidus*)	USA (Arizona)	W*	2	2	—	Stensrude (1965)
Red-shouldered Hawk (*Buteo lineatus*)	USA (Florida)	W*	1	1	—	Forrester and Spalding (2003)
	USA (Pennsylvania)	E*	1	1	—	Stabler and Shelanski (1936)
Red-tailed Hawk (*Buteo jamaicensis*)	USA (Florida)	W*	1	1	—	Forrester and Spalding (2003)
Eurasian Buzzard (*Buteo buteo*)	England	W*	374	9	<1	Cousquer (2003)
	Germany	W	NG	1	—	Knispel (2005)
	South Africa	W*	1	1	—	Oettlé (1990); Pepler and Oettlé (1992)
Golden Eagle (*Aquila chrysaetos*)	USA (Idaho/Oregon)	W*	10	4	40	Beecham and Kochert (1975)
	USA (Pennsylvania)	E*	1	1	—	Stabler (1941b)
Bonelli's Eagle (*Aquila fasciata*)	Portugal	W*	12N	6	50	Höfle et al. (2000)
	Spain	W*	39	14	36	Real et al. (2000)
		W	NG	3N	—	Knispel (2005)
Booted Eagle (*Aquila pennata*)	Spain	W*	NG	1A	—	Knispel (2005)
Secretary-Bird (*Sagittarius serpentarius*)	Japan/Republic of Botswana[†]	C?*	6	3	—	Koyama et al. (1971)
Lesser Kestrel (*Falco naumanni*)	Bahrain	C*	NG	5	—	Samour et al. (1995)
	Spain	W*	NG	1J	—	Knispel (2005)
Eurasian Kestrel (*Falco tinnunculus*)	Bahrain	C*	NG	7	—	Samour et al. (1995)
American Kestrel (*Falco sparverius*)	USA (New York)	W*	NG	1	—	Tangredi (1978)
		W*	1	1	—	Stone and Janes (1969)
	USA (Pennsylvania)	C*	1	1	—	Stabler and Shelanski (1936)

(*continues*)

Table 6.2. (*Continued*)

Host	Location	Status	Number of birds		%	Literature source
			Examined	Infected		
Red-necked Falcon (*Falco chicquera*)	England	C*	1	1	—	Keymer (1972)
Merlin (*Falco columbarius*)	USA (Pennsylvania)	C	1	1	—	Stabler (1969)
Lanner Falcon (*Falco biarmicus*)	Bahrain	C*	NG	310	—	Samour et al. (1995)
	Saudi Arabia	C*	NG	8	—	Samour and Naldo (2003)
Saker Falcon (*Falco cherrug*)	Bahrain	C*	NG	1,345	—	Samour et al. (1995)
	Saudi Arabia	C*	2	2	—	Samour (2000a)
		C*	12	12	100	Samour (2000b)
		C*	NG	346	—	Samour and Naldo (2003)
Gyrfalcon (*Falco rusticolus*)	USA (Colorado)	C*	1	1	—	Hamilton and Stabler (1953)
	USA (Pennsylvania)	C	1	1	—	Stabler (1969)
	Saudi Arabia	C*	NG	5	—	Samour and Naldo (2003)
Peregrine Falcon (*Falco peregrinus*)	Canada (Saskatchewan)	W*	NG	1	—	CCWHC (2007)
	England	W*	NG	1	—	DEFRA (2003)
	Germany	NG	NG	NG	—	Knispel (2005)
	Saudi Arabia	C*	NG	30	—	Samour and Naldo (2003)
	South Africa	C*	1	1	—	Pepler and Oettlé (1992)
	Spain	W*	NG	1N	—	Knispel (2005)
	USA (Pennsylvania)	W	10	2	20	Stabler (1941b)
Strigiforms						
Barn Owl (*Tyto alba*)	England	W*	180	1	<1	Hardy et al. (1981)
		W*	98	1	<1	Cousquer (2003)
	Italy	W*	20	10	50	Delogu et al. (1997)
	South Africa	W*	1	1	—	Pepler and Oettlé (1992)
	Spain	W*	NG	1A	—	Knispel (2005)

	USA (California)	W*	1,638	40	2	Schulz (1986)
		W*	8	8	—	Pokras et al. (1993)
	USA (Hawaii)	W*	81	20	25	Work and Hale (1996)
	USA (Louisiana)	W	NG	1	—	Pokras et al. (1993)
European Scops-Owl (*Otus scops*)	Italy	W*	10	1	10	Delogu et al. (1997)
Eastern Screech-Owl (*Megascops asio*)	USA (Florida)	W*	3	1	—	Forrester and Spalding (2003)
Great Horned-Owl (*Bubo virginianus*)	Canada (Ontario)	W*	1	1	—	CCWHC (2007)
	USA (California)	W*	3	3	—	Jessup (1980)
	USA (Florida)	W*	NG	1	—	Forrester and Spalding (2003)
Eurasian Eagle-Owl (*Bubo bubo*)	Germany	C	NG	1	—	Knispel (2005)
Spotted Eagle-Owl (*Bubo africanus*)	South Africa	W*	1	1	—	Oettlé (1990); Pepler and Oettlé (1992)
Tawny Owl (*Strix aluco*)	England	W*	338	10	<1	Cousquer (2003)
	Italy	W*	40	11	28	Delogu et al. (1997)
Barred Owl (*Strix varia*)	USA (Florida)	W*	NG	3	—	Forrester and Spalding (2003)
	USA (Louisiana)	W	NG	2	—	Pokras et al. (1993)
	USA (Massachusetts)	W	1	1	—	Pokras et al. (1993)
Little Owl (*Athene noctua*)	Italy	W*	118	1	1	Delogu et al. (1997)

NG, not given; A, adults; J, juveniles; N, nestlings; W, wild bird infected naturally; C, captive bird infected naturally; E, bird experimentally infected.

*Lesions reported.

†These birds were captured in Botswana and subsequently brought to Japan. Eleven to thirteen days after being put in a zoo, three of the birds died with lesions compatible with trichomonosis. Therefore, it is not known whether the birds were infected in Botswana or in Japan.

the liver of a pigeon and called it *Cercomonas hepaticum*. Later, both these species of *Cercomonas* were recognized as *T. gallinae* and were considered synonyms (Stabler 1938). Taxonomically, *T. gallinae* is in the family Trichomonadidae (phylum Parabasalia, order Trichomonadida) and is closely related to several other parasitic flagellates of veterinary and medical importance, including *Tritrichomonas foetus* in cattle, *Trichomonas phasiani* in game-farm pheasants, and *Trichomonas vaginalis* in humans. The molecular phylogeny of *T. gallinae* and other trichomonads has been studied extensively during the past 10 years (see Kleina et al. 2004; Cepicka et al. 2005, 2006; Gaspar da Silva et al. 2007).

The living organism varies from pear-shaped to round and measures from 12.5 to 20 μm in length, has four anterior flagella, no free posterior flagellum, an axostyle that protrudes posteriorly, and a well-developed undulating membrane (BonDurant and Honigberg 1994) (Figures 6.2a–6.2c). Recently, it has been discovered that *T. gallinae* has pseudocyst stages (Tasca and DeCarli 2003). These are spherical forms that internalize the flagella, do not have a true cyst wall (Figures 6.2d–6.2f), and behave as resistant forms under stressful environmental conditions. Their formation is reversible (Pereira-Neves et al. 2003). Their role in the epidemiology of trichomonosis is not known, but pseudocysts of a related species, *T. foetus*, have been found to adhere to vaginal epithelial cells at a higher ratio than the trophozoite forms and may have some role in host cell infectivity (Mariante et al. 2004). For additional details on morphology, the reader is referred to Stabler (1941a), Abraham and Honigberg (1964), and Tasca and DeCarli (2003).

Trichomonas gallinae reproduces by binary fission (Stabler 1941a) and grows readily in agnotobiotic and axenic cultures in a number of media, both liquid and semiliquid (BonDurant and Honigberg 1994). As a result, there is an extensive amount known about carbohydrate, nitrogen, and lipid metabolism and various nutritional requirements of this organism (see reviews by Stabler (1954) and BonDurant and Honigberg (1994)).

It has been known for some time that there are variations in the strains of *T. gallinae* (Stabler 1954; BonDurant and Honigberg 1994). Most strains are either nonpathogenic or moderately pathogenic, but there are also virulent strains. One of the most virulent is the Jones' Barn (JB) strain that kills nonimmune pigeons at approximately 8 days postinfection (PI). The pathogenicity of the JB strain decreases when the organism is grown in nonliving media and is restored subsequently when the strain is passed serially in nonimmune pigeons (Stabler et al. 1964). Another strain, the avirulent Amherst (AG) strain, loses its infectivity to pigeons after prolonged growth in culture (Honigberg 1979). Although strains can lose their pathogenicity and infectivity while being cultured, no changes in virulence or antigenic properties have been reported after 12 years of cryopreservation (Bosch and Frank 1972). DNA and RNA from a virulent stain can increase the virulence of a nonpathogenic strain grown in culture (Honigberg et al. 1971).

The pathogenicity of strains of *T. gallinae* has been determined by several techniques. One is the use of a subcutaneous mouse assay developed and evaluated by Honigberg (1961) and Frost and Honigberg (1962). This assay consists of injection of quantified numbers of axenically cultured trophozoites of *T. gallinae* under the skin of purebred mice and measurement of the mean volumes of any subsequent lesions at 6 days after injection. There is a close correlation between the lesion volumes and pathogenicity, larger lesions resulting from highly pathogenic strains. Two virulent strains (JB and Eiberg or IBERG) were distinguished from the avirulent Stabler-gallinae (SG) strain by the use of isoenzyme electrophoresis (Nadler and Honigberg 1988). Mattos et al. (1997) were also able to distinguish between three strains of *T. gallinae* by the use of isoenzyme electrophoresis, but pathogenicity of the three strains was not reported. Restriction enzyme analyses have been used to distinguish between strains of *T. gallinae* from several avian species (pigeons, raptors, canaries, and parakeets), but the technique has not been applied to comparisons of pathogenic versus nonpathogenic strains (Knispel 2005). Hemolysis of erythrocytes has been correlated with pathogenicity of strains of *T. vaginalis* (Dailey et al. 1990), but this has not been determined to be useful for the differentiation of pathogenic and nonpathogenic strains of *T. gallinae*. Comparative sequence analyses of 5.8S rRNA genes and internal transcribed spacer regions have been used to identify genera and species of trichomonads, including *T. gallinae*. This technique was used to study 24 isolates of *T. gallinae*—19 from Pink Pigeons (*Nesoenas mayeri*) and 5 from Madagascar Turtle Doves (*Streptopelia picturata*) from 6 different sites on the Indian Ocean island of Mauritius (Gaspar da Silva et al. 2007). All isolates had identical sequences both to each other and to an unrelated but previously sequenced isolate of *T. gallinae*, indicating that the locus (ITS1/5.8S/ITS2) can be used as a species marker. Random amplified polymorphic DNA analyses of these same isolates allowed the authors to identify geographic and host species differences, indicating that these were different strains of *T. gallinae*. These techniques have not been applied to the differentiation of pathogenic and nonpathogenic strains of *T. gallinae* (Felleisen 1997; Knispel 2005; Gaspar da Silva et al. 2007). Further studies on these techniques are needed so that assays can be developed to readily identify

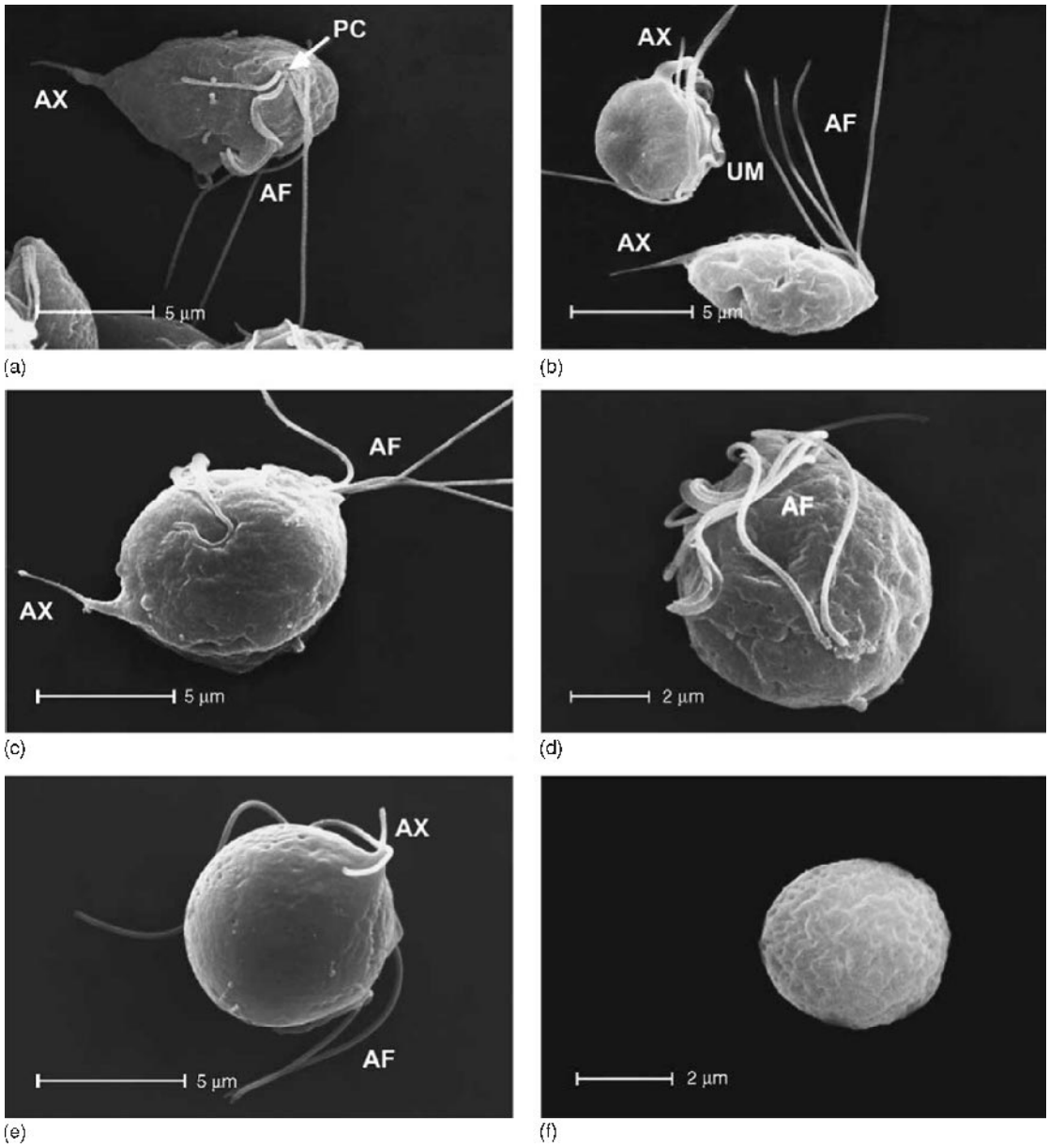


Figure 6.2. Scanning electron micrographs of trophozoites (a–c) and the formation of pseudocysts (d–f) of a strain of *Trichomonas gallinae* obtained from a Rock Pigeon (*Columba livia*) and cultured axenically in vitro. Note the invagination of the flagellae and disappearance of the undulating membrane in the organisms shown in (d) and (e). Part (f) represents a pseudocyst. AF, anterior flagella; UM, undulating membrane; AX, axostyle; PC, periflagellar canal. Reprinted from Tasca and DeCarli (2003), with permission of *Veterinary Parasitology*.

pathogenic strains of *T. gallinae*. Such assays would revolutionize our understanding of many epizootiological aspects of trichomonosis and the impact of this disease on populations of wild birds.

Some strains exhibit sites of tissue/organ predilection. For example, the JB strain and the Eiberg strains are predominantly hepatotrophic while the Mirza strain primarily infects the head (sinuses, orbital regions,

brain, and neck tissues) and the mucosa of the upper digestive tract (Narcisi et al. 1991; BonDurant and Honigberg 1994). When the JB strain was given to Mourning Doves (*Zenaida macroura*), however, the lesions occurred predominately in the lungs rather than in the liver (Kocan 1969b). This is usually the case with infections with the JB strain in Rock Pigeons.

EPIZOOTIOLOGY

Among columbiforms, the primary source of infection by *T. gallinae* is the Rock Pigeon (Stabler 1954; BonDurant and Honigberg 1994). Some pigeons and doves develop immunity to the harmful effects of virulent strains of *T. gallinae* because of previous infection with an avirulent strain and act as carriers. These birds can have concomitant infections of both virulent and avirulent strains that can be transmitted to other birds, a virulent strain on one occasion and an avirulent strain during another (Kocan and Herman 1971).

The life cycle of *T. gallinae* is direct, the organism being passed from one host to another without involvement of intermediate or paratenic hosts. There are no resistant cyst stages (although there are pseudocysts; see Etiology section) and the trichomonads are very sensitive to desiccation (Kocan and Herman 1971). Transmission from host to host occurs by several methods. In the case of columbiforms, the organism is transferred directly from the upper digestive tract and mouth cavity of infected adults to squabs via regurgitation of pigeon milk produced in the crop of the adult bird (Stabler 1947; Kietzmann 1990). Thus, newly hatched squabs become infected during their first feeding. Transmission also occurs via direct contact between infected and uninfected columbiforms while cross-feeding or billing during courtship (Kocan and Herman 1971). Infected birds with oral or throat lesions have difficulty swallowing large pieces of grain and will pick them up, contaminate them with the organism, and subsequently drop them (Kocan and Herman 1971). These contaminated pieces of grain can then be ingested by an uninfected bird. Under normal circumstances, both uninfected doves and pigeons and those infected with avirulent strains of *T. gallinae* will also drop seeds that they pick up while feeding. These contaminated seeds can be ingested by another susceptible uninfected bird. A third method is by the ingestion of contaminated water. *Trichomonas gallinae* can live for 20 min to several hours in water depending on the salinity and for at least 5 days in moist grains (Kocan 1969a). Transmission of *T. gallinae* via contaminated drinking water was demonstrated experimentally by Kietzmann (1990) using caged Ring Turtle Doves (*Streptopelia risoria*). Using InPouch culture kits (see Diagnosis section), Bunbury et al. (2007) cultured water from sources utilized by infected columbids in Mauritius and found that 2 of 15 samples were positive for *T. gallinae*. Galliforms, psittaciforms, and passeriforms are infected by using the same feeding and watering areas as infected columbiforms. Raptors, however, are infected by feeding on infected prey, especially columbiforms (Stabler 1941b). *Trichomonas gallinae* has been shown to survive in dove carcasses for up to 48 h after death of the host (Erwin et al. 2000). Pseudocysts might have a role in this survival, which in turn could influence the transmission of the disease to raptors that feed on these carcasses, but this has not been investigated.

Trichomonosis can result from the transfer of only one trichomonad. This was shown by Stabler and Kihara (1954), who infected five Rock Pigeons each with one trichomonad of the highly pathogenic JB strain; all five birds died with typical lesions and signs of trichomonosis within 8–14 days after infection.

The prevalences of *T. gallinae* in various hosts from a variety of geographical locations are presented in Table 6.1 for columbiforms and Table 6.2 for raptors. The Rock Pigeon is the most commonly and widely reported host, and infected birds have been recorded from 31 countries. The prevalence, based on examinations of 4,778 Rock Pigeons from 17 countries, ranged from 7 to 100%, while the mean was 47%. Next to the Rock Pigeon, the Mourning Dove in the US has been studied the most extensively. Prevalences, based on examinations of 6,932 doves from 20 states, ranged from 2 to 100%, with a mean prevalence of 11%. These values for both Rock Pigeons and Mourning Doves are probably underestimates since most of the earlier reports were based on standard wet-mount microscopy which has been shown to be considerably less sensitive than culture or polymerase chain reaction (PCR) techniques that have been applied more recently (Bunbury et al. 2005).

Bunbury (2006) found no differences in prevalence between sexes in endangered Pink Pigeons in Mauritius and an increasing probability of infection with increasing age. She also found that although higher temperatures and lower rainfall were associated with higher prevalences in Madagascar Turtle-Doves in Mauritius (Bunbury et al. 2007), temperature had the most significant influence.

Although a determination of the prevalence of the etiologic agent *T. gallinae* in a population of birds by swab, culture, and PCR techniques is useful information, these values provide only minimal epizootiological data. This is because the number of birds that had been infected at one time, but are free of infection and thereby immune at the time of sampling, is unknown. Kocan and Knisley (1970) used a challenge technique as a means to determine the prevalence of immune birds in a population. They livetrapped Rock Pigeons

and Mourning Doves in the Maryland–Washington DC area and determined by swab and culture techniques that the prevalence of infection by *T. gallinae* was 52% for the Rock Pigeons and 0% for the Mourning Doves. When the negative birds were challenged subsequently with the JB strain of *T. gallinae*, all became positive for the organism (trichomoniasis), but 88% of the Rock Pigeons and 82% of the Mourning Doves were immune and did not develop the disease (trichomonosis).

As discussed in the Immunity section, the duration and loss of infections and the possibility of premunition could both be important factors in the epidemiology of trichomonosis. When the same individuals were tested every 2 months over a 20-month period in Mauritius, Pink Pigeons were more likely to remain either positive or negative than they were to acquire or lose infections of *T. gallinae* (Bunbury 2006). However, a number of birds also gained and lost infections several times over the screening period. More information is needed on this topic.

Information on the relative importance of trichomonosis as a mortality factor in avian populations is sparse. Although studies of carcasses submitted for necropsy are sometimes limited by collection biases, they can provide some insights into causes of death when interpreted with caution. In Mauritius, 54% of 35 free-living Pink Pigeons found dead over a 4-year period died of trichomonosis, the leading cause of death (Bunbury 2006). Similarly, Gerhold et al. (2007) reported that trichomonosis accounted for 40% of the cases and was the leading cause of death of 135 Mourning Doves submitted for diagnostic determination from 8 southeastern states in the US over a 35-year period.

The factors that trigger an epizootic of trichomonosis are unknown, although a number of suggestions have been made, particularly in the case of Rock Pigeons living in a pigeon loft. These include improper food, crowding, poor ventilation, wet and dirty litter, and lack of sunlight (Stabler 1947). It is uncertain if such factors apply to other columbiforms. A massive outbreak of trichomonosis in Common Wood-Pigeons (*Columba palumbus*) on wintering roosts in southern Spain during 2001 was attributed to concentrations of Wood-Pigeons at birdfeeders that were numerous and set up for pheasants and partridges at a nearby hunting estate (Höfle et al. 2004). It was felt that transmission was enhanced by contamination of grain at the feeders as described earlier (see also Kocan 1969a).

CLINICAL SIGNS

Most of the clinical signs of infected columbiforms are related to the oral lesions which prevent or impair feeding. These include weight loss, listlessness, and ruffled feathers. Yellowish caseous lesions can be seen around the beak or eyes of infected birds and their faces look swollen (Cole 1999). Also, there can be an excess of watery saliva and a foul cheese-like smell (Bunbury 2006). Some of these same signs can be seen in infected raptors along with dyspnea and nasal and oral exudation (Pepler and Oettlé 1992). Signs in captive psittaciforms such as Budgerigars (*Melopsittacus undulatus*) include wasting, matted feathers, diarrhea, and repeated vomiting (Baker 1986; Murphy 1992).

PATHOGENESIS AND PATHOLOGY

As mentioned previously, infections by some strains of *T. gallinae* occur in the absence of apparent disease (i.e., trichomoniasis), while others vary from being mildly pathogenic to very pathogenic (i.e., trichomonosis) (Stabler 1948a; Kocan and Herman 1971). This variation is thought to be related to a greater antigenic diversity in avirulent strains that may stimulate a stronger host immune response than occurs during infection with more virulent strains with lower antigenic diversity (Stepkowski and Honigberg 1972). Infection with mild strains results in excessive salivation and some inflammation of the oral cavity and throat, whereas infections with more virulent strains result in caseous lesions in the mouth (Figure 6.3), throat, and crop and even invasion of the sinuses, skull, and skin of the neck. Some highly virulent strains cause lesions only in the head, neck, and crop, but other strains invade internal organs such as liver, lungs, pericardium, air sacs, and pancreas via the bloodstream (Stabler and Engley 1946; Jaquette 1950; Kocan and Herman 1971).

In severe cases, death can result as early as 4 days after infection. Kocan and Herman (1971) described the progression of the disease. Oral lesions are well-circumscribed, yellow masses located on the floor or roof of the mouth or in the pharyngeal region. Early lesions may be small and flush with the surface of the epithelium, but later they often develop small spur-like projections in their centers and may coalesce to form large, caseous masses in the mouth and throat. These can completely block the passage of food, so that the bird becomes emaciated and dies of starvation. Death may also occur as a result of respiratory failure if the lesion blocks the trachea (Kocan and Herman 1971) or hepatic dysfunction if organisms invade the liver (Narcisi et al. 1991). Lungs and other organs may be involved in infections by highly pathogenic strains. Host factors rather than characteristics of the etiologic agent may be more important in determining which organs are invaded. For example, the highly pathogenic JB strain of *T. gallinae* invades the liver of Rock Pigeons, whereas the same strain invades the lungs of Mourning Doves (Kocan 1969b).

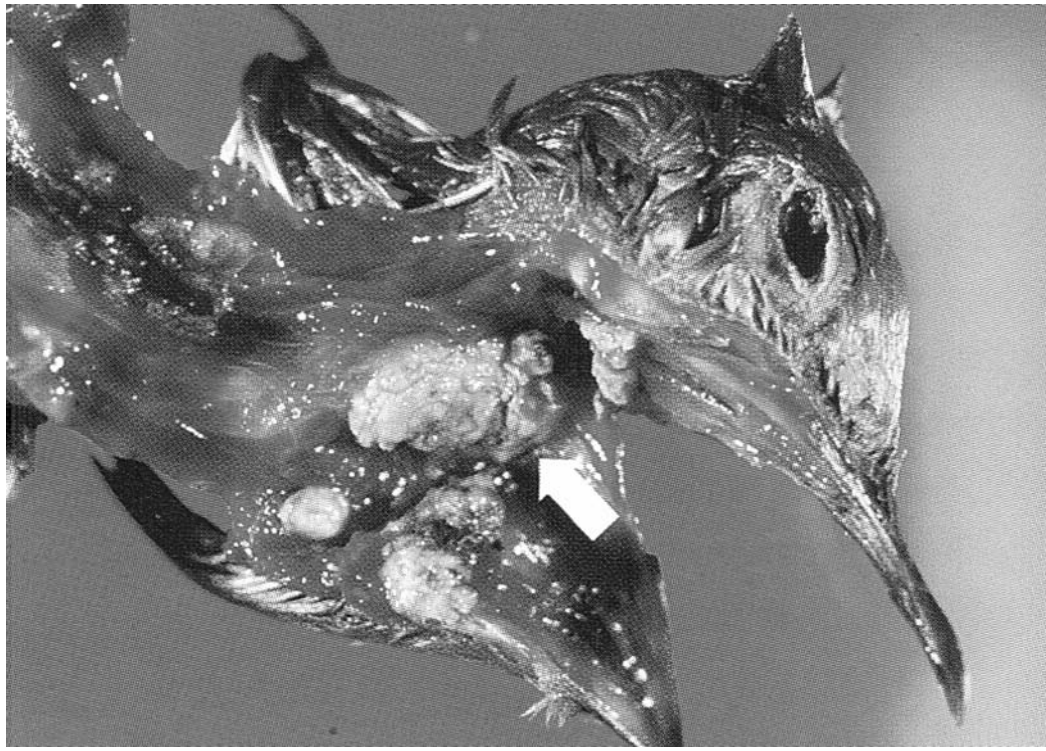
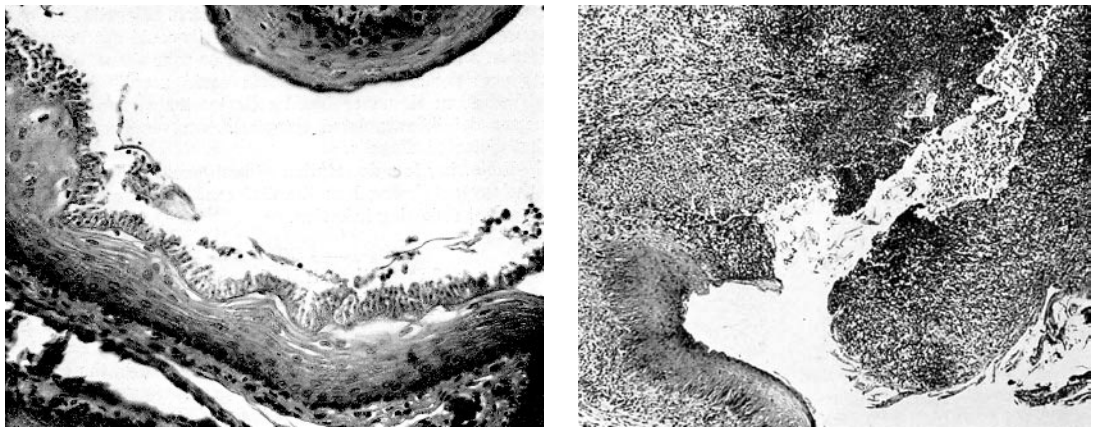


Figure 6.3. Gross lesion (arrow) of *Trichomonas gallinae* in the oral cavity of a Mourning Dove (*Zenaida macroura*). Reproduced from Cole (1999), with permission of the author.

Histopathological changes associated with infections of pathogenic strains of *T. gallinae* in Rock Pigeons have been studied experimentally by Perez-Mesa et al. (1961) for the JB strain and by Narcisi et al. (1991) for the Eiberg strain. Times required for the trichomonads to reach the liver and cause death were 3 and 7–10 days PI for the JB strain and 7 and 14–17 days for the Eiberg strain, but other histopathological findings were similar. Highlights of the histopathological findings of Perez-Mesa et al. (1961) follow. On day 2, trichomonads were arranged side by side, perpendicular to the surface of the epithelium of the pharynx and formed a layer that resembled columnar epithelium (Figure 6.4a). There was no inflammatory reaction in this area except for a mild mononuclear reaction near the gland openings. On day 3, there were occasional shallow pharyngeal ulcers with an infiltration of leukocytes in the submucosa around the glands. In one bird, there were lung abscesses with necrotic centers surrounded by lymphocytes, mononuclear cells, and rare giant cells. Trichomonads were seen between the necrotic centers and the peripheral normal lung tissues. They were also seen in the sinusoidal capillaries and in Disse's spaces in the liver. There were abscesses in the liver described as focal necrosis with infiltrations of mononuclear cells and heterophils. On day 4, pharyngeal ulcers had massive inflammatory reactions. The liver contained large advanced abscesses with necrotic areas surrounded by heterophils, mononuclear cells, and trichomonads. On days 5 and 7, the pharyngeal ulcers became deeper (Figure 6.4b) and the liver abscesses were larger than on day 4. On day 7, trichomonads were seen close to the vessels in the pharynx and were very numerous near the periphery of hepatic abscesses. During the course of this study, birds died between days 5 and 10 PI. Perez-Mesa et al. (1961) described the basic pathological response in Rock Pigeons infected with the JB strain as purulent inflammation. They further concluded that the pigeons died of massive hepatic destruction.

Further insights into the pathogenesis of trichomonosis were reported by Kietzmann (1993) in a study using scanning electron microscopy. He infected juvenile Ring Turtle-Doves with a pathogenic strain of *T. gallinae* obtained from a Rock Pigeon and followed the progression of infection for 240 h PI, with special emphasis on the events prior to canker formation. Between 6 and 19 h PI, small numbers of amoeboid



(a) (b)

Figure 6.4. Histopathological changes related to experimental infection of a Rock Pigeon (*Columba livia*) with the Jones' Barn strain of *Trichomonas gallinae*. (a) Trichomonads arranged perpendicularly to the surface of intact squamous epithelium in the pharynx on day 2 postinfection. 200×. (b) Ulcer in the pharynx on day 5 postinfection. The center of the ulcer consists of necrotic purulent exudate surrounded by mononuclear cells. 80×. Reproduced from Perez-Mesa et al. (1961), with permission of *Avian Diseases*.

trophozoites of *T. gallinae* attached to microfolds and cell borders of squamous epithelium of the palatal–esophageal junction (Figure 6.5a). Kietzmann (1993) postulated that some unknown parasite-secreted factor initiated squamous cell damage, separation, and removal. This was followed by invasion of areas beneath the squamous cells by trichomonads (Figure 6.5b) and accelerated desquamation, invasion of the mucosa, and the development of cankers between 19 and 240 h PI (Figures 6.6 and 6.7).

There are no comparable experimental studies of the pathogenesis of trichomonosis in birds of prey, psittaciforms, and other birds. However, a number of authors have described the lesions of trichomonosis in raptors (Jessup 1980; Cooper and Petty 1988; Pokras et al. 1993; Samour et al. 1995; Heidenreich 1997; Samour 2000a). Infected areas include oral and nasal cavities, esophagus, crop, and sometimes soft tissues, the skull, and various internal organs such as the heart. Stomatitis due to bacterial infection by *Pseudomonas aeruginosa* has been reported as a sequel to trichomonosis in captive Saker Falcons (*Falco cherrug*) in Saudi Arabia (Samour 2000b).

In captive psittaciforms, the disease has been reported to involve the oral cavity, crop, esophagus, pharynx, inner nares, sinuses, and other parts of the respiratory tract (Ruhl et al. 1982; Baker 1986; Ramsay et al. 1990; Garner and Sturtevant 1992; Murphy 1992).

Intracellular viruses or virus-like particles have been found in some strains of *T. vaginalis* and *T. foetus* (Benchimol et al. 2002; Vancini and Benchimol 2005). The role of these viruses in the pathogenesis of the diseases caused by these trichomonads is not understood. Such viruses or virus-like particles have not been identified in *T. gallinae*.

DIAGNOSIS

The presence of trichomonads can be determined by microscopic examination of wet smears prepared with sterile cotton-tipped swabs from the mucus of the mouth and oropharyngeal area for the presence of motile, flagellated protozoans (BonDurant and Honigberg 1994). In situations where the numbers of organisms are low, it is helpful to inoculate scrapings into a suitable growth medium and examine samples after the organisms have had time to multiply. *Trichomonas gallinae* grows readily in a variety of liquid and semisolid media. Diamond's medium or a modification of it has been used by a number of authors (Diamond 1954; Kocan and Amend 1972) and these were discussed by Stabler (1954) and BonDurant and Honigberg (1994). Cover et al. (1994) used a commercial product originally designed to culture infections of *T. foetus* in cattle (InPouch TF, BioMed Diagnostics, White City, Oregon, USA). These pouches had the same sensitivity as Diamond's medium and were convenient and effective for use in the field. This InPouch system has been used successfully in a number of studies involving columbiforms (Glass et al. 2001; Schulz

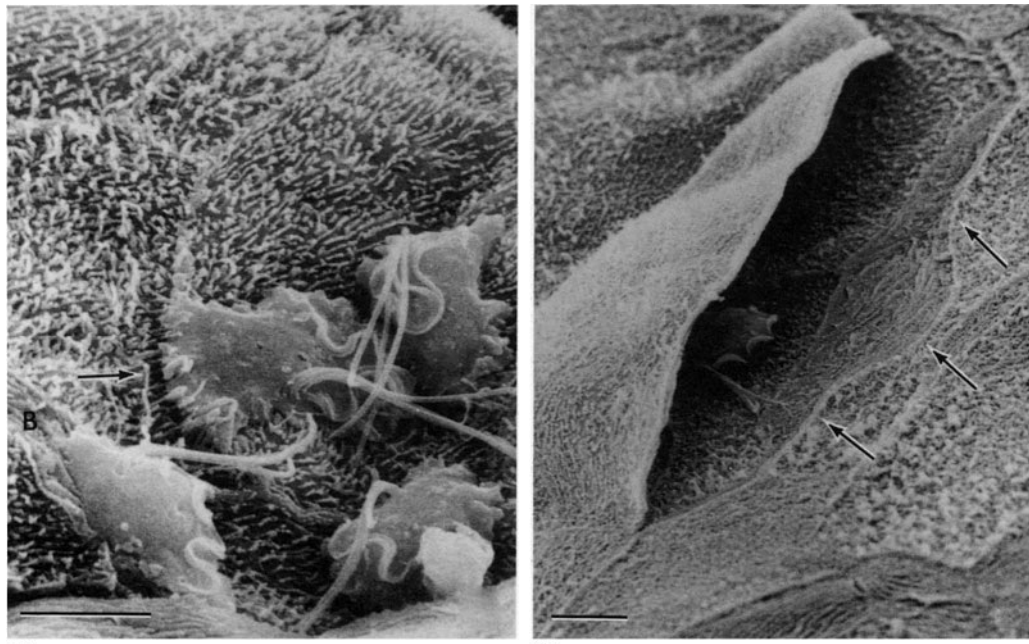


Figure 6.5. Scanning electron photomicrographs of the palatal–esophageal junction of a Ring Turtle-Dove (*Streptopelia risoria*) infected experimentally with a pathogenic strain of *Trichomonas gallinae*. (a) Nineteen hours postinfection (PI). One trichomonad (arrow) is located at a squamous cell border and another is involved at a cell border separation (B). (b) Nineteen hours PI. A trichomonad can be seen under a loosened squamous cell within the intercellular space. Border remnants (arrows) can be seen where the cell was once attached to other cells. Scale bars = 5 μm. Reproduced from Kietzmann (1993), with permission of *The Journal of Parasitology*.

et al. 2005; Bunbury et al. 2007) and raptors (Boal et al. 1998). Bunbury et al. (2005) found the sensitivity of the InPouch system to be more than twice that of conventional wet-mount microscopy.

Definitive identification of trichomonads is accomplished by the amplification of the 5.8S rRNA region by PCR. Since the assay was first developed (Felleisen 1997), it has been used for studies on *T. vaginalis* in humans (e.g., Mayta et al. 2000), *T. foetus* in cattle (e.g., Grahn et al. 2005), as well as *T. gallinae* in birds (e.g., Höfle et al. 2004; Villanua et al. 2006; Gaspar da Silva et al. 2007). At least two commercial companies in North America offer diagnostic testing of samples for *T. gallinae* by PCR (Zoologix Inc., Chatsworth, California and HealthGene Corporation, Toronto, Ontario, Canada). Liebhart et al. (2006) developed an in situ hybridization procedure for detecting *Histomonas meleagridis* in paraffin-embedded tissue samples and also evaluated probes for *T. gallinae* and other organisms. Their probes were specific for *H. meleagridis*, but could not differentiate between *T. gallinae* and *Tetratrichomonas gallinarum*. Refinement of this technique might be possible and would provide a useful diagnostic tool for retrospective studies of trichomonosis in birds.

Lesions in the throat or oral cavity of a living or dead bird can also be used to obtain a diagnosis of trichomonosis. A definitive diagnosis of the disease is made by demonstrating the presence of the organism by PCR assay and by observing the typical lesions. Lesions caused by fungi (*Aspergillus* sp., *Candida* sp.), poxvirus, nematode infections (*Capillaria* sp.), or vitamin A deficiency can be similar superficially to those of trichomonosis and this should be considered when making a diagnosis (Kocan and Herman 1971; Cole 1999). Birds that recover from trichomonosis often lack pharyngeal folds (located in the back of the throat) as a result of the necrotizing process that occurs during

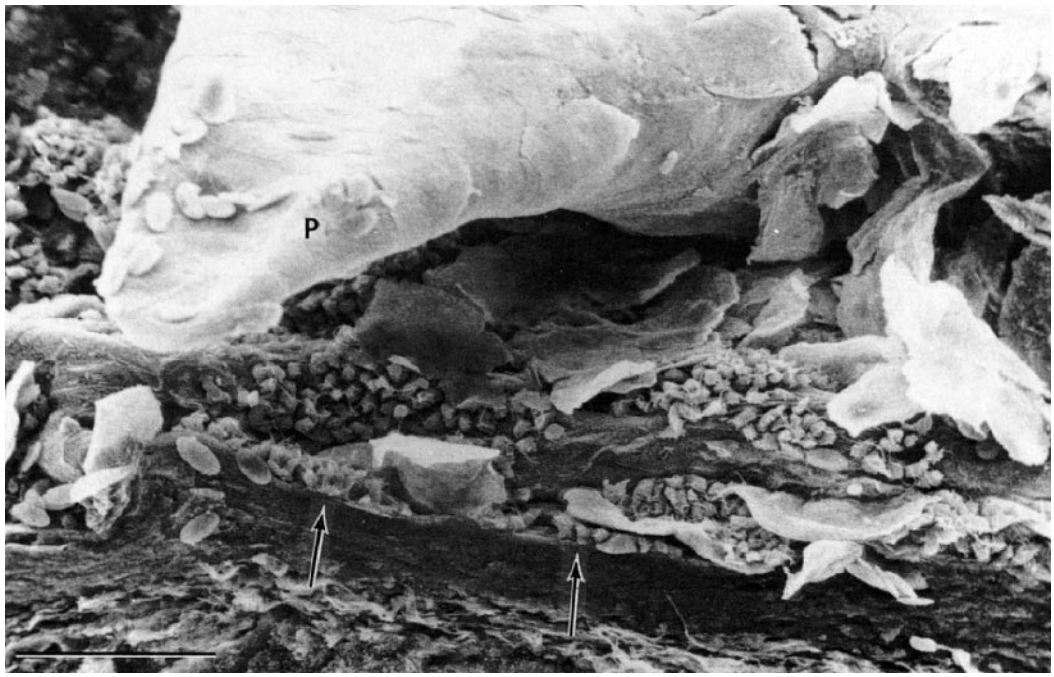


Figure 6.6. Scanning electron photomicrograph of the palatal–esophageal junction of a Ring Turtle-Dove (*Streptopelia risoria*) 48 h after being infected experimentally with a pathogenic strain of *Trichomonas gallinae*. Note the extensive desquamation of squamous cell sheets and palisading of trichomonads (arrows). A palatal papilla is labeled (P). Scale bar = 50 μm. Reproduced from Kietzmann (1993), with permission of *The Journal of Parasitology*.

infection. This observation can be helpful in identifying previous cases of disease, at least in some species (Kocan and Herman 1971).

IMMUNITY

Several authors have reviewed immunologic aspects of infection with *T. gallinae* in columbiforms (Stabler 1954; Kocan and Herman 1971; Honigberg and Lindmark 1987; BonDurant and Honigberg 1994). Kocan and Amend (1972) challenged Mourning Doves from an epizootic area in South Carolina and from an area in Maryland where no epizootic had occurred for at least 3 years. Eighty-five percent of the doves from the epizootic area were immune to trichomonosis, whereas only 69% of the doves from the nonepizootic area were immune.

Birds infected with a moderately virulent or avirulent strain of *T. gallinae* have strong protection against the pathogenic effects of a subsequent infection by a virulent strain (Stabler 1948b). This immunity has both cellular and humoral components. Phagocytosis of trichomonads by leukocytes appears to be sufficient to arrest the disease in primary infections that involve avirulent or mildly virulent strains, but this is not true for infections with highly virulent strains. The exact role of phagocytosis in immune birds is not known (Kocan and Herman 1971). Humoral antibodies may be more important and have been shown to provide protection. This protection can be passively transferred from immune to nonimmune hosts via plasma or serum (Kocan 1970; Kocan and Herman 1970). The exact mechanism is not clear, but Kocan and Herman (1971) suggested that the trichomonads might be inhibited from penetrating the epithelium of the upper digestive tract or that they might be lysed after penetration. Goudswaard et al. (1979) demonstrated that IgA is found in pigeon milk and is also transferred into the bloodstream of squabs, probably by pinocytosis. Secretory IgA may play a role in transfer of immunity to *T. gallinae*, but this has not been investigated.

Experimental studies using mice have resulted in additional evidence that protective immunity occurs in infections with *T. gallinae*. Warren et al. (1961) used a mouse model that included subcutaneous injections of antigens from *T. gallinae* and found that complete protection against infection was observed in 50% of the animals tested. Honigberg (1978) was unable to

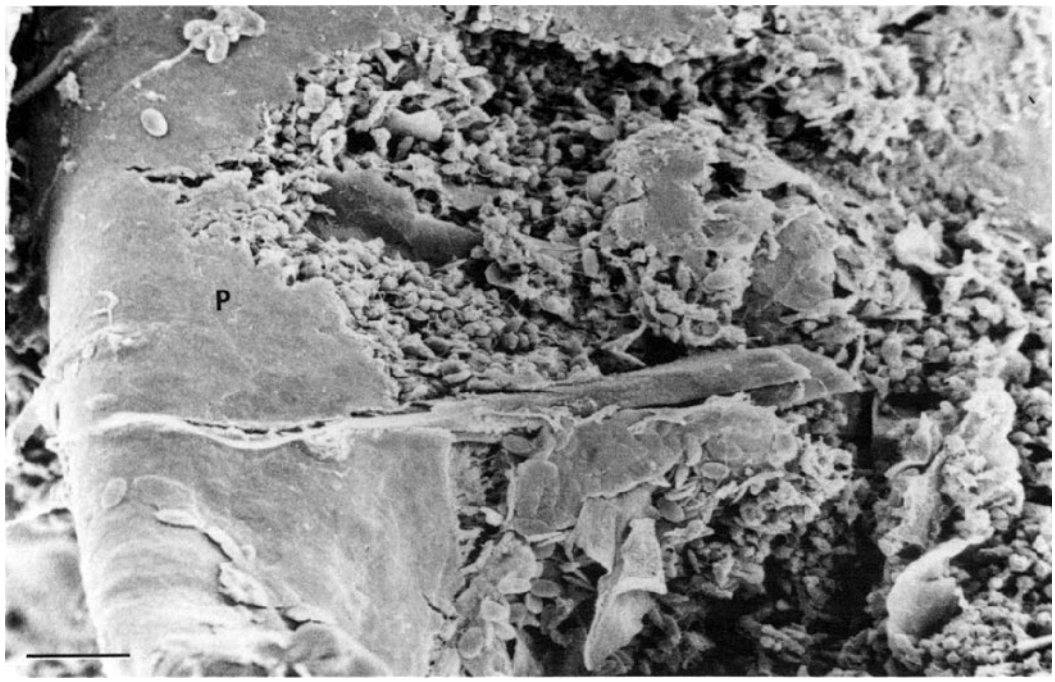


Figure 6.7. Scanning electron photomicrograph of the palatal–esophageal junction of a Ring Turtle-Dove (*Streptopelia risoria*) 216 h after being infected experimentally with a pathogenic strain of *Trichomonas gallinae*. Note erosion of palatal papilla (P) where several layers of epithelium have been removed. Scale bar = 50 μm. Reproduced from Kietzmann (1993), with permission of *The Journal of Parasitology*.

confirm these findings, but did observe protection of mice via intraperitoneal injections of a living strain of *T. gallinae* of mild pathogenicity.

There is some evidence that premunition may be important (Jaquette 1948; Stabler 1954). Although some pigeons are positive for *T. gallinae* for up to 620 days after infection, others lose their infections with time. Stabler (1954) stated that immunity gradually diminishes after infections are lost, but gave no data to back up this assertion. This should be further investigated.

Little new information on immunity has been published since the review of BonDurant and Honigberg (1994). However, studies on immunology of related organisms such as *T. foetus* in cattle and *T. vaginalis* in humans are numerous and may provide some clues to immunologic aspects of trichomonosis and *T. gallinae* infections in birds. In the case of immunity to *T. foetus* infections, antibody on the mucosal surface is critical for protection and vaccination to stimulate production of IgA and IgG1 pathogen-specific antibodies has proven successful (Corbeil et al. 2003). There is no clear proof of the protective characteristics of antibodies in immunity to *T. vaginalis* infections and it has been concluded that in addition to antibody, innate immune and acquired cellular responses are likely as important (Schwebke and Burgess 2004). Similar patterns may hold true for avian trichomonosis and should be investigated in order to understand more about the host response to *T. gallinae*.

PUBLIC HEALTH CONCERNS

There are no reports of infections of *T. gallinae* in humans (Cole 1999).

DOMESTIC ANIMAL HEALTH CONCERNS

There are reports of trichomonosis and infections with *T. gallinae* in domestic poultry. However, such infections are seen only occasionally in turkeys, and are even more rare in chickens (Willoughby et al. 1995). Feral Rock Pigeons are the source of infection in domestic pigeons and poultry (Kocan and Herman 1971), although other free-ranging doves might also be involved. There are also a number of records of trichomonosis in captive psittaciforms kept as pets (Garner and Sturtevant

1992; Murphy 1992; Rosskopf and Woerpel 1996). These infections may have originated from Rock Pigeons or other columbiforms as well.

WILDLIFE POPULATION IMPACTS

Trichomonosis has a negative effect on its avian host, but as with most wildlife diseases, the impact at the population level is difficult to measure. Although trichomonosis has caused mortality in many different species of free-ranging columbiforms and raptors in various parts of the world (Tables 6.1 and 6.2), these have usually involved small or moderate numbers of birds. The most significant recorded outbreaks have occurred in Mourning Doves in the US, although many of these were local and involved only 100–800 birds. Such outbreaks, for example, have been reported in California (Stabler and Herman 1951; Cole 1999), Nebraska (Greiner and Baxter 1974), New Mexico (Cole 1999), North Carolina (Cole 1999), and South Carolina (Kocan and Amend 1972).

The largest epizootic on record occurred in the southeastern US over a 2-year period (1950–1951) when an estimated 50,000–100,000 Mourning Doves died (Haugen 1952; Haugen and Keeler 1952). The die-off was centered in Alabama, but other neighboring states were also involved. Within Alabama, mortality was widespread, with losses being reported in 43 of the state's 67 counties. The overall negative impact of this die-off on dove populations was reflected by poor hunting success during the 2 years following the outbreak (Haugen 1952). Another major epizootic occurred in California during 1988 in which at least 16,000 Band-tailed Pigeons (*Patagioenas fasciata*) died (Cole 1999). During the spring and summer of 2001 an epizootic involving at least 1,000 Eurasian Collared-Doves (*Streptopelia decaocto*) occurred in Florida (Forrester and Spalding 2003). Trichomonad lesions were primarily in the liver, although there were a few birds with cankers in the oral cavity, throat, and crop. This outbreak was complicated by the presence of concurrent infections with pigeon paramyxovirus, which made interpretation of the cause of the mortality difficult. In 2001 and 2002, large numbers of Common Wood-Pigeons died of trichomonosis in southwestern Spain (Höfle et al. 2004) and southern England (Duff 2002). The number of birds dying in Spain was estimated at 2,600, which represented about 15% of the wintering population. Even so, the disease did not appear to have a serious effect on the population as judged by the numbers of birds counted in the following winter (Höfle et al. 2004). The impact on the Common Wood-Pigeon population in England is not known.

Several authors have suggested that trichomonosis may have played an important role in the final extinction of the Passenger Pigeon (*Ectopistes migratorius*) (Stabler 1954; Hanson 1969). There are no definitive data to support this idea, but it has been noted that Rock Pigeons were introduced into North America by the earliest colonists and may have been infected with virulent strains of *T. gallinae*. These Rock Pigeons may have been the source of infections in native species of columbiforms including the Passenger Pigeon (Haugen 1952).

The role of trichomonosis in the downward trend in populations of Mourning Doves in Utah was investigated (Ostrand et al. 1995). In a 2-year study, the prevalence of *T. gallinae* was 17%, but only 1 of 230 doves examined had lesions. Because of the low prevalence of birds with lesions, it was concluded that trichomonosis was not a factor contributing to the decline in Mourning Doves. However, the use of data on the prevalence of lesions might lead to an underestimation of the impact of trichomonosis since many, if not most, infected birds might die and thereby would not be included in the analysis.

In a recovery program in Mauritius for the endangered endemic Pink Pigeon, survival of squabs to 30 days of age increased from 27 to 62% when the birds were treated with carnidazole (Swinnerton et al. 2005). However, treatment did not significantly increase juvenile (postfledging) survival to 150 days. In the same program, a negative effect of infection with *T . gallinae* on adult survival, reproductive success, and fledgling survival of Pink Pigeons was documented (Bunbury 2006; Bunbury et al. 2008). Birds that were not infected with *T. gallinae* had a significantly higher probability of surviving for 2 years after examination than those that were (Figure 6.8).

Common Wood-Pigeons infected with a nonpathogenic strain of *T. gallinae* were lesion-free, but had lower body masses and fat reserves (Villanua et al. 2006). It was concluded that, although not fatal in and of themselves, these effects could lead to increased susceptibility of these birds to predation or other diseases and thereby exert a negative impact on the population. Additionally, the authors stated that this increased susceptibility of infected birds to predation would put birds of prey at a higher risk of exposure to *T. gallinae* after ingestion of these infected pigeons.

The effect of trichomonosis on populations of Peregrine Falcons (*Falco peregrinus*), which commonly feed on columbiforms, has been addressed (Stabler 1969). It was concluded that even though there is evidence that some Peregrine Falcons and other columbiform-eating raptors are infected with *T. gallinae* and contract the disease, the population impact was negligible.

Trichomonosis was diagnosed as the cause of death in 14 nestling Northern Goshawks (*Accipiter gentilis*)

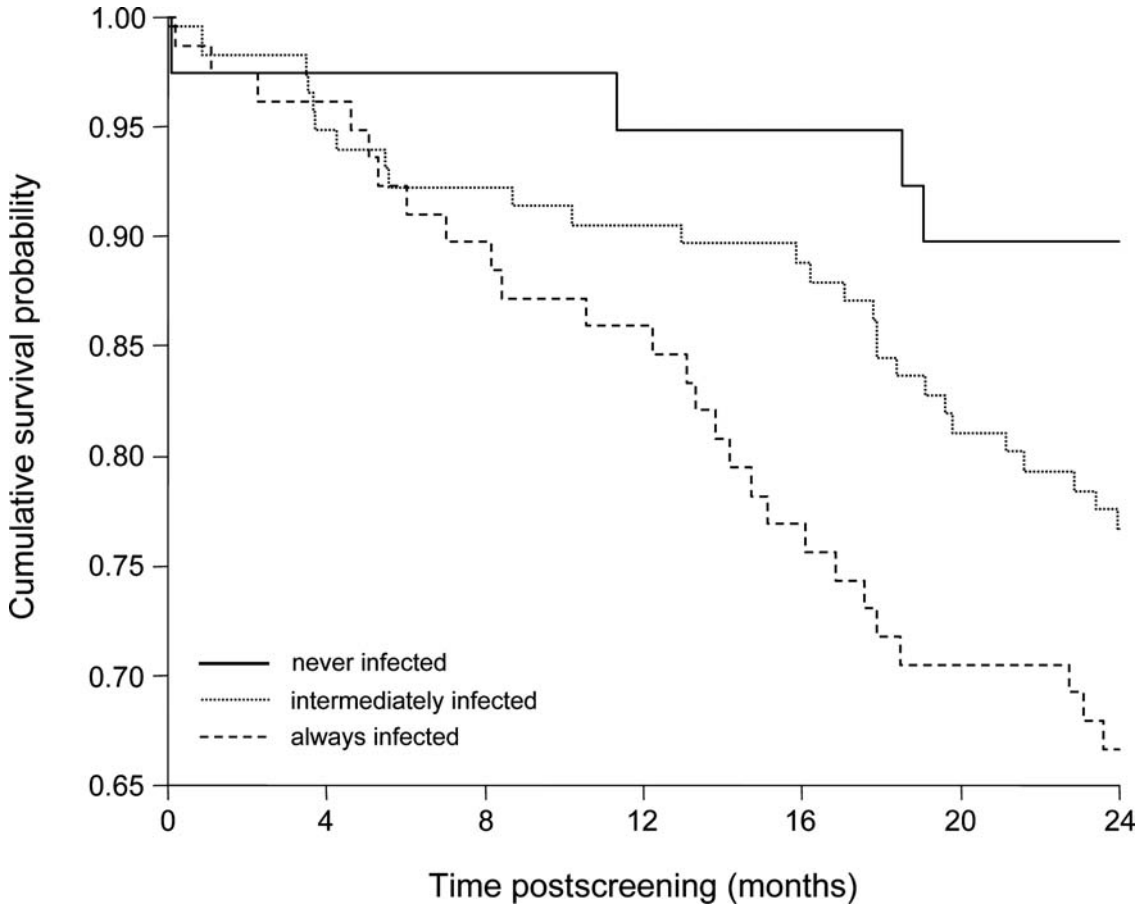


Figure 6.8. Kaplan–Meier survivorship curves for Pink Pigeons (*Nesoenas mayeri*) in Mauritius that were tested for *Trichomonas gallinae* in three consecutive 2-month periods ($n = 233$). Pigeons were either not infected, intermediately infected, or always infected. Reproduced from Bunbury et al. (2008), with permission of *Biological Conservation*.

during a study conducted in Scotland over a 13-year period (Cooper and Petty 1988). It was concluded that the disease slowed the expansion of the reintroduced population of Northern Goshawks by about 15%. During the course of a comparative study of the breeding ecology of Cooper's Hawks (*Accipiter cooperii*) in urban and exurban (undeveloped, natural) areas in southeastern Arizona, no nestling mortality of exurban birds due to trichomonosis was found (Boal and Mannan 1999). By contrast, 80% of the 73 nestlings found dead in the urban study area died of the disease. The food habits of Cooper's Hawks in the two areas were very different. Only a small proportion of the diet of exurban Cooper's Hawks consisted of columbiforms, whereas in the urban area 84% of the diet was Mourning Doves and Inca Doves (*Columbina inca*), which were known to have prevalences of *T. gallinae* infections of 16 and 52%, respectively.

TREATMENT AND CONTROL

Treating free-ranging wild birds is problematic. While mortality during an outbreak of trichomonosis in Common Wood-Pigeons in Spain ceased after dimetridazole was used to treat grain at game bird feeders, harmful effects were documented in nontarget species (Höfle et al. 2004). Dimetridazole can be toxic to birds (Reece et al. 1985), and in the area where the 2001 outbreak occurred, reductions in numbers of chicks and lower than normal populations of adult Red-legged Partridges (*Alectoris rufa*) were noted in the following autumn. By contrast, carnidazole, ronidazole, and

dimetridozole have been used with limited success to treat trichomonosis in one subpopulation of Pink Pigeons on Mauritius, successfully increasing survival rates of squabs, juveniles, and adults (Swinnerton et al. 2005).

Drugs that have been used to successfully treat infections in captive pigeons, raptors, and psittaciforms include some of the nitroimidazoles such as metronidazole, dimetridazole, ronidazol, and carnidazole (Emanuelson 1983; Ramsay et al. 1990; Pokras et al. 1993). Dimetridazole has been used successfully in drinking water to treat captive Rock Pigeons (Inghelbrecht et al. 1996), while metronidazole and carnidazole have been used effectively in raptors (Redig 2003). However, therapeutic failures due to drug resistance to several nitroimidazoles have been documented (Franssen and Lumeij 1992; Munoz et al. 1998). Currently, dimetridazole and metronidazole are not approved for use in birds in the US (Janzen 2006). Several synthetic compounds (chalcones) show evidence of potent activity against *T. gallinae* along with low toxicity to the host (Oyedapo et al. 2004) and may be good alternatives to nitroimidazoles when drug resistance is a problem.

Appropriate measures to control trichomonosis among wild and captive columbiforms and other birds should include actions to reduce sources of infection (Swinnerton et al. 2005). These include measures to minimize the use of contaminated communal food and water sources and measures to reduce stress from factors such as other pathogens and food shortages which can lead to reduction in resistance to trichomonosis. Backyard bird feeders and artificial watering areas should be kept clean. Food should be changed regularly and the feeders and other types of food platforms should be disinfected with a 10% bleach solution. To prevent disease transmission, attention should also be given to prevention of flocks of doves and pigeons coming to feed at grain storage facilities and feedlots for livestock (Cole 1999). The control of trichomonosis in wild raptors is very difficult, if not impossible, but in captive birds it can be prevented by avoiding the use of infected columbiforms as food sources (Halliwell 1979).

When outbreaks of trichomonosis occur in birds other than Rock Pigeons, the Rock Pigeons in the immediate area should be checked to determine if they contain lethal strains of *T. gallinae*. Until an assay is developed that can distinguish pathogenic from nonpathogenic stains, this must be done by culturing the parasite and then conducting transmission tests using Rock Pigeon squabs that have never been exposed to *T. gallinae* (Conti et al. 1985). Flocks of wild Rock Pigeons that are identified as being infected with lethal strains can be captured and treated or humanely eliminated. This approach may not be acceptable in many areas or countries, but might be worthy of consideration under appropriate circumstances.

MANAGEMENT IMPLICATIONS

Since there are pathogenic and nonpathogenic strains of *T. gallinae*, management of trichomonosis requires knowledge about the distribution of lethal strains before actions to minimize or even eliminate their impact on wild and captive populations of columbiforms, falconiforms, strigiforms, domestic poultry, and other susceptible birds of concern can be taken. This can be accomplished by conducting surveillance of free-ranging doves and pigeons, especially Rock Pigeons that are the primary source of lethal strains (Stabler 1954; BonDurant and Honigberg 1994). This could be followed by treatment or eradication of infected birds.

The translocation of nonendemic columbiforms into new areas should be avoided or done with great caution to eliminate the possibility of introducing pathogenic strains of *T. gallinae* into new areas. This is particularly true for islands where endemic species may be highly susceptible (see review by Wikelski et al. 2004). For example, pathogenic strains of *T. gallinae* are believed to have been introduced to Mauritius after release of Rock Pigeons and several species of exotic doves. This disease is now a serious threat to the survival of the endemic Pink Pigeon (Swinnerton et al. 2005; Bunbury 2006).

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7
Histomonas

William R. Davidson

INTRODUCTION

Histomoniasis is a disease of galliform birds (order Galliformes) caused by the protozoan *Histomonas meleagridis*. For many years following its recognition in the late 1800s, this disease was a major problem in the production of domestic poultry, especially turkeys. Histomoniasis is also recognized as a severe disease of Wild Turkeys (*Meleagris gallopavo*) and has been reported on occasion among other species of wild galliform birds.

SYNONYMS

Blackhead disease, infectious enterohepatitis, typhlohepatitis.

HISTORY

Excellent historical reviews of histomoniasis, including descriptions of earlier controversies regarding its etiology and epizootiology, have been published by Reid (1967) and Lund (1977). On the basis of a series of experimental infections in various species of galliform birds, Lund and Chute (1974) theorized that *H. meleagridis* evolved in Asia, probably as a parasite of Ring-necked Pheasants (*Phasianus colchicus*) or related *Phasianus* spp. Recognition that domestic chickens were also reservoir hosts for *H. meleagridis* led to the poultry industry axiom of not raising chickens and turkeys together (Reid 1967).

DISTRIBUTION

Histomoniasis has been reported throughout the world in regions where chickens, turkeys, or other domesticated galliform birds are raised. The disease is more prevalent in warmer regions of the globe, but has occurred with some frequency near the limits of both northern and southern temperate zones (Lund 1972). Prior to implementation of effective prevention and control practices, the frequency of occurrence of the disease in domestic poultry was correlated with areas with climate and soils suitable for transmission of the parasite. Most reports of histomoniasis among wild birds have been from North America, but whether this reflects true prevalence of the disease is unclear.

HOST RANGE

Histomoniasis is a disease almost exclusively of birds in the order Galliformes. At least 12 species of nondomestic galliform birds are susceptible to infection (Table 7.1), although there is wide variation in susceptibility and clinical response to infection among species. Among wild populations, histomoniasis has been reported most frequently as a disease of Wild Turkeys and much less often in other species. However, histomoniasis is common in several species of galliform game birds raised in captivity. Reports in nongalliform birds, such as captive Ostriches (*Struthio camelus*) (Borst and Lambers 1985), are rare. Mallards (*Anas platyrhynchos*) and domestic geese were essentially refractory to experimental infection (Lund et al. 1974).

ETIOLOGY

Histomonas meleagridis is the only member within the genus and is a pleomorphic flagellate in the family Monocercomonadidae, order Trichomonadida, phylum Parabasalia (Brugerolle and Lee 2000). Histomonads are 4–30 μm in diameter, rounded to elongate, have a single nucleus, exhibit active ameboid movement, and may have a single flagellum. The morphologic form is dependent on the stage of the infection and location of the parasite within the avian host. Both ameboid and flagellate trophozoites exist in the cecal lumen of infected birds. Organisms within lesions in the cecal wall or in the liver lack a flagellum. Reproduction is by binary fission. There is no cyst or environmentally resistant stage, and trophozoites shed in feces do not survive outside the avian host.

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Table 7.1. Species of the order Galliformes reported to be susceptible to infection by *Histomonas meleagridis.*

Species	Host status	Disease severity	Status as reservoir	Reference
Wild Turkey (*Meleagris gallopavo*)	Wild, captive, experimental	Severe	Poor	Stoddard (1935, 1936), Mosby and Handley (1943), Kozicky (1948), Snyder (1953), Roberts (1956), Thomas (1964), Bailey and Rinell (1968), Prestwood et al. (1973), Lund et al. (1975), Hurst (1980), Davidson et al. (1985), Schorr et al. (1988), Ley et al. (1989), Davidson and Wentworth (1992), and Forrester (1992)
Northern Bobwhite (*Colinus virginianus*)	Wild, captive, experimental	Moderate	Marginal	Kellogg and Reid (1970), Lund and Chute (1971b), Davidson et al. (1978), Zeakes et al. (1981), and Davidson et al. (1982)
Ruffed Grouse (*Bonasa umbellus*)	Captive	Severe	Poor	Bump et al. (1947)
Greater Prairie-Chicken (*Tympanuchus cupido*)	NR	NR	NR	Braun and Willers (1967)
Ring-necked Pheasant (*Phasianus colchicus*)	Captive, experimental	Mild	Superior reservoir	O'Roke (1933), and Lund and Chute (1972b–d, 1974)
Chukar (*Alectoris chukar*)	Captive, experimental	Severe	Poor	Chaddock (1948), Sims (1960), and Lund and Chute (1971a, 1972b, 1974)
Indian Peafowl (*Pavo cristatus*)	Captive, experimental	Severe	Poor	Lund and Chute (1972b, c, 1974)
Gray Partridge (*Perdix perdix*)	Experimental	Mild	Poor	Lund and Chute (1972b, 1974)
Black Francolin (*Francolinus francolinus*)	Wild	NR	NR	Bump and Bump (1964)
Red Junglefowl (*Gallus gallus*)	Wild (released), Captive	Mild	Important	Kellogg et al. (1971, 1978)
Japanese Quail (*Coturnix japonica*)	Experimental	Mild	Poor	Lund and Ellis (1967), and Lund and Chute (1972b, 1974)
Helmeted Guineafowl (*Numida meleagris*)	Experimental	Mild to severe	Suitable	Chute and Lund (1972, 1974), and Lund and Chute (1972e)

NR, not reported.

EPIZOOTIOLOGY

The epizootiology of *H. meleagridis* is unusual in that under natural conditions transmission is dependent on the cecal nematode *Heterakis gallinarum*, which also infects many species of galliform birds. This discovery by Graybill and Smith (1920) is considered a milestone in parasitology (Lund 1977). Histomonads, in addition to infecting the ceca of the bird, also infect the cecal worms. Within the ovaries of female cecal worms, the histomonads become incorporated in the eggs of *H. gallinarum* (Lee 1969; Lund and Chute 1973). The protective covering of the cecal worm egg shields the delicate histomonads from deleterious environmental factors which otherwise rapidly kill the protozoans. When infective (larvated) histomonad-bearing heterakid eggs are ingested by a suitable host, both parasites are released in the ceca. Although some histomonads are released when the heterakid eggs hatch, the majority is liberated from cecal worm larvae that die and decompose within the ceca (Lund and Chute 1972a, 1974). This is important because infection of *H. gallinarum* by *H. meleagridis* occurs principally when the heterakid larvae are 10–20 days old, and histomonads from simultaneously acquired heterakid larvae that have died are most numerous in the cecal lumen during this time period (Lund 1968, 1971; Lund and Chute 1974).

In addition to direct transmission via heterakid eggs, earthworms serve as important paratenic hosts of histomonad-infected *H. gallinarum* larvae. Under field conditions, earthworms can accumulate and store large numbers of heterakid larvae in somatic tissues. Both parasites are transmitted together, especially following periods of rain, when earthworms come to the soil surface and are consumed easily by susceptible birds (Lund et al. 1966; Kemp and Franson 1975). Grasshoppers can also serve as paratenic hosts for *Heterakis* and *Histomonas* (DeVolt and Davis 1936; Frank 1953) but their role is less important than earthworms (Reid 1967).

It was recently demonstrated that histomonads can be effectively transmitted laterally by the fecal–oral route (Hu and McDougald 2003). However, this means of transmission appears to be restricted only to the crowded conditions of confinement-reared domestic poultry production and is not important among wild bird populations.

Different members of the order Galliformes exhibit wide variation in their susceptibility to clinical histomoniasis, spanning the spectrum from an essential tolerance of the protozoan with minimal lesions to severe disease with a high case-fatality rate (Lund and Chute 1972b). Species such as Ring-necked Pheasants, domestic chickens, or junglefowl that develop minimal disease harbor and readily transmit both *H. meleagridis* and *H. gallinarum*. Such species function as critical reservoir hosts for both parasites. Apart from the potential reduction in transmission caused by host deaths from histomoniasis, the clinical response to *H. meleagridis* infection has a major influence on transmission of both parasites. In ceca with lesions caused by *Histomonas*, the survival of the heterakid nematode is extremely poor, and heterakid infrapopulations in diseased hosts are often completely eliminated by altered conditions within the ceca. Thus, individual birds or species in which severe cecal lesions develop are poor reservoir hosts (Lund and Chute 1972b, 1974).

Lund and Chute (1974) presented a well-supported hypothesis that both *H. meleagridis* and *H. gallinarum* evolved in Asia with the Ring-necked Pheasant or a close relative of the pheasant. This concept was based on experimental studies in various galliform species in which the number of histomonad-infected heterakid eggs produced per heterakid egg ingested were measured. There were great differences in the egg output/input ratios among the host species, ranging from less than 1 to 1 for Japanese Quail (*Coturnix japonica*), Gray Partridge (*Perdix perdix*), Peafowl, and Northern Bobwhites (*Colinus virginianus*) to greater than 5 to 1 for pheasants, chickens, and guineafowl (Lund and Chute 1974). These experiments delineated reservoir hosts, in which disease is rare or mild, from vulnerable hosts, in which disease is more severe. Junglefowl also are efficient reservoir hosts (Kellogg et al. 1978).

Worldwide, domestic chickens currently are the most widely distributed and abundant reservoir host for *H. meleagridis*; however, in areas where they occur, wild populations of pheasants and junglefowl can serve as important reservoirs. In addition, *H. meleagridis* and *H. gallinarum* are both common in captive-reared game birds, and if they are released in the wild, these birds can serve as sources of infection for vulnerable species. Captive-reared game birds can also differ from their wild counterparts in the risk they pose for transmitting *H. meleagridis*. For example, captive-reared Northern Bobwhites are often infected with *H. gallinarum*, whereas wild Northern Bobwhites rarely harbor *H. gallinarum* but are commonly infected with another cecal worm, *Heterakis isolonche* (or *Heterakis bonasae*). This distinction is important because *Heterakis isolonche* apparently does not transmit *H. meleagridis*, and thus, captive-reared and wild bobwhites differ in their epizootiologic risks (Davidson et al. 1978).

CLINICAL SIGNS

Clinical signs of histomoniasis are well described for domestic poultry, especially turkeys, and typically appear from 1 to 3 weeks following infection. Alterations

of serum enzymes, serum proteins, and histochemical changes in the ceca and liver have been described in infected domestic poultry (Clarkson 1966; McDougald and Hansen 1970; Wilkins and Lee 1976). Affected birds are inactive, depressed, develop inappetence, and stand with drooped wings, closed eyes, retracted head, and ruffled feathers. Feces may be sulfur colored or contain flecks of blood and mucus. Birds that survive several weeks often become emaciated. Similar signs have been reported in wild and captive galliform game birds; however, because of the difficulties of monitoring wild populations, most affected individuals are reported only because they are so ill that they are easily captured or are found dead.

PATHOGENESIS AND PATHOLOGY

Histomonas meleagridis organisms are liberated from *H. gallinarum* larvae in the ceca where they begin to reproduce within the lumen. Within 4–7 days, organisms can be detected within small ulcerations in the cecal mucosa where they elicit a mixed inflammatory response that is predominantly composed of heterophils. Necrosis ensues and focal mucosal and submucosal lesions expand to become confluent, eventually extending into the muscularis layer. The lumen of the ceca fills with a mixture of cecal contents, serous and hemorrhagic exudates, inflammatory cells, and necrotic debris. The concentric layers of this admixture typically form a caseous cecal core. Histomonads continue to reproduce within lesions in the cecal wall and may gain entry into the venous blood vessels. They then are carried to the liver where they continue to reproduce, setting up discrete foci of hepatic necrosis that are grossly visible at about 10 days. Initially, foci of hepatic necrosis appear as small white spots that progress to gray depressed areas surrounded by a narrow rim of hemorrhage. As they enlarge, these lesions may coalesce and become more firm and white or yellow as fibrosis occurs. In severely affected birds, death often occurs between 14 and 21 days (Clarkson 1962).

Although *H. meleagridis* is the critical etiologic agent of histomoniasis, experiments with gnotobiotic turkeys have demonstrated clearly that intestinal bacteria are essential for development of lesions. During the 1960s, separate laboratories independently confirmed that intestinal bacteria play two essential roles in the pathogenesis of histomoniasis (Doll and Franker 1963; Franker and Doll 1964; Bradley and Reid 1966). One role is enabling *H. meleagridis* to colonize the ceca of the bird; bacteria-free domestic turkey poults rarely could be infected with *H. meleagridis* and did not develop histomoniasis. The second role is enhancing the development of lesions. In marked contrast to bacteria-free poults, both conventional turkey poults with normal intestinal bacterial flora and gnotobiotic poults with monospecific intestinal bacterial flora consisting of *Escherichia coli*, *Escherichia intermedia*, *Clostridium perfringens*, *Streptococcus fecalis*, or *Bacillus subtilis* developed mild to severe disease. Monospecific infection with certain other bacteria allowed colonization of ceca by *H. meleagridis* but did not result in lesions (Reid 1967).

Other host compromising factors may also influence outcome of infection. For example, exposure to the insecticide Sevin (1-napthyl *N*-methyl carbamate) increased the susceptibility of bobwhites to histomoniasis and markedly increased mortality (Zeakes et al. 1981).

DIAGNOSIS

The combination of necrotic cecal cores and multifocal hepatic necrosis in a galliform bird (Figure 7.1) is strong presumptive evidence of histomoniasis. Confirmation of infection in diseased hosts can be accomplished by histologic demonstration of histomonads (Figure 7.2) in ceca or liver using various stains such as periodic acid Schiff or silver stains (Kemp and Reid 1966), demonstration of live histomonads in saline mount preparations (75–80°F slide warming device required), in vitro cultivation of histomonads from ceca

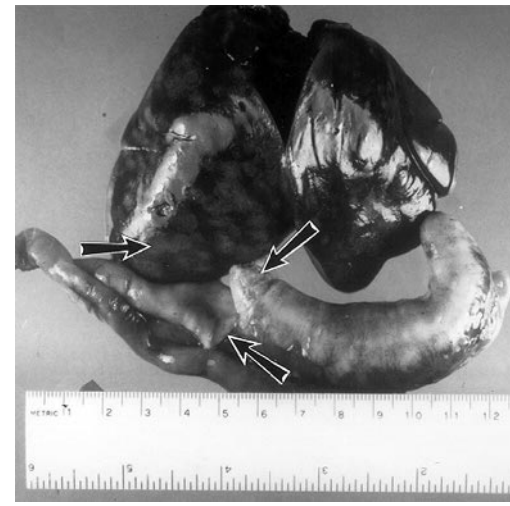


Figure 7.1. Gross lesions of *Histomonas meleagridis*-infected liver (upper) and ceca (lower) from an experimentally infected turkey poult illustrating (arrows) multifocal hepatic necrosis, thickened and hemorrhagic cecal walls, and caseous cecal core. Reprinted with permission from Waters (1992).

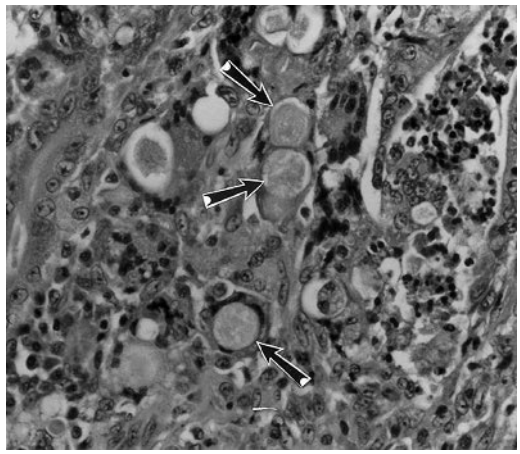


Figure 7.2. Photomicrograph of submucosal region of cecum demonstrating *Histomonas meleagridis* (arrows) within lesions. Hematoxylin and eosin stain (891×). Reprinted with permission from Waters (1992).

or liver (McDougald and Galloway 1973), or by rectal inoculation of turkey poults with saline suspensions of cecal or liver lesions. Confirmation of infection in unaffected reservoir hosts is best accomplished by in vitro cultivation of cecal contents, by rectal inoculation of turkey poults with cecal suspensions, or by feeding embryonated heterakid eggs from the suspected host to turkey poults. Fresh specimens are required for in vitro cultivation, direct visualization in wet mounts, and bioassay procedures using turkey poults. Freezing may impair confirmation by histologic means. Differential diagnoses should include coccidiosis, coligranuloma, salmonellosis, and neoplasia.

IMMUNITY

Several species of galliform birds exhibit an age resistance to infection by *H. meleagridis*, with younger hosts being more susceptible to infection and developing more severe disease (Lund and Chute 1970; Lund 1972; Levine 1985). However, virulent strains of *H. meleagridis* can cause disease in hosts of any age. Birds that recover from histomoniasis develop immunity to reinfection (Lund 1972; Levine 1985).

DOMESTIC ANIMAL AND PUBLIC HEALTH CONCERNS

Historically, histomoniasis was a major disease among domestic turkeys; however, recognition of the reservoir role played by domestic chickens, the separation of commercial chicken and turkey flocks, and improved husbandry practices in range turkeys have greatly reduced losses due to histomoniasis. Appropriate husbandry and biosecurity practices are effective in preventing transmission of histomoniasis from wild or captive game birds and noncommercial chickens to commercial turkey flocks. Histomoniasis has no known public health implications.

WILDLIFE POPULATION IMPACTS

Although histomoniasis is frequently mentioned in scientific, semitechnical, and popular literature as a significant disease risk for wild galliform birds, especially Wild Turkeys, there are relatively few primary accounts of the disease in wild populations (Davidson and Wentworth 1992). Despite the few reports from Wild Turkeys, histomoniasis is believed to be one of the more important diseases of this species in the southeastern US (Hurst 1980; Davidson et al. 1985). Histomoniasis was the second most common infectious disease among sick or dead Wild Turkeys from eight southeastern states diagnosed and accounted for 14% of nontrauma diagnoses (Davidson et al. 1985). In sick or dead Wild Turkeys from Florida, histomoniasis was less frequent but accounted for 5% of nontrauma diagnoses (Forrester 1992). Histomoniasis appears to be rare among populations of other wild galliform birds, although information for many species is sparse.

PREVENTION, CONTROL, AND MANAGEMENT IMPLICATIONS

Prevention and control of histomoniasis is predicated on separating reservoir hosts from vulnerable species and on breaking the cycle of the cecal worm vector. These objectives can be accomplished among captive flocks by not commingling reservoir and vulnerable species (e.g., chickens and turkeys), by housing flocks in deep stone or wire floored pens that reduce ingestion of eggs and earthworm paratenic hosts, or by removal of *H. gallinarum* with appropriate anthelmintics. Preventive actions applicable for wild galliform populations that achieve similar objectives include not introducing reservoir hosts (e.g., Ring-necked Pheasants or junglefowl) in habitats occupied by wild vulnerable species (e.g., Wild Turkey) and not using untreated manure from domestic chickens as fertilizer on areas frequented by vulnerable species. Although manure from commercial broiler chickens grown under modern husbandry practices poses little risk of histomoniasis, commercial breeder and layer flocks and noncommercial chickens still have high prevalences of infection (Waters et al. 1994). Risk of introducing

histomoniasis from junglefowl into native wild turkey populations was one factor in the abandonment of an earlier foreign game bird introduction program in the US (Kellogg et al. 1978).

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8
Eimeria

Michael J. Yabsley

INTRODUCTION

The coccidia infect all classes of vertebrates and are a large and complex group of obligate intracellular parasites in the phylum Apicomplexa. Many of the coccidia are important medical and veterinary pathogens, but in this chapter, only the species of *Eimeria* that infect the intestines, kidneys, and liver of wild birds are discussed.

Classification of the coccidia is based on morphologic characteristics, especially those of the environmentally-resistant sporulated oocyst. This stage is the only one that is passed in feces of the host and is therefore the stage most often observed. The vast majority of coccidia are described solely on the morphology of voided oocysts, and in many cases nothing more about their development in the host is known.

The majority of avian species of *Eimeria* infect and develop within intestinal epithelial cells; however, some species of *Eimeria* develop in extraintestinal locations. Asexual stages, sexual stages, and the oocysts all develop within the cytoplasm or nucleus of infected cells. Compared with intestinal species of *Eimeria*, little is known regarding the host specificity and endogenous development of renal coccidia due to the difficulty in getting oocysts to sporulate to the infective stage. To date, virtually all extraintestinal species of *Eimeria* have been detected within infected renal epithelial cells. Exceptions are two species of *Eimeria—Eimeria gruis* and *Eimeria reichenowi*—that infect multiple organs in cranes (Chapter 9) and a single case of hepatic coccidiosis in a Magpie-lark (*Grallina cyanoleuca*).

An important consideration when discussing coccidia is to differentiate infection and disease. Coccidian infections are frequently asymptomatic; thus, the correct terminology for infection is "infection with" or "coccidiasis." Coccidiosis refers to infections resulting in clinical disease, but the term is often used inappropriately to indicate infection. Under certain varying circumstances, including age of host, high inoculation dose, stress, lack of previous infection, concurrent disease, or immunosuppression, species of *Eimeria* that normally do not cause disease can produce pathogenic effects and cause coccidiosis. In general, however, species of *Eimeria* rarely cause disease in free-ranging birds. Young birds or adults that are stressed or unhealthy are more likely to develop clinical coccidiosis.

HISTORY

Coccidiosis of domestic birds was recognized as early as the late 1800s (Railliet and Lucet 1890; Salmon 1899). *Coccidium truncatum* (=*Eimeria truncata*) from domestic geese was the first species of renal *Eimeria* to be named and described (Railliet and Lucet 1890). The life cycle of the first intestinal species of avian *Eimeria* was described in 1910 (Fantham 1910a). This species, detected in grouse in the UK, was named *Eimeria avium* and initially was thought to cause coccidiosis in numerous avian species (Fantham 1910b, 1911). It is now known that members of this genus are generally host specific and almost 200 species of avian *Eimeria* have been formally described. Numerous more have been reported but not described as distinct species.

INTESTINAL *EIMERIA*

DISTRIBUTION AND HOST RANGE

Intestinal species of avian *Eimeria* have been reported throughout the world. Intestinal coccidiosis is a common and economically important disease of domesticated fowl such as chickens, turkeys, and geese, whereas only sporadic cases of intestinal coccidiosis in wild birds have been reported.

Infection with intestinal coccidia is probably ubiquitous among avian species; however, prevalence of infection with *Eimeria* varies among the avian orders (Table 8.1). To date, approximately 196 species of *Eimeria* have been formally described from 17 avian orders. However, uncharacterized species of *Eimeria* have been reported from numerous other wild avian

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Table 8.1. The approximate number of species of *Eimeria* that have been reported from avian hosts.

Host order	Number of avian species	Number of avian species with *Eimeria* (%)	Number of *Eimeria* spp.	Undescribed Coccidia reported
Struthioniformes	1	0	0	Yes
Casuariiformes	7	0	0	Yes
Rheiformes	2	0	0	Yes
Tinamiformes	46	2 (4.3)	2	No
Sphenisciformes	17	0	0	Yes
Gaviiformes	5	1 (20)	1	No
Podicipediformes	19	0	0	No
Procellariiformes	107	2 (1.9)	2	Yes
Pelecaniformes	62	4 (6.5)	5	Yes
Ciconiiformes	124	4 (3.2)	4	Yes
Phoenicopteriformes	5	0	0	No
Anseriformes	157	36 (22.9)	30	Yes
Falconiformes	296	2 (0.7)	2	Yes
Galliformes	287	39 (13.6)	79	Yes
Gruiformes	183	10 (5.5)	13	Yes
Charadriiformes	360	18 (5)	19	Yes
Columbiformes	298	12 (4)	9	Yes
Psittaciformes	352	5 (1.4)	4	Yes
Cuculiformes	161	1 (0.6)	2	Yes
Strigiformes	194	9 (4.6)	8	Yes
Caprimulgiformes	115	0	0	Yes
Apodiformes	425	0	0	Yes
Coliiformes	6	0	0	No
Trogoniformes	39	0	0	Yes
Coraciiformes	208	3 (1.4)	4	No
Piciformes	396	5 (1.3)	5	Yes
Passeriformes	5,593	9 (0.16)	8	Yes
Total	9,465	162 (1.7)	196	Yes

Note: Host orders and species follow Dickinson (2003).

hosts. The majority of species of *Eimeria* found in free-ranging wild birds are reported from the orders Anseriformes and Galliformes. Likewise, coccidiosis is also reported most often from these two orders. Species of birds from some orders, such as Passeriformes, are primarily infected with *Isospora* and/or *Atoxoplasma* (Chapter 5). Although undescribed species of *Eimeria* have been detected in many orders of birds, there are many other orders where *Eimeria* likely occurs but has not been observed because few hosts have been examined. In other orders, for example Passeriformes, a considerable number of hosts have been examined for coccidia, but few *Eimeria* spp. have been detected.

In general, *Eimeria* are highly host specific, but some species of *Eimeria* do infect multiple, often closely related, hosts. For example, *Eimeria dispersa* infects turkeys, chickens, Chukar (*Alectoris chukar*), Ring-necked Pheasants (*Phasianus colchicus*), and Northern Bobwhite (*Colinus virginianus*) (Doran 1978), and *Eimeria mulardi* infects domestic ducks (*Anas platyrhynchos*), Muscovy Ducks (*Cairina moschata*), and their hybrid mule duck offspring (Sercy et al. 1996). Controlled experimental infections and/or genetic studies are needed to prove or disprove the occurrence of a single *Eimeria* species in multiple hosts, especially those hosts in distinct genera.

ETIOLOGY

Several genera of coccidia infect the intestinal epithelium of avian hosts, including *Eimeria*, *Isospora*, *Atoxoplasma*, *Tyzzeria*, *Caryospora*, *Cryptosporidum*, and *Sarcocystis*. The genus *Eimeria* is in the family Eimeriidae, order Eucoccidiorida, phylum

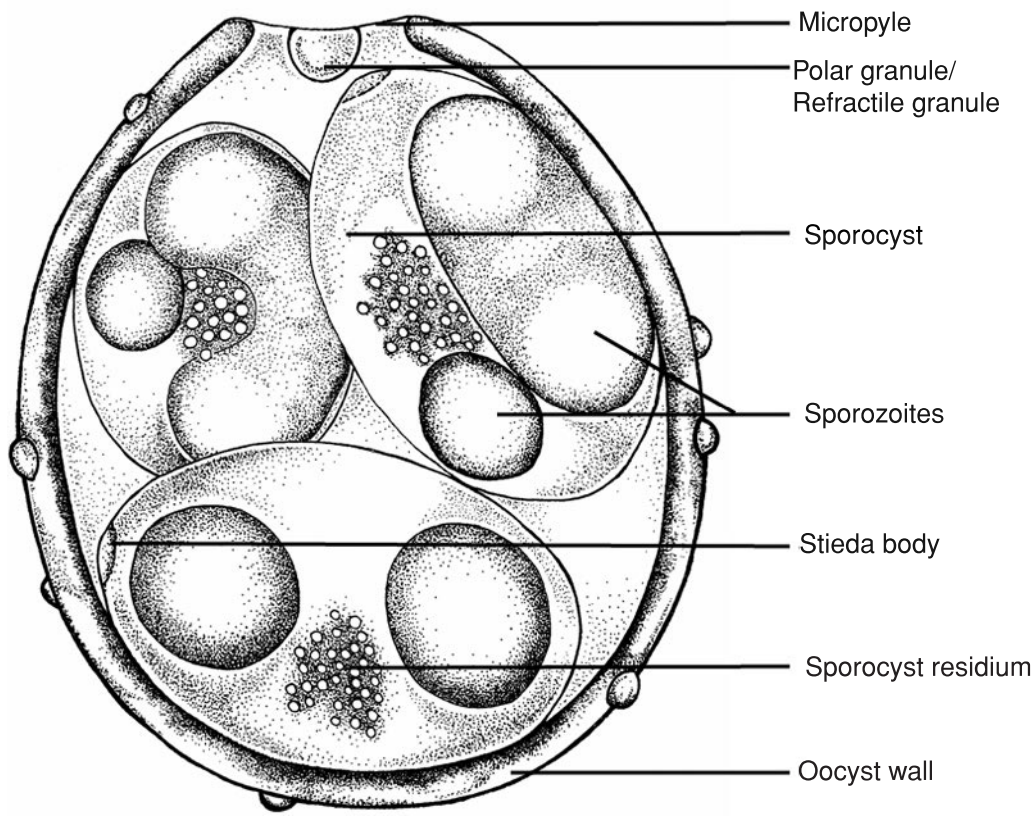


Figure 8.1. Major structural characteristics of the sporulated oocyst of a typical species of *Eimeria*. Drawing by S. E. J. Gibbs, CSIRO. Reproduced from Yabsley and Gibbs (2006), with permission of the *Journal of Parasitology*.

Apicomplexa and is one of the most common coccidia reported from birds. *Eimeria* can be differentiated from other genera by their characteristic oocysts—each contains four sporocysts, each of which contains two sporozoites (Figure 8.1).

Other genera of avian coccidia have oocysts that differ in morphology. Members of the genera *Isospora* and *Atoxoplasma* (Chapter 5) have two sporocysts, each with four sporozoites. Members of the genera *Cryptosporidium* (Chapter 10) and *Tyzzeria* do not have sporocysts and contain four and eight sporozoites, respectively, within the oocyst. Members of the genus *Caryospora* have oocysts that possess a single sporocyst with eight sporozoites. Oocysts of species of *Sarcocystis* are extremely thin and free sporocysts containing four sporozoites are often the only forms detected in feces (Chapter 5). Molecular characterization of avian coccidia has shown that many of these early classifications have created polyphyletic genera and that morphologic characters are not sufficient to determine relationships (e.g., avian *Isospora* are more closely related to *Eimeria* than to species of mammalian *Isospora*) (Carreno and Barta 1999; Yabsley and Gibbs 2006).

EPIZOOTIOLOGY

Coccidia in the genus *Eimeria* have direct life cycles (i.e., they are transmitted from one host to another without the aid of vectors or intermediate hosts). Development within the host includes both asexual and sexual stages that reside in epithelial cells. Unsporulated, noninfective oocysts passed in the feces of the host undergo sporulation in the environment to become infective. The first step of sporulation is the asexual process of sporogony by which sporocysts and sporozoites (infective stage) are produced from the germ ball within the environmentally resistant oocyst (Figure 8.1). This process is regulated by various environmental variables (oxygen, light, temperature, etc.)

that are parasite species dependent. In general, oocysts are extremely resistant and can even tolerate desiccation and freezing (Sathyanarayanan and Ortega 2006), although hard freezes or extreme heat can kill them (Parker and Jones 1990).

Once ingested by an appropriate host, various chemical and physical cues cause the oocysts to rupture, releasing sporocysts which then rupture and release sporozoites. The sporozoites invade intestinal epithelial cells and transform into trophozoites. Trophozoites replicate asexually to form meronts, which later transform into merozoites by a process called merogony. These merozoites break out of the cell and enter other epithelial cells either to undergo additional rounds of merogony or to begin gametogony. The number of cycles of merogony and number of merozoites produced during each cycle differs among species of *Eimeria*. During gametogony, merozoites transform into macrogametocytes (female cell) or microgametocytes (male cell) (Figure 8.2). A macrogametocyte develops into a single macrogamete while a microgametocyte buds to form many flagellated microgametes. These microgametes exit the host cell and enter cells containing macrogametes, where fertilization occurs. A fertilized macrogamete develops an outer wall to become an oocyst (Figure 8.2), which is passed in the feces of the host.

Coccidiosis is rare in free-ranging birds and is usually related to captive rearing, crowding, or stress. For several species of *Eimeria*, disease severity increases with ingestion of increasing numbers of oocysts in a dose-dependent fashion (Ruff and Wilkins 1987; Williams 2001). Young or naïve birds exposed to high numbers of infective oocysts are most likely to exhibit disease.

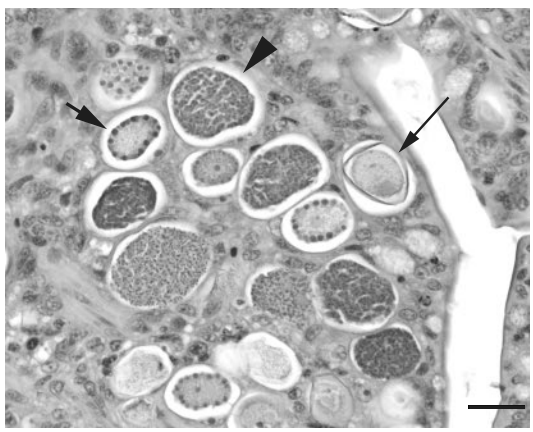


Figure 8.2. Developmental stages of *Eimeria*. Macrogametocytes (short arrow), microgametocytes (arrow head), and oocyst (long arrow) within villous epithelial cells. Flattened host cell nuclei are seen in some cells. Hematoxylin and eosin stain. Bar = 20 μm. Courtesy of A. E. Ellis, University of Georgia.

Anseriformes

Species of *Eimeria* have been reported from only 22% of avian species in the order Anseriformes, but this group accounts for the second highest number of species of *Eimeria* reported from birds (Table 8.1). Several of these species of *Eimeria* have been reported to infect multiple avian hosts, but this needs to be confirmed by both experimental studies and application of molecular methods to identify morphologically similar cryptic species. An excellent review of coccidia of Anseriformes, including full morphologic descriptions and life history information, has been published (Gajadhar et al. 1983b).

Several epizootics of intestinal coccidiosis caused by *Eimeria aythyae* have been reported from free-ranging Lesser Scaup (*Aythya affinis*) in the US (Table 8.2; Bump 1937; Farr 1965; Windingstad et al. 1980; Southeastern Cooperative Wildlife Disease Study, unpublished data; US Geological Survey, National Wildlife Health Center, unpublished data). All outbreaks occurred during the spring. In Nebraska, at least 29% of Lesser Scaup died in each of three consecutive outbreaks during the springs of 1976–1978 (Windingstad et al. 1980). These outbreaks were associated with low water levels that could have crowded and stressed the birds. Attempts to transmit the infection to the Tufted Duck (*Aythya fuligula*) failed. An outbreak of intestinal and renal coccidiosis in 1993 (caused by an undescribed *Eimeria* sp. and *Eimeria somateriae*, respectively) resulted in the death of hundreds of Common Eider ducklings (*Somateria mollissima*) in Iceland (Table 8.2; Skirnisson 1997). This outbreak could have been caused by the washing of large amounts of mud into the sea near the nesting sites, which led to decreased foraging success and undernourishment and stress of ducklings (Skirnisson 1997).

Galliformes

Coccidiosis is a worldwide economically important disease of domestically raised fowl, primarily chickens and turkeys. In general, species of Galliformes harbor multiple species of coccidia (e.g., at least eight in chickens and seven in turkeys). Because of the economic importance of coccidiosis to domestic chickens, many excellent reviews of domestic chicken coccidia have been published (Allen and Fetterer 2002; Shirley

Table 8.2. Common pathogenic intestinal species of *Eimeria*.

Host order	Host common name	Host scientific name	*Eimeria* spp.	Locality	Location of tissue stages	Clinical signs and/or lesions	Mortality	Citations
Anseriformes	Lesser Scaup	*Aythya affinis*	*Eimeria aythyae*	USA	Cytoplasm of small intestine epithelial cells	Sloughing of intestinal mucosa with extensive hemorrhage	Extensive outbreaks	Bump (1937), Farr (1965), Windingstad et al. (1980), and U.S. Geological Survey, National Wildlife Health Center and Southeastern Cooperative Wildlife Disease Study, unpublished data
	Common Eider	*Somateria mollissima*	*Eimeria* sp.	Iceland	Cytoplasm of small intestine epithelial cells	Focal necrosis of infected intestinal cells	Small localized outbreaks in malnourished ducklings; contributed to outbreak of renal coccidiosis	Skirnisson (1997)
	Common Goldeneye	*Bucephala clangula*	*Eimeria bucephalae*	Denmark	Cytoplasm of small intestine epithelial cells	Thickening of intestinal wall with hemorrhagic lesions and grayish-white foci; necrosis of subepithelial tissues	Outbreaks in young birds	Christiansen and Madsen (1948)
	Canada Goose	*Branta canadensis*	*Eimeria fulva*	USA	Cytoplasm of small intestine epithelial cells	Thickening of the intestinal wall and accumulation of greenish mucus in the intestinal lumen	Small localized outbreaks	Farr (1953)

(*continues*)

	Canada Goose and Lesser Snow Goose	*Branta canadensis* and *Chen caerulescens caerulescens*	*Eimeria stigmosa*	USA, Canada	Nucleus of ileum and colon epithelial cells	Reddening of mucosal surface and focal enteritis	Small localized outbreaks	Gajadhar et al. (1986)
Galliformes	Wild Turkey	*Meleagris gallopavo*	*Eimeria adenoeides*	USA	Cytoplasm of lower ileum, ceca, and rectum epithelial cells	Dilated intestine and thickened wall, thick creamy material, or caseous casts	Not associated with disease in wild birds	Blakey (1932), and Kozicky (1948)
	Wild Turkey and Northern Bobwhite	*Meleagris gallopavo* and *Colinus virginianus*	*Eimeria dispersa*	USA	Cytoplasm of small intestine and ceca epithelial cells	Creamy, mucoid enteritis	Not associated with disease in wild birds	Blakey (1932), and Kozicky (1948)
	Wild Turkey	*Meleagris gallopavo*	*Eimeria gallopavonis*	USA	Cytoplasm of lower ileum, ceca, and rectum epithelial cells	Dilated intestine and thickened wall, thick creamy material, or caseous casts	Not associated with disease in wild birds	Blakey (1932), and Kozicky (1948)
	Wild Turkey	*Meleagris gallopavo*	*Eimeria meleagrimitis*	USA	Cytoplasm of upper and mid-small intestine epithelial cells, lamina propria, or deeper tissues	Necrotic enteritis	Not associated with disease in wild birds	Blakey (1932), and Kozicky (1948)
	Northern Bobwhite	*Colinus virginianus*	*Eimeria lettyae*	USA	Duodenum with rare parasites in the ileum and cecum	Listlessness, droopiness, and anorexia but no gross or histopathologic lesions noted	Not associated with disease in wild birds	Ruff (1985), and Ruff and Wilkins (1987)
	Greater Sage-Grouse and Ruffed Grouse	*Centrocercus urophasianus* and *Bonasa umbellus*	*Eimeria angusta*	Western USA	Cytoplasm of cecum epithelial cells	Thickening of mucosa, hemorrhage; diarrhea, depression, and weight loss	Historically, significant losses of young sage grouse, no cases documented since 1960s; deaths of ruffed grouse only in captivity	Simon (1940), Honess and Post (1968), Barker et al. (1984), and Connelly et al. (2000)

(*continues*)

Table 8.2. (*Continued*)

Host order	Host common name	Host scientific name	*Eimeria* spp.	Locality	Location of tissue stages	Clinical signs and/or lesions	Mortality	Citations
	Ring-necked Pheasant	*Phasianus colchicus*	*Eimeria colchici*	Worldwide	Cytoplasm of cecum epithelial cells	Petechial hemorrhages in heavy infections of captive birds	Not associated with disease in wild birds	Jones (1966)
	Ring-necked Pheasant	*Phasianus colchicus*	*Eimeria phasiani*	Worldwide	Cytoplasm of small intestine epithelial cells	Ruffled feathers, incoordination, mucoid diarrhea, and decreased weight gain	Not associated with disease in wild birds	Jones (1966), and Trigg (1967)
	Ring-necked Pheasant	*Phasianus colchicus*	*Eimeria duodenalis*	Worldwide	Cytoplasm of small intestine epithelial cells	Anorexia and slight depression but no mortality in captive birds	Not associated with disease in wild birds	Jones (1966)
	Partridge species	*Perdix* spp.	*Eimeria procera*	Europe	Cytoplasm of cecum epithelial cells	White cecal cores with clotted blood, thickened intestinal walls	Not associated with disease in wild birds	Goldová et al. (2000)
	Helmeted Guineafowl	*Numida meleagris*	*Eimeria* sp.	Nigeria, Niger	Cytoplasm of intestine epithelial cells	Intestines thickened and congested, edematous, and/or hemorrhagic	Not typically associated with disease in wild birds	Ayeni et al. (1983)
Columbiformes	Various pigeons and doves	*Columba* and *Streptopelia* spp.	*Eimeria labbeana* and *Eimeria columbarum*	Worldwide	Cytoplasm of small intestine epithelial cells	Intestines distended, inflamed, and contain petechial hemorrhages	Not associated with disease in wild birds	Hunt and O'Grady (1976)
Psittaciformes	Budgerigar	*Melopsittacus undulatus*	*Eimeria dusingi*	Worldwide	Cytoplasm of duodenum epithelial cells	Yellow, pasty feces, enlarged duodenum	Not associated with disease in wild birds	Farr (1960), and Panigrahy et al. (1981)

et al. 2005). The eight species of *Eimeria* described from domestic chickens (*Eimeria acervulina*, *Eimeria brunetti*, *Eimeria maxima*, *Eimeria mitis*, *Eimeria mivati*, *Eimeria necatrix*, *Eimeria praecox*, and *Eimeria tenella*) each infect different regions of the lower gastrointestinal tract and cause variable presentations of disease (McDougald 2003). Not surprisingly, similar species of *Eimeria* have been found in the free-living ancestors of domestic chickens from Asia, the wild Red Junglefowl (*Gallus gallus*) and Ceylon Junglefowl (*Gallus lafayetii*); however, reports of disease in these hosts are lacking (Fernando and Remmler 1973a, b; Long et al. 1974).

Numerous species of Eimeria—*E. dispersa, Eimeria meleagrimitis, Eimeria gallopavonis, Eimeria meleagridis, Eimeria innocua, Eimeria subrotunda, Eimeria adenoeides*, and an undescribed *Eimeria* sp.—have been reported from Wild Turkeys (*Meleagris gallopavo*). None of these species of *Eimeria* have been associated with disease in free-ranging birds, but four species have been associated with disease in captive birds (Table 8.2). In the southeastern US, Prestwood et al. (1971) reported that 50% of poults and 17% of juvenile and adult Wild Turkeys were positive for species of *Eimeria*. Despite the high prevalence, no lesions or clinical disease was observed in any birds. Similar findings were found for pen-raised Wild Turkeys; 66% were positive for *Eimeria* and mixed infections were common (Ruff et al. 1988a).

At least 12 species of *Eimeria* have been reported from various species of quail. *E. dispersa*, the first coccidian described from a quail and also a common parasite of turkeys and pheasants, may cause disease in young Northern Bobwhite (Table 8.2). *Eimeria lettyae* from Northern Bobwhite has been reported from Pennsylvania and Florida (Ruff 1985) but probably occurs throughout the range of the Northern Bobwhite. *E. lettyae* appears to be host specific, as attempts to experimentally infect Japanese Quail (*Coturnix japonica*), Chukar, Ring-necked Pheasants, domestic turkeys, and chickens have failed. Although *E. lettyae* can be pathogenic for young Northern Bobwhite (Table 8.2) (Ruff and Wilkins 1987), coccidiosis does not appear to be a significant disease problem of wild free-ranging species of quail.

Captive Japanese Quail are also susceptible to coccidiosis caused by *Eimeria uzura*, *Eimeria tsunodai*, and *Eimeria taldykurganica* (Ruff et al. 1984), although they have not been reported from wild Japanese Quail. Three-day-old quail were more susceptible to disease (100% mortality) than seventeen-day-old quail (8% mortality) when birds were inoculated with 10^5 oocysts of a mixture of these three species of *Eimeria*. Experimental inoculations of Northern Bobwhite, Chukar, Ring-necked Pheasants, domestic chickens, and domestic turkeys failed to produce infections (Ruff et al. 1984).

Ten species of *Eimeria* have been described from pheasants, of which three—*Eimeria colchici*, *Eimeria duodenalis*, and *Eimeria phasiani*—are associated with severe disease in captive-bred Ring-necked Pheasants (Table 8.2) (Jones 1966). *E. colchici* is considered to be the most pathogenic. Chickens are experimentally susceptible when inoculated with large numbers of oocysts, but development in the cecal epithelial cells is limited and no clinical signs have been observed (Looszova et al. 2001).

Eimeria angusta has been associated with cecal coccidiosis in both captive and free-ranging species of grouse (Table 8.2). Mortality has not been observed in free-ranging Greater Sage-Grouse (*Centrocercus urophasianus*) since the 1960s, presumably because numbers of grouse have declined, leading to decreased crowding and/or stress and reduced transmission of the parasite (Honess and Post 1968; Connelly et al. 2000).

Gruiformes

A total of 13 species of *Eimeria* have been reported from the order Gruiformes, 7 from cranes and 6 from coots and their relatives. All these species infect and develop in intestinal epithelial cells, but 2 species—*Eimeria gruis* and *Eimeria reichenowi*—can disseminate, develop extraintestinally, and cause severe disease (Chapter 9).

Columbiformes

Several species of *Eimeria* have been reported from free-ranging pigeons and doves (Table 8.1). None have been associated with disease among free-ranging birds; however, two species—*Eimeria labbeana* and *Eimeria columbarum*—have caused significant losses of captive pigeons and doves (Table 8.2) (Wages 1987; McDougald 2003).

Psittaciformes

Four species of *Eimeria* have been described from the Psittaciformes, but only *Eimeria dunsingi* has been associated with clinical disease in captive Budgerigars (*Melopsittacus undulatus*) (Farr 1960; Panigrahy et al. 1981). No disease was noted in naturally infected free-ranging Budgerigars or Musk Lorikeets (*Glossopsitta concinna*) from Australia (Gartrell et al. 2000).

Piciformes

There is a single report of clinical coccidiosis and high mortality in captive Toco Toucan (*Ramphastos toco*) infected with an *Eimeria* sp., but clinical or

epizootiological details of the infections were not reported (Martins et al. 2006). *Eimeria forresteri* was described from feces of captive Toco Toucans that did not exhibit clinical disease (Upton et al. 1984).

Passeriformes

Perching birds in the order Passeriformes are rarely infected with species of *Eimeria*. Some reports may be erroneous and could be pseudoparasites ingested with food items. There is one report of hemorrhagic enteritis associated with an *Eimeria* sp. in a Common Hill Mynah (*Gracula religiosa*) (Korbel and Kosters 1998).

CLINICAL SIGNS

Most birds infected with species of intestinal *Eimeria* do not exhibit any clinical signs because low-intensity infections destroy a limited number of epithelial cells that can be quickly replaced. Large numbers of cells are destroyed in infections of moderate to high intensity, leading to reduced food and water consumption, decreased intestinal absorption, and hemorrhage. Affected birds sometimes exhibit diarrhea tinged with blood or mucus, lack of appetite, emaciation, droopiness, loss of coordination, ruffled feathers, and decreased egg production (Hunt and O'Grady 1976; Wages 1987).

Development of clinical signs depends on many factors including intensity of infection, species of parasite, and host factors such as age and health. For example, aberrant hosts may become infected, but replication and oocyst shedding by the parasite may be limited, resulting in no disease (Looszova et al. 2001; Revajová et al. 2006). Dosage can be an important factor that leads to development of disease. Ring-necked Pheasants experimentally infected with low numbers (10,000 oocysts) of *E. phasiani* developed only diarrhea, but when pheasants were inoculated with 100,000 oocysts, birds exhibited ruffled feathers, incoordination, mucoid diarrhea, and decreased weight gain (Trigg 1967). Similarly, experimental infections of Ring-necked Pheasants with variable numbers of *E. colchici* resulted in a dose-dependent disease (Norton 1967). Eight of ten birds exposed to low numbers (20,000 oocysts) survived infections, but none survived exposure to 80,000 or 320,000 oocysts.

PATHOLOGY

Pathologic changes vary widely, depending on the host and parasite species and severity of infection. Grossly, intestines may be ballooned, filled with mucus, hemorrhagic, and discolored (Figure 8.3). Sloughing of the intestinal mucosa is often observed in severe infections. Some species of *Eimeria* cause formation of white caseous cores in the ceca. Among Lesser Scaup with chronic infections, dry crusts have been reported on the mucosal surface of the intestine (Cole 1999). Among species of Galliformes, infections with *Eimeria* cause intestinal damage and changes in intestinal motility that may predispose the gut to infections with other pathogens such as *Clostridium perfringens* or *Salmonella typhimurium*. Cecal coccidiosis can exacerbate infections with *Histomonas meleagridis* (blackhead) (McDougald 2003).

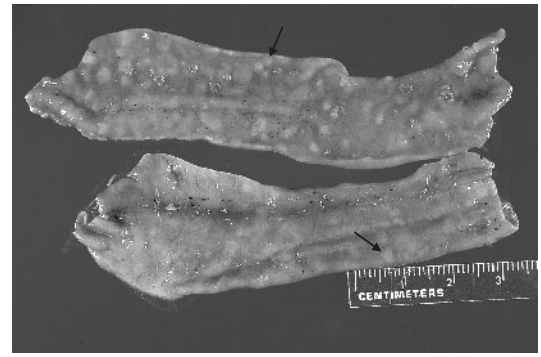


Figure 8.3. Intestine, Common Eider (*Somateria mollissima*). Distinct light-colored areas (arrows) within the wall of the intestine. Courtesy of J. C. Franson, U.S. Geological Survey.

Developing meronts, gamonts, and oocysts of *Eimeria* can easily be observed within intestinal epithelial cells by microscopy (Figure 8.2). Most species of *Eimeria* develop in the cytoplasm of infected cells, but several species from geese develop within the epithelial cell nuclei. In clinically ill birds, extensive histopathologic lesions should be evident including host cell destruction and lymphocytic infiltration (Figure 8.4). However, lymphocytic infiltration may or may not be present depending on species of parasite and severity of infection.

DIAGNOSIS

A diagnosis of coccidiosis is based on detection and identification of oocysts in feces along with clinical signs of the disease in live birds or characteristic lesions at necropsy. Feces collected from live birds or at necropsy can be examined directly for oocysts or by first concentrating oocysts by flotation using standard zinc sulfate or Sheather's sugar. Diurnal periodicity in the shedding of oocysts has not been reported for

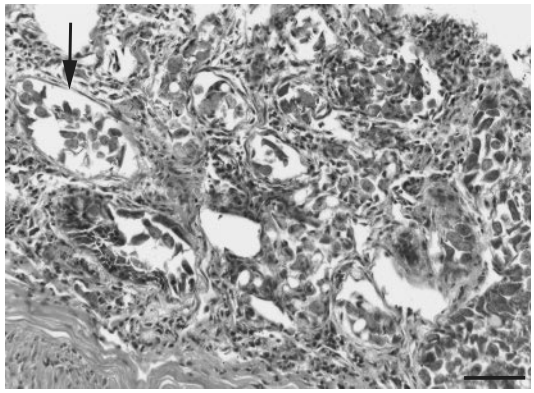


Figure 8.4. Intestine, Lesser Scaup (*Aythya affinis*). Crypts are dilated and contain multiple coccidian stages with small amounts of necrotic cell debris. Crypt epithelium is attenuated or lost (arrow). Small numbers of inflammatory cells, primarily lymphocytes, are present in the surrounding lamina propria. Hematoxylin and eosin stain. Bar = 50 μm. Courtesy of R. W. Gerhold and A. E. Ellis, University of Georgia.

species of poultry *Eimeria* (Long 1982), but has been reported for an *Eimeria* sp. from the Red-legged Partridge (*Alectoris rufa*), which more commonly sheds oocysts in the late afternoon (Villanúa et al. 2006). This phenomenon is also commonly observed among species of *Isospora* that occur in passerines (Barre and Troncy 1974; Brawner and Hill 1999; Misof 2004; M. J. Yabsley, unpublished data). It is unknown whether species of *Eimeria* from wild birds exhibit diurnal periodicity in oocyst shedding, but this should be considered when surveys of wild hosts are done.

For identification of species, oocysts must be allowed to sporulate by placing feces in 1–3% (w/v) potassium dichromate, stored at room temperature, and examined daily for evidence of sporulation. Sporulation is facilitated by placing the fecal/potassium dichromate solution in a covered petri dish. Enough potassium dichromate solution must be used to prevent desiccation. Once sporulated, feces should be stored at 4°C to maintain morphologic characteristics. Because most infections are nonclinical, the finding of oocysts in a fecal sample does not indicate that a species of *Eimeria* is the cause of disease; significant pathologic lesions must be present at necropsy or other potential causes of the illness must be ruled out in live birds.

IMMUNITY

The bulk of knowledge related to development of host immunity to intestinal species of *Eimeria* is derived from studies of the domestic fowl. Immunity in chickens against coccidiosis is primarily T-cell mediated (reviewed by Lillehoj and Lillehoj 2000 and Yun et al. 2000). Anti-*Eimeria* IgM, IgY, and IgA antibodies are produced, but these antibodies are not effective at eliminating the parasite. Some level of protection develops in young birds that survive infection; however, this protection is specific to species of *Eimeria*; that is, it does not confer cross-protection against other species or strains of *Eimeria*. In some cases, birds may not develop complete immunity and the host becomes infected, but the infection will be less severe and result in development of fewer numbers of infective stages. For example, experimental infection of Northern Bobwhite with *E. lettyae* did not prevent reinfection (Ruff 1985). Lack of disease in wild birds is probably related to repeated exposures to low numbers of oocysts, which causes limited pathology and allows development of immunity.

PUBLIC HEALTH CONCERNS

There are no known public health concerns regarding avian species of intestinal *Eimeria*.

DOMESTIC ANIMAL CONCERNS

Intestinal coccidiosis is a serious disease of many species of domesticated and captive wild birds and is associated with how birds are managed in captivity. Because of the presumed strict host specificity of the avian species of *Eimeria*, wild avian coccidia pose little threat to unrelated domesticated birds, but mixing of wild birds of different species is still discouraged.

Wild birds and their domesticated counterparts (e.g., Wild Turkey and domesticated turkey) can be infected by the same species of coccidia; however, rarely would these wild birds pose any unusual threat to domesticated birds, as domesticated birds are commonly infected with the same repertoire of coccidia. For example, four species of *Eimeria* of Wild Turkeys—*E. adenoeides*, *E. dispersa*, *E. gallopavonis*, and *E. meleagrimitis*—are significant pathogens of domestic turkeys (McDougald 2003); however, these infections circulate within domestic turkeys without exposure to Wild Turkeys. Interestingly, several species of *Eimeria* have been associated with coccidiosis in domestic ducks (*Eimeria saitamae*) and domestic geese (*Eimeria anseris*, *Eimeria kotlani*, and *Eimeria nocens*) but have not been found to cause disease in

free-ranging waterfowl (Levine 1953; Inoue 1967; Gajadhar et al. 1983a).

Wild birds that are kept in zoological parks or other captive facilities are more likely to develop coccidiosis than their free-ranging counterparts (Panigrahy et al. 1981; Barker et al. 1984; Swayne et al. 1991; Giacomo et al. 1997; Novilla and Carpenter 2004). This problem can be compounded in facilities where large numbers of very closely related host species are housed together because closely related hosts may be susceptible to the same coccidian species.

WILDLIFE POPULATION IMPACTS

Outbreaks of intestinal coccidiosis are occasionally reported and cause mortality of free-ranging birds, but this condition does not appear to have a significant impact on wild populations. Reduced egg production and fertility have been reported in experimental studies of coccidiosis and could also occur in free-ranging birds. For example, adult Northern Bobwhite and Japanese Quail experimentally infected with species of *Eimeria* did not die, but egg production and fertility were reduced and maturation of males was delayed (Ruff et al. 1984, 1988b; Ruff and Wilkins 1987). Reductions in weight gain have not been reported in young wild birds with eimerian infections; however, this phenomenon is common in both domestic fowl and experimental studies and could be unrecognized in wild birds (Ruff et al. 1984). Field studies of the subclinical effects of coccidian infections are needed.

TREATMENT AND CONTROL

Much of what is known about treatment or control of avian coccidiosis is derived from studies concerning *Eimeria* of domestic fowl and birds in zoological collections. Historically, the use of anticoccidial feed or water supplements (e.g., amprolium and monensin) has been the primary method for controlling coccidiosis for poultry producers. In recent years, resistance has been documented against many of the common anticoccidial drugs (Martin et al. 1997).

Poultry coccidia induce a strong immunity; therefore, vaccination has been investigated as an alternative to drugs for controlling disease. Early vaccines were made of live, wild-type, or attenuated parasites, but these vaccines were specific to species of *Eimeria* and, in some cases, specific to particular parasite strains. Wild-type vaccines work by providing a low-level of exposure, so uniform exposure among all birds is essential to preventing development of disease and for the development of protective immunity against future infections with large numbers of parasites (Shirley et al. 2005). Attenuated strains (those that have a reduced reproductive capacity) are as immunogenic as wild-type strains but reduce the risk of clinical disease. New vaccines based on recombinant protective antigens are under development and may further increase the ability to vaccinate poultry safely and cheaply (Shirley et al. 2007). Currently, vaccines are parasite species/strain specific, difficult to produce, costly, and would not be feasible for use in wild birds.

Captive wild birds that develop coccidiosis may respond to commercially available anticoccidial drugs, but studies on their effectiveness and safety are limited. In addition, each species of *Eimeria* may vary in susceptibility to the most commonly used drugs. For example, cecal coccidiosis (*Eimeria colchici*) in Ring-necked Pheasants is easily controlled with medicated feed containing zoalene or amprolium, but sulfaquinoxaline is ineffective (Norton 1967). Treatment of captive Rock Pigeons (*Columba livia*) infected with *E. labbeana* was successful with amprolium and sulfaquinoxaline (Hunt and O'Grady 1976). Sulfamethazine has also been used successfully to treat coccidiosis in captive Rock Pigeons and Budgerigars when added to drinking water (Panigrahy et al. 1981; McDougald 2003). Maintaining clean housing or raising birds on wire prevents buildup of infective oocysts and can decrease the risk of coccidiosis.

Outbreaks of coccidiosis in free-ranging birds are difficult to treat because neither dosage nor regular dosing intervals can be easily controlled. Preventing crowding or stress may be more effective approaches to reducing or preventing outbreaks of coccidiosis in free-ranging birds.

MANAGEMENT IMPLICATIONS

Infections of free-ranging birds with species of *Eimeria* are common, but coccidiosis among these birds in undisturbed habitat is rarely a significant problem. Outbreaks can occur when factors conspire to crowd or stress birds (e.g., breeding and loss of habitat). Most importantly, keeping wild birds in captivity can result in significant disease from coccidiosis.

RENAL *EIMERIA*

ETIOLOGY

Species of *Eimeria* are the primary cause of renal coccidiosis in birds. Although many host species harbor both renal and intestinal coccidia, the species of *Eimeria* that infect the kidneys are distinct and different from those that occur in intestinal tissues (Gajadhar et al. 1983a, b; Yabsley and Gibbs 2006).

EPIZOOTIOLOGY

Similar to intestinal *Eimeria*, species of renal *Eimeria* have direct life cycles. Instead of developing in intestinal epithelial cells, sporozoites of renal species invade and develop in renal epithelial cells. Oocysts are passed unsporulated into the ureters and out of the cloaca. Oocysts shed in the feces of an infected host sporulate in the environment to become infective. As with the intestinal coccidia, sporulation is dependent on several factors including temperature, moisture, and levels of oxygen.

Transmission of renal coccidia probably occurs in the fall as the prevalence in ducks is significantly higher in the fall than in spring in Saskatchewan, Canada, and Sweden (Walden 1963; Gajadhar et al. 1983a). Likewise, more geese were found infected in the fall than spring in Saskatchewan and Manitoba, Canada, and all reported outbreaks of renal coccidiosis of Double-crested Cormorants (*Phalacrocorax auritus*) have occurred between November and January (Gajadhar et al. 1983a; Clinchy and Barker 1994; Yabsley et al. 2002). As with intestinal coccidia, juvenile birds are more likely to be infected (Walden 1963; Nation and Wobeser 1977; Yabsley and Gibbs 2006). This may explain why prevalence increases during the fall when large numbers of naïve young birds enter the population.

HOST RANGE AND PREVALENCE

Renal coccidia have been reported from numerous families of birds, but the greatest diversity of infected hosts is among species of Anseriformes. Pathogenic species of renal *Eimeria* and their associated traits are listed in Table 8.3.

Procellariiformes

In a study of the potential causative agents of "limey disease" (soiling of vent feathers by whitish excrement) in Short-tailed Shearwaters (*Puffinus tenuirostris*) from Tasmania, Munday et al. (1971) discovered renal coccidia (later described as *Eimeria serventyi*) in underweight chicks (Table 8.3; Pellerdy 1974). Renal coccidia have also been reported from Cory's Shearwater (*Calonectris diomedea*), but no morphologic or pathologic information is available (Munday et al. 1971).

Pelecaniformes

From 1984 to present, more than 1,300 Double-crested Cormorants were reported to have died of renal coccidiosis during 11 mortality events (Table 8.3) (Yabsley et al. 2002; US Geological Survey, National Wildlife Health Center, unpublished data). *Eimeria auritusi* was described during an outbreak of coccidiosis in Georgia (Yabsley et al. 2002). While several substantial outbreaks of renal coccidiosis have been reported, a recent study in Georgia showed that 18 of 80 (23%) healthy double-crested cormorants were positive for renal coccidia identified as *E. auritusi* (Yabsley and Gibbs 2006). In general, low numbers of oocysts (<10) were detected in positive kidney samples and no gross lesions were noted, indicating that *E. auritusi* is not always pathogenic for double-crested cormorants.

Anseriiformes

Two species of renal *Eimeria* have been described from ducks: *Eimeria somateriae* from the common eider and long-tailed duck (*Clangula hyemalis*) and *Eimeria boschadis* from the mallard (*Anas platyrhynchos*) (Table 8.3) (Christiansen 1952; Walden 1963). However, the description of *E. boschadis* was based on unsporulated oocysts, making it an invalid description (Gajadhar et al. 1983a, b). Uncharacterized renal coccidia have been reported from virtually all species of ducks where substantial numbers of individuals have been examined. Many of these unidentified species of *Eimeria* differed morphologically from recognized species, indicating that many new species still need to be described.

In a survey of 336 ducks of 12 species from Saskatchewan, Canada, 151 (45%) were infected with renal coccidia, most of which are undescribed species (Gajadhar et al. 1983a). Renal coccidia were detected in 11 of the species of ducks that were examined including the American Widgeon (*Anas americana*), Blue-winged Teal (*Anas discors*), Eurasian Teal (*Anas crecca*), Gadwall (*Anas strepera*), Mallard, Northern Pintail (*Anas acuta*), Northern Shoveler (*Anas clypeata*), Canvasback (*Aythya valisineria*), Common Goldeneye (*Bucephala clangula*), Lesser Scaup, and Redhead (*Aythya americana*). Eleven Bufflehead (*Bucephala albeola*) were sampled and were found to be negative. Renal coccidia were detected in one of six Red-breasted Mergansers (*Mergus serrator*) from Florida (Forrester and Spalding 2003).

Renal coccidia are common in several species of geese and have been reported worldwide from Greater and Lesser Snow Geese (*Chen caerulescens atlantica* and *Chen caerulescens caerulescens*), Ross' Geese (*Chen rossii*), Graylag Geese (*Anser anser*), domestic geese, and multiple subspecies of Canada Geese (*Branta canadensis*) (Gajadhar et al. 1983a, b). To date, *E. truncata* is the only species formally described from geese (Table 8.3). It is likely that multiple species of renal *Eimeria* infect geese, but controlled experimental infections and/or molecular studies are

Table 8.3. Common pathogenic species of renal *Eimeria*.

Host order	Host common name	Host scientific name	*Eimeria* spp.	Locality	Mortality	Citations
Sphenisciformes	Little Penguin	*Eudyptula minor*	*Eimeria* sp.	Victoria, Australia	Outbreaks associated with concurrent parasitic and fungal infections	Obendorf and McColl (1980)
Procellariiformes	Short-tailed Shearwater	*Puffinus tenuirostris*	*Eimeria serventyi*	Tasmania	Loss of young underweight birds; morbidity as high as 1–5%; can cause high mortality	Munday et al. (1971), and Pellerdy (1974)
Pelecaniformes	Double-crested Cormorant	*Phalacrocorax auritus*	*Eimeria auritusi*	USA	Small- to medium-sized outbreaks	Yabsley et al. (2002)
Anseriformes	Common Eider	*Somateria mollissima*	*Eimeria somateriae*	Denmark, Iceland, Sweden, and Scotland	Occasional deaths; a few epornitics	Christiansen (1952), Persson et al. (1974), Mendenhall (1976), and Skirnisson (1997)
	Canada Goose	*Branta canadensis*	*Eimeria truncata*	USA	Few numbers of birds; many infections are subclinical	Farr (1954), and Tuggle and Crites (1984)
	Lesser Snow Goose	*Chen caerulescens caerulescens*	*Eimeria truncata*	USA, Canada	Occasionally in malnourished geese	Gomis et al. (1996)
	Graylag Goose	*Anser anser*	*Eimeria truncata*	Finland	Small outbreaks; many infections are subclinical	Oksanen (1994)

needed to establish the true number. Because of the difficulty in getting oocysts of renal coccidia to sporulate under typical conditions that are successful for intestinal *Eimeria*, experimental infections are difficult, but Gajadhar et al. (1982) have successfully studied the life cycle of a renal *Eimeria* in Lesser Snow Geese.

Mortality events caused by *E. truncata* have been reported in free-ranging Canada Geese, Lesser Snow Geese, and Graylag Geese (Table 8.3) (Farr 1954; Tuggle and Crites 1984; Oksanen 1994; Gomis et al. 1996). The prevalence of renal coccidia from 309 Canada Geese along the Mississippi flyway was almost 7% (Tuggle and Crites 1984). The majority of infections were regarded as subclinical, but renal coccidiosis was determined as the cause of death for one goose found dead during the study.

Charadriiformes

Eimeria wobeseri and *Eimeria goelandi* were described from the European Herring Gull (*Larus argentatus*) by Gajadhar and Leighton (1988). Neither species was associated with disease. Interestingly, *E. goelandi* sporulated endogenously, which is unusual for members of the genus *Eimeria*. Morphologically, *E. goelandi* is correctly described as a species of *Eimeria* as it has four sporocysts, each containing two sporozoites.

A single coccidian species, *Eimeria fraterculae*, has been described from the Atlantic Puffin (*Fratercula arctica*) from Newfoundland, Canada (Leighton and Gajadhar 1986). In the original description, 7 of 50 nestling puffins were infected, but none exhibited any clinical signs.

Renal coccidia have been reported from both captive-raised and free-ranging American Woodcocks (*Scolopax minor*) from numerous states in the eastern US and Ontario, Canada (Locke et al. 1965; Pursglove 1973). Prevalence in free-ranging and captive American Woodcocks was 28 and 19% (72/265 and 6/31), respectively (Pursglove 1973). Clinical disease was not apparent and kidneys of many infected birds appeared grossly normal with the exception of occasional whitish streaks. Developmental stages of the parasites were observed in collecting tubules in histological sections. These tubules were markedly enlarged with cell nuclei displaced toward the basement membrane. No fresh material was available in either study to attempt sporulation and description of oocysts.

Sphenisciformes

Renal coccidiosis, together with extreme gastric parasitism and aspergillosis, was associated with mortality of Little Penguins (*Eudyptula minor*) in Victoria, Australia (Table 8.3) (Obendorf and McColl 1980). Three-quarters of the 48 penguins submitted for necropsy between 1977 and 1978 were in poor body condition and 23% had gross and histopathologic lesions associated with renal coccidiosis.

Apterygiformes

An unclassified coccidian species has been reported to cause renal coccidiosis in a captive-bred, 1-month-old North Island Brown Kiwi (*Apteryx mantelli*) (Thompson and Wright 1978). The bird was depressed and at necropsy had pale kidneys. Three other clinically normal kiwis at the same location were found to be passing oocysts in their feces. None sporulated, so specific identification was not possible.

CLINICAL SIGNS AND PATHOLOGY

As with intestinal *Eimeria* species, most reports of species of renal *Eimeria* come from surveys of presumably healthy birds and clinical signs are rarely observed. If birds develop disease due to renal coccidia, they are often found dead. Clinical signs observed in experimentally infected or domestic birds include diarrhea, weakness, ataxia, difficulty in flying, depression, lack of appetite, and emaciation (Gajadhar et al. 1983b; McDougald 2003).

The majority of infections with renal coccidia result in few or no gross lesions. Birds may be emaciated, but if death occurred quickly, they may still be in good physiological condition (Obendorf and McColl 1980; Yabsley et al. 2002). In low-intensity infections, kidneys may be slightly enlarged and mottled with rare white streaks or nodules. In severe, very intense infections, kidneys are pale, grossly enlarged, friable, and mottled with white streaks and nodules (Figure 8.5).

Microscopic lesions consist primarily of dilation of infected tubules with distortion of normal architecture and associated inflammation (Leighton and Gajadhar 1986; Yabsley et al. 2002). Infected cells are swollen and contain numerous developing intracellular parasites (Figure 8.6). Large numbers of oocysts can obstruct tubules (Figure 8.6) and may be found in ureters. Rarely, oocysts may enter the bloodstream and become lodged in organs such as the lungs (Yabsley et al. 2002). Affected tubules are often surrounded by infiltrates of macrophages, lymphocytes, plasma cells, and heterophils. Necrosis of infected cells and tubules is often evident (Thompson and Wright 1978; Gajadhar et al. 1983b; Yabsley et al. 2002). In mild infections, lesions are limited to few infected tubules and are surrounded by normal kidney tissue, but in severe cases, most tubules are infected with parasites, resulting in little normal kidney tissue being present.

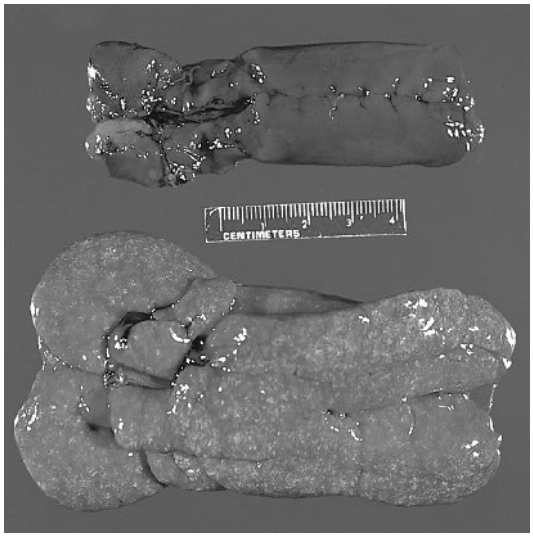


Figure 8.5. Kidney, Double-crested Cormorants (*Phalacrocorax auritus*). (a) Normal size and color; (b) enlarged kidneys with pale areas from a bird infected with renal coccidiosis. Courtesy of J. C. Franson, U.S. Geological Survey.

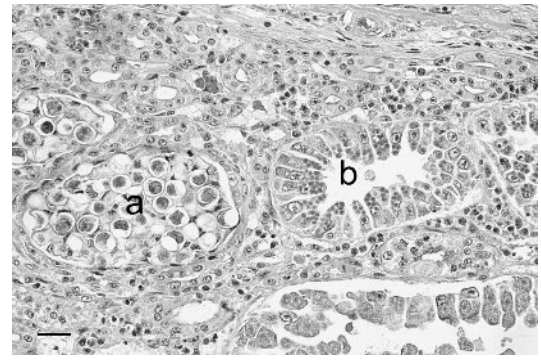


Figure 8.6. Kidney, Double-crested Cormorant (*Phalacrocorax auritus*). (a) Renal tubular epithelial cells distended by oocysts of *Eimeria auritusi* in their cytoplasm. (b) Multiple developing gamonts per infected epithelial cell. Hematoxylin and eosin stain. Bar = 25 μm. Reproduced from Yabsley et al. (2002), with permission of the *Journal of Parasitology.*

DIAGNOSIS

In the cases of suspected fatal renal coccidiosis, gross pathology is highly suggestive; however, lesions in fatal cases must be sufficient to impede kidney function, and other causes of death must be ruled out. For confirmation, oocysts can be observed in direct smears of kidney tissue or parasites can be observed in histological sections of kidney tissue.

Many hosts infected with renal coccidia have only developing parasites in a limited number of renal tubules; therefore, morbidity and mortality in most infected hosts is low. For detection of oocysts in low-intensity infections from asymptomatic birds, kidney tissues should be placed in 2% (w/v) potassium dichromate and disrupted with a blender or tissue macerator. Care must be taken during dissection to avoid contamination of samples with intestinal contents that may contain unrelated species of coccidia. The tissue can be filtered through a layer of cheese cloth and the filtrate, containing oocysts and small pieces of kidney tissue, centrifuged so that the resulting pellet can be examined for oocysts by direct examination or standard zinc sulfate or Sheather's sugar flotation. Asymptomatic infections can also be detected during histopathologic examination of kidney tissue; developing meronts, gamonts, and oocysts are easily observed in the cytoplasm of tubular epithelial cells.

For specific diagnosis, oocysts must be sporulated in potassium dichromate as described for intestinal *Eimeria*. To date, researchers have had limited success in sporulation of many samples of renal coccidia. This has hampered detailed studies of oocyst morphology and made experimental studies difficult.

Other renal coccidia reported from avian species include disseminated toxoplasmosis (*Toxoplasma gondii*) in Wild Turkeys (Chapter 11) (Quist et al. 1995), renal cryptosporidiosis in many avian species (Chapter 10) (Gardiner and Imes 1984; Randall 1986; Trampel et al. 2000), and a probable case of *Klossiella* from a Great Horned Owl (*Bubo virginianus*) (Helmboldt 1967).

IMMUNITY

Domestic geese develop immunity to reinfection with *E. truncata* (McDougald 2003). Nothing is known about the development of immunity to renal *Eimeria* in naturally infected free-ranging birds. Similar to intestinal coccidia, infection of young birds with low levels of renal coccidia probably results in mild infections that may protect birds from future heavy infections and subsequent clinical disease.

PUBLIC HEALTH CONCERNS

There are no known public health concerns associated with renal coccidia.

DOMESTIC ANIMAL HEALTH CONCERNS

Eimeria truncata is a significant pathogen of domestic geese throughout the world (Gajadhar et al. 1983b). There is evidence to suggest that *E. truncata* occurs in multiple species of wild geese, but controlled experimental infections and/or molecular studies are needed to confirm this finding. Wild geese probably do not pose a threat to domestic geese because the pathogen is frequently present in domestic geese populations that do not have exposure to wild geese.

WILDLIFE POPULATION IMPACTS

Small- to medium-sized outbreaks of renal coccidiosis involving up to several hundred birds have been reported in several species of free-ranging birds, but these outbreaks do not appear to have had a significant impact on wild populations.

TREATMENT AND CONTROL

The efficacy and safety of common anticoccidial drugs for treating renal coccidiosis in wild birds is unknown. Numerous coccidiostats and anticoccidial drugs commonly used in domestic chickens (e.g., amprolium, dulfaquinoxaline, clopidol, zoalene, narasin, nicarbazin, robenidin, and salinomycin) are tolerated by domestic geese and are used to control both intestinal and renal coccidiosis. However, current therapy in domestic birds is aimed at reducing clinical signs and numbers and transmission of parasites rather than complete elimination of the parasites. Control of transmission in the wild is not feasible, based on difficulties in administering drugs and the widespread occurrence of asymptomatic infections in wild birds.

MANAGEMENT IMPLICATIONS

Coccidian infections in free-ranging birds in undisturbed habitats are rarely a significant problem because birds harbor asymptomatic infections that rarely cause mortality. Any activity that concentrates birds (e.g., extreme weather conditions or habitat degradation) can lead to outbreaks of coccidiosis. Renal coccidiosis may become a problem when susceptible wild avian species are kept in captivity.

HEPATIC *EIMERIA*

One species of *Eimeria* has been reported to develop in the cytoplasm of bile duct epithelial cells of the Magpie-lark from Australia (Reece 1989). The single infected bird was extremely emaciated and the liver was enlarged with many white foci. Oocysts of this species of *Eimeria* sporulated within the liver tissue and were passed in the feces as fully developed oocysts. Endogenous sporulation also occurs in species of renal *Eimeria* from gulls. While *Eimeria grallinida* was proposed as the name for the parasite, a detailed description was not given.

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9
Disseminated Visceral Coccidiosis in Cranes

Marilyn G. Spalding, James W. Carpenter, and Meliton N. Novilla

INTRODUCTION

Disseminated visceral coccidiosis (DVC) is a widely distributed intestinal and extraintestinal granulomatous disease of cranes caused by infection with intracellular apicomplexan protozoan parasites from the genus *Eimeria*. Two species of *Eimeria* are associated with the disease: *Eimeria reichenowi* and, to a lesser extent, *Eimeria gruis*.

Disseminated visceral coccidiosis has caused morbidity in various species of cranes. The most significant role of this disease has been in the captive rearing of Whooping Cranes (*Grus americana*) for reintroduction (Carpenter et al. 1980). Although the prevalence of DVC in wild Sandhill Cranes (*Grus canadensis*) and recently released Whooping Cranes is high, there have been no records of mortality of wild birds without other contributing factors.

SYNONYMS

Systemic coccidiosis, extraintestinal coccidiosis.

HISTORY

The agents of DVC were discovered prior to the recognition of the disseminated form of the disease. Both species, *E. reichenowi* and *E. gruis*, were originally described by Yakimoff and Matschoulsky (1935) from a captive Demoiselle Crane (*Anthropoides virgo*) in a zoo in Russia. Pande et al. (1970) then described *Eimeria grusi* n. sp. (note different spelling, considered a junior synonym of *E. reichenowi*) from a Sarus Crane (*Grus antigone*) in a zoo in India. Nodular granulomatous oral lesions containing protozoa, later to be ascribed to DVC, were first observed in captive sandhill cranes at the Patuxent Wildlife Research Center (PWRC) in Laurel, Maryland (Carpenter et al. 1979).

The disease was first recognized and named after a die-off event took place at PWRC in 1978. Three juvenile (13–18 days old) and one adult (9 years old) Whooping Cranes died (Carpenter et al. 1980). At the time of the outbreak, the Whooping Cranes were highly endangered with fewer than 100 birds remaining in both the captive and wild population (Doughty 1989). Documentation of DVC in wild cranes based on oral granulomas and a few mortalities seems to be limited to North America, Korea, and Japan (Carpenter et al. 1979; Forrester and Spalding 2003; Watanabe et al. 2003). The pathogenesis of DVC was later characterized experimentally in Sandhill Cranes and reviewed by Novilla and Carpenter (2004).

HOST RANGE

Five species of *Eimeria* have been described in 8 of the 15 species of cranes in the world, including cranes from North America, Europe, Asia, and Africa, but only two, *E. reichenowi* and *E. gruis*, can be considered common, and are implicated as causes of DVC. It is these two species that are further discussed in this chapter (Table 9.1). The only documentation of DVC-infected wild birds comes from North American Whooping and Sandhill Cranes, and Asian White-naped (*Grus vipio*) and Red-crowned Cranes (*Grus japonensis*). Information from other crane species comes from captive birds in zoos and is not necessarily representative of wild populations. Table 9.1 lists host species for which data are available, their natural distribution, and evidence of infection with *E. reichenowi* and *E. gruis*. We found no reports of *E. reichenowi* and *E. gruis* in cranes of the subspecies *Balearica*, a separate subfamily that includes the crowned cranes of Africa, in spite of their common occurrence in zoos.

ETIOLOGY

Two species of *Eimeria* are implicated as causes of DVC. This is somewhat surprising since species of

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Table 9.1. Distribution and prevalence of oocyst shedding, oral granulomas, postmortem lesions, and mortality of *Eimeria* spp. infections in both captive and wild cranes.

Host and distribution	Fecal oocysts			Oral granulomas	Postmortem lesions	Mortality	References
	Eimeria sp.	*Eimeria gruis*	*Eimeria reichenowi*				
Blue Crane (*Anthropoides paradiseus*): southern Africa							
Georgia, USA	—	—	—	—	1	1	S. E. Little (unpublished data) and S. P. Terrell (unpublished data)
Demoiselle Crane (*Anthropoides virgo*): Asia, Africa							
Russia zoo, captive	+*	+	+	—	—	—	Yakimoff and Matschoulsky (1935)
Sandhill Crane (*Grus canadensis*): North America							
Florida, USA	116/226 (51%)	61/161 (38%)	68/164 (41%)	192/423 (45%)	—	3/226 (1%)	M. G. Spalding (unpublished data)
New Mexico, USA	160/212 (75%)	139/212 (66%)	118/212 (56%)	42/64 (67%)	64/64 (100%†) 24/58 (41%) GI	—	Parker and Duszynski (1986)
Maryland, USA, captive	—	—	—	31/95 (33%)	—	6	Carpenter et al. (1979)
Maryland, Texas, USA, captive	16/16 (100%)	15/16 (94%)	16/16 (100%)	—	—	—	Forrester et al. (1978)
Maryland, USA, experimental	—	—	—	—	0/10 (0%)	—	Carpenter et al. (1992)
Maryland, USA, experimental	—	—	—	—	—	3/11 (27%)	Novilla et al. (1989)

Lesser Sandhill Crane (*Grus canadensis canadensis*): East Siberia, western North America							
New Mexico, USA	60/90 (67%)	52/90 (58%)	42/90 (47%)	—	—	—	Parker and Duszynski (1986)
Texas, USA	+	4/16 (25%)	5/16 (31%)	—	—	—	Courtney et al. (1975)
Canadian Sandhill Crane (*Grus canadensis rowani*): western North America							
Texas, USA	3/3 (100%)	3/3 (100%)	3/3 (100%)	—	—	—	Forrester et al. (1978)
Greater Sandhill Crane (*Grus canadensis tabida*): North America							
Arizona, USA	+	5/14 (36%)	4/14 (29%)	—	—	—	Courtney et al. (1975)
Florida, USA	2/73 (3%)	62/72 (86%)	66/72 (92%)	—			Courtney et al. (1975)
Florida, USA	32/51 (63%)	11/29 (38%)	9/29 (31%)	14/51 (27%)	—	—	M. G. Spalding (unpublished data)
Indiana, USA	+	—	+	—	4/4 (100%)	—	Carpenter et al. (1984)
New Mexico, USA	40/50 (80%)	36/50 (72%)	32/50 (64%)	—	—	—	Parker and Duszynski (1986)
Saskatchewan, Canada	—	—	—	—	1/1 (100%)	—	Carpenter et al. (1984)
Texas, Nebraska, Oklahoma, Alaska, USA, and Saskatchewan, Canada	—	—	—	—	91/382 (24%)	—	Carpenter et al. (1984)

(*continues*)

Table 9.1. (*Continued*)

Host and distribution	Fecal oocysts			Oral granulomas	Postmortem lesions	Mortality	References
	Eimeria sp.	*Eimeria gruis*	*Eimeria reichenowi*				
Maryland, USA, captive	4/4 (100%)	3/4 (75%)	4/4 (100%)	—	—	—	Forrester et al. (1978)
Maryland, USA, experimental	—	—	—	0/12 (0%)	8/12 (67%)	1/12 (8%)	Carpenter et al. (1984)
Florida Sandhill Crane (*Grus canadensis pratensis*): Florida, USA							
Florida, USA	3/14 (21%)	11/14 (79%)	12/14 (86%)	—	—	—	Courtney et al. (1975)
Florida, USA	72/144 (50%)	46/119 (39%)	55/122 (45%)	173/423 (41%)	—	—	M. G. Spalding (unpublished data)
Maryland, USA, captive	5/5 (100%)	5/5 (100%)	5/5 (100%)	—	—	—	Forrester et al. (1978)
Mississippi Sandhill Crane (*Grus canadensis pulla*): Mississippi, USA							
Mississippi, USA[‡]	+	—	—	—	2	2	N. J. Thomas (unpublished data) and J. C. Franson (unpublished data)
Maryland, USA, captive	1	1	1	—	—	—	Forrester et al. (1978)
White-naped Crane (*Grus vipio*): East Asia							
Chulown, Korea		—	—	—	1	1	Kwon et al. (2006)
Korea zoo, captive	—	—	—	—	1	1	Kim et al. (2005)
the Netherlands, captive	—	—	—	—	1	1	Dorrestein and Van Den Brand (2006)
Sarus Crane (*Grus antigone*): India, Asia, Australia							
India zoo, captive	+	—	+[§]	—	—	—	Pande et al. (1970)

Hooded Crane (*Grus monacha*): East Asia							
New York, USA, captive	—	—	—	—	1	1	T. McNamara and E. C. Greiner, unpublished data
Kagoshima, Japan, captive[¶]	—	—	—	—	4	4	Shimizu et al. (1987)
Red-crowned Crane (*Grus japonensis*): East Asia							
Hokkaido, Japan	91/377 (24%)	+	+	—	—	—	Watanabe et al. (2003)
Hokkaido, Japan, captive	+	+	+	—	—	—	Watanabe et al. (2003)
Whooping Crane (*Grus americana*): North America							
Florida, USA, reintroduced	56/686 (8%)	22/656 (3%)	33/656 (5%)	305/656 (46%)	79/121 (65%)	0/121 (0%)	M. G. Spalding (unpublished data)
Florida, USA, >1 year reintroduced	12/143 (8%)	4/139 (3%)	9/139 (6%)	62/146 (42%)	38/66 (58%)	0/66 (0%)	M. G. Spalding (unpublished data)
Texas, USA	6/19 32%	4/19 21%	2/19 11%	—	—	—	Forrester et al. (1978)
Maryland, USA, captive	—	—	—	—	4/4 (100%)	4/21 (19%)	Carpenter et al. (1980) and Novilla et al. (1989)
Maryland, USA, captive	2/16 (13%)	—	2/16 (13%)	—	—	—	Forrester et al. (1978)

Note: Hosts are wild unless otherwise indicated. Dashes indicate that no data are available.
[*]Indicates samples were positive but exact numbers were not given.
[†]Each bird had a lesion in at least one tissue type; GI, gastrointestinal tract.
[‡]Released captive raised birds in wild for >5 months.
[§]Described as *Eimeria grusi*, synonym with *Eimeria reichenowi*.
[¶]Reported as *Hepatozoon*-like protozoal disease lacking sexual reproductive stages, probably DVC.

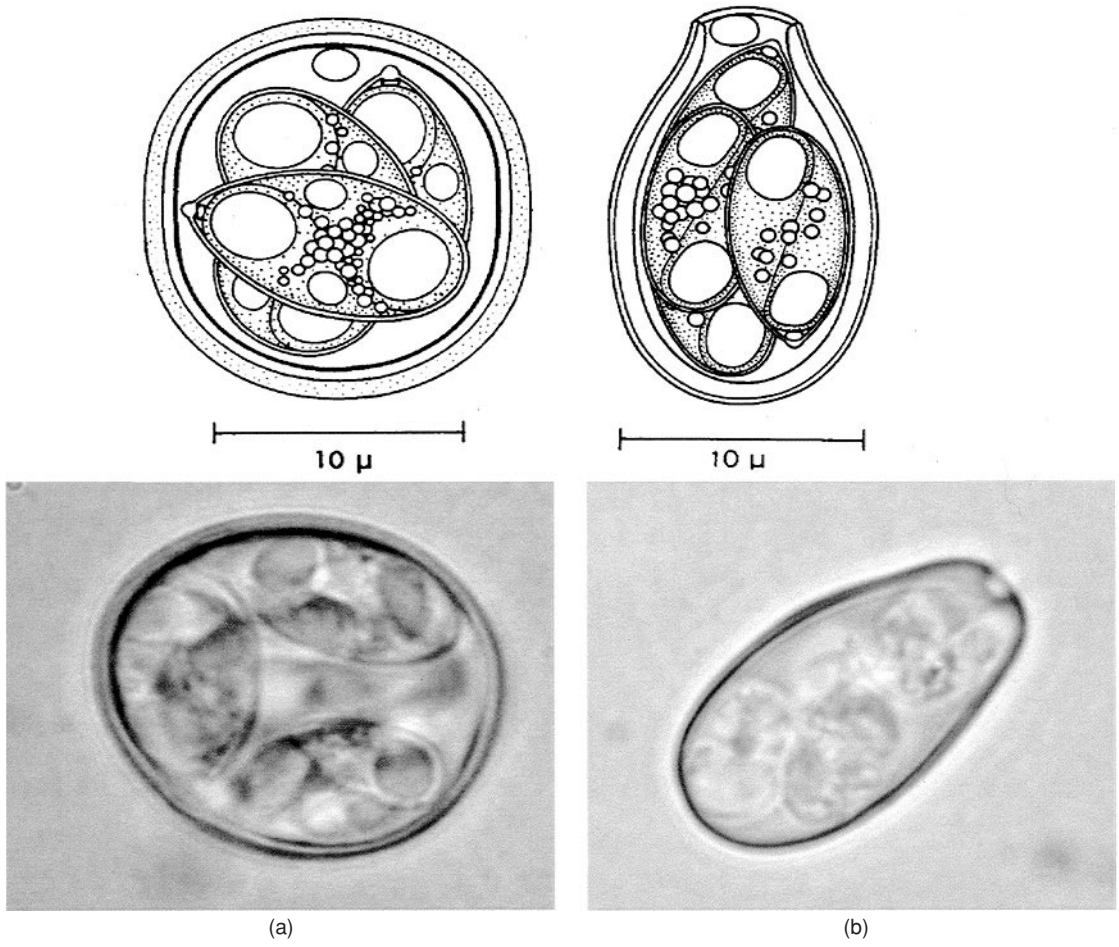


Figure 9.1. Photomicrographs and line drawings of oocysts of *Eimeria reichenowi* (a) and *Eimeria gruis* (b) from the feces of a Sandhill Crane (*Grus canadensis*). Line drawing reprinted from Courtney et al. (1975), with permission of the *Journal of Parasitology*.

Eimeria are generally very host specific (Marquardt 1973). Even more interesting is the fact that both *E. reichenowi* and *E. gruis* frequently coinfect most of the host populations that have been examined. Phylogenetically, *E. reichenowi* and *E. gruis* collected from Red-crowned, Hooded (*Grus monacha*), and White-naped Cranes are similar but distinct from each other, forming their own cluster when compared with species of *Eimeria* from chickens, ruminants, and rodents that infect the intestine only. This indicates that these two eimerians may have evolved independently from the species of *Eimeria* found only in the intestine (Matsubayashi et al. 2005; Honma et al. 2007).

In experimental studies, DVC has been reproduced using inocula-containing mixtures of oocysts of both *E. reichenowi* and *E. gruis* and inocula-containing oocysts of either species alone (Novilla et al. 1981, 1989; Augustine et al. 1998, 2001). However, the predominance of data from experimental studies and mortality in captive birds indicates that *E. reichenowi* is more often associated with the acute pathogenic form of DVC. In Whooping Cranes that died at PWRC, oocysts recovered from the feces and intestinal stages of the parasites morphologically resembled *E. reichenowi* (Carpenter et al. 1980).

Oocysts of *E. reichenowi* and *E. gruis* are distinctly different in morphology (Figure 9.1). Oocysts of *E. gruis* are ellipsoid to pyriform and measure on average 18 × 11.4 μm, whereas oocysts of *E. reichenowi* are round to ovoid/ellipsoid measuring on average

17.8 × 15.3 μm (Courtney et al. 1975). Comparative measurements with other species are presented elsewhere (Courtney et al. 1975; Novilla et al. 1981; Parker and Duszynski 1986).

Three other species of *Eimeria* have been described in single reports from cranes, but no evidence of their involvement in DVC has been demonstrated. They include *Eimeria tropicalis* and *E. grusi* from a captive Sarus Crane (Pande et al. 1970), and *Eimeria bosquei* from Lesser Sandhill Cranes (*Grus canadensis canadensis*) in New Mexico (Parker and Duszynski 1986).

EPIZOOTIOLOGY

While most eimerian protozoa confine their parasitic life cycle to the intestinal tract, the infectious agents of DVC can be found in almost any tissue in cranes. The sexual cycle can be completed in both the respiratory and the digestive tracts. A probable life history is illustrated in Figure 9.2 (Novilla et al. 1981, 1989; Novilla and Carpenter 2004).

The intestinal infection in cranes follows a typical eimerian life cycle and consists of repeated cycles of asexual reproduction (merogony resulting in merozoites) followed by sexual reproduction (gametogony resulting in oocysts). This phase of the life cycle can be completed in 12 days with a noninfectious unsporulated oocyst passing in the feces by 12 days postinfection (PI).

Orally ingested sporulated oocysts rupture when digested, releasing sporozoites that invade the intestinal epithelium. The site of invasion for *E. gruis* and *E. reichenowi* in experimentally infected Sandhill Cranes is predominantly the distal jejunum and ileum. By 6 h PI they can be found in the lamina propria (Augustine et al. 1998, 2001). It is not known whether these sporozoites actually invade or are engulfed by the epithelium. As with other species of *Eimeria*, these then undergo one or more cycles of merogony (asexual reproduction) before they become gamonts that produce macrogametocytes (female) or microgametocytes (male), which unite to form a zygote (gametogony, sexual reproduction) which matures to produce oocysts (Novilla and Carpenter 2004). The eimerians of cranes appear to uniquely differ from other eimerians in their ability to complete their life cycle in extraintestinal tissues.

Unlike the asexual stages of more typical intestinal coccidians, the asexual stages of *E. reichenowi* and *E. gruis* are taken up by mononuclear cells, including large lymphocytes or macrophages, and are transported to other tissues by way of blood or lymph where further cycles of merogony can occur and where gametogony can also occur in the lung (Figure 9.3)

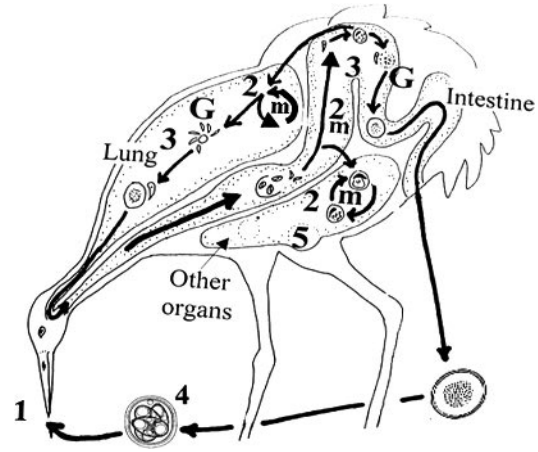


Figure 9.2. Probable life cycle of *Eimeria* sp. in cranes. (1) A sporulated oocyst is consumed and ruptures in the gut, releasing sporocysts. Sporocysts rupture to release sporozoites, which penetrate the mucosal epithelium of the distal jejunum and (2) undergo asexual merogony (m) for one or more generations. The merozoites reinfect intestinal cells or are taken up by mononuclear phagocytes and move to other organs and the lungs. (3) Merozoites initiate sexual reproduction or gametogony (G) and develop into microgametocytes (male) and macrogametocytes (female). These join to form a zygote that matures to form an oocyst. Oocysts produced in the lung are coughed up, swallowed, and pass out in feces. Oocysts produced in the intestine pass out in the feces. (4) Oocysts passed out in feces are exposed to the environment and sporulate under favorable conditions to become infective. (5) In chronic DVC, merozoites survive in granulomas in various tissues. Adapted from Novilla et al. (1981).

(Novilla et al. 1981). Why this happens in some hosts and not others is not known. Intracellular sporozoites or merozoites initiate additional cycles of merogony in a variety of tissues and form granulomas. If development occurs in the lungs, they undergo gametogony to produce oocysts. These oocysts are coughed up, swallowed, and are passed in the feces along with oocysts that are produced in the intestines. Oocysts can occur in the lung as early as 14 days PI (Novilla et al. 1989). Grossly visible nodular granulomas do not appear until 28 days PI and represent the chronic and more commonly observed phase of the disease (Figure 9.4).

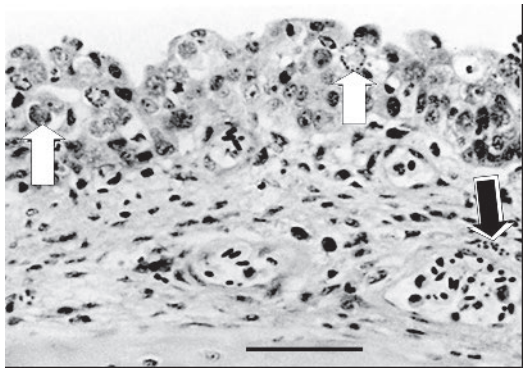


Figure 9.3. Section of pulmonary bronchus from a Sandhill Crane chick (*Grus canadensis*) killed 24 days after exposure to a pen contaminated with oocysts of *Eimeria* spp. Note gamonts (white arrows) and merozoites (black arrow) in the submucosa. Periodic acid–Schiff stain. Bar = 50 μm. Reproduced from Novilla et al. (1989), with permission of the *Journal of Wildlife Diseases*.

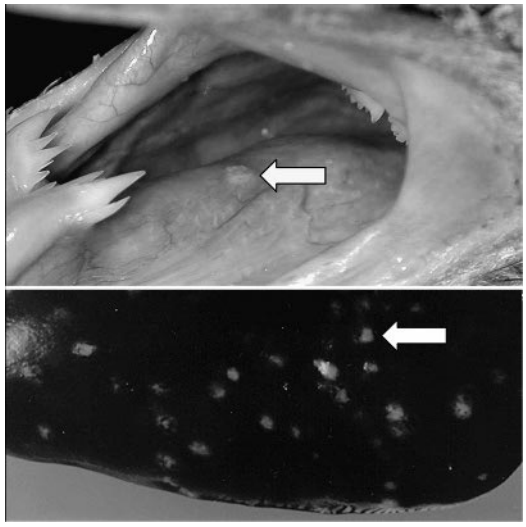


Figure 9.4. Photograph of a granuloma (arrow) in the oral cavity (top) of a wild Whooping Crane (*Grus americana*) and multiple granulomas (arrow) in the liver (bottom) of an experimentally infected Sandhill Crane (*Grus canadensis*) that died from disseminated visceral coccidiosis.

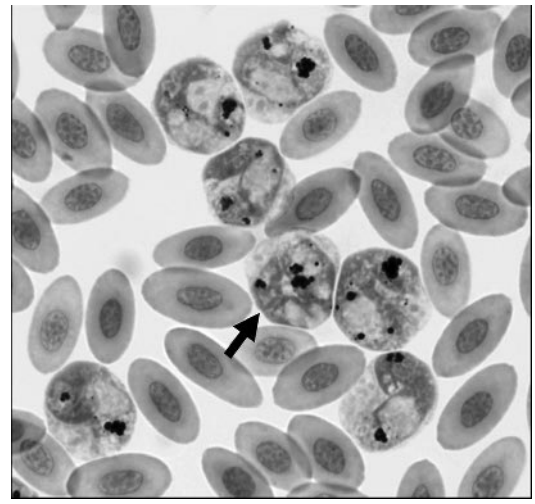


Figure 9.5. Peripheral mononuclear blood cells infected with *Eimeria* sp. (arrow) from a captive Hooded Crane (*Grus monacha*). Courtesy of Ellis Greiner, Department of Pathobiology, College of Veterinary Medicine, University of Florida, Gainesville, Florida.

In an experimental study using *E. reichenowi*, sporozoites were also noted in capillaries, possibly indicating the route of extraintestinal infection (Augustine et al. 1998, 2001). Extracellular merozoites may also be transported to other tissues in this way. Infected phagocytes and free merozoites have been found in peripheral blood 9 days PI in experimentally infected Sandhill Cranes, at death in a captive White-naped Crane (Dorrestein and Van Den Brand 2006), and 4 days prior to death and on the day of death in a captive Hooded Crane (Novilla et al. 1989; T. McNamara and E. C. Greiner, unpublished data) (Figure 9.5). These parasites have not been observed in the blood of wild cranes, possibly because of the low level and transient nature of the parasitemia (Box 1977).

Gametogony occurs in the epithelium of the digestive and respiratory tracts with gamonts and oocysts visible by 14 days PI (Novilla et al. 1981). There have been rare observations of macrogamonts in the liver of experimentally infected Sandhill Cranes (Augustine et al. 2001) and in the oral mucosa of a Wild Sandhill Crane (Parker and Duszynski 1986).

Once oocysts reach the external environment after being passed in the feces, they sporulate and become infective. Transmission occurs when a crane ingests sporulated oocysts. Cranes forage on the ground and frequently probe the soil for subterranean food. In situations where birds are crowded, particularly in

captivity, cranes may feed on numerous food items that are contaminated with feces. Cranes that are more widely distributed are less likely to be exposed to large numbers of sporulated oocysts. Oocysts can also be mechanically transmitted in or on insects. Since this parasite is specific to cranes, it appears that the only reservoir is an infected crane.

Little information is available about environmental tolerances for the oocysts of *E. gruis* and *E. reichenowi*. Eimerian oocysts of other species can generally survive in the environment for several weeks, especially under cool, moist conditions (McDougald and Reid 1997). Oocysts do not survive freezing or high temperatures (55°C). The oocysts of *E. reichenowi* are able to survive prolonged refrigeration, longer than those of *E. gruis* (Novilla et al. 1989).

Infection with species of *Eimeria* is very common in North American cranes and both *E. reichenowi* and *E. gruis* are commonly found (Table 9.1). Coinfection with both species has been reported in up to 72% of Sandhill Cranes in New Mexico (Parker and Duszynski 1986; M. G. Spalding, unpublished data).

There is some association between the presence of visceral nodules and oocyst shedding, with the highest correlation being 84% (Parker and Duszynski 1986). Age has an effect on both prevalence and intensity of infection. Juvenile cranes more commonly shed oocysts and have oral granulomas (40%) than adults (20%). Intensity of infections in juveniles when measured as number of oral granulomas was approximately twice as high (4.0 granulomas per bird) as that in adults (1.7 granulomas per bird). No difference in prevalence was noted between males and females (Carpenter et al. 1984; Parker and Duszynski 1986; M. G. Spalding, unpublished data).

Factors important in the epizootiology of the coccidia are reviewed by Fayer (1980). Species of *Eimeria* have only a single host and the oocyst is the only mechanism for parasites to infect new hosts. As a result, magnitude of oocyst production, duration of shedding of oocysts, prevalence of infected hosts, environmental conditions that affect sporulation and survival of oocysts, distribution of oocysts in the environment, and density of hosts are all factors that influence transmission. Novilla et al. (1989) proposed that low mortality of infected cranes reflects tolerance to the parasites and may represent a mechanism for the host to act as a carrier of the organism. The disseminated form of coccidiosis may make hosts better carriers by prolonging the shedding of oocysts or facilitating reemergence of the disease. In cranes, prolonged shedding may ensure contamination of both wintering and summering grounds. Since most cranes migrate out of cold weather during the winter when oocysts would be killed by cold temperatures, maintenance of the parasite in the population would require that birds either act as carriers or become reinfected on the summering grounds. Most cranes that migrate gather on staging grounds during migration, and fly together in large flocks. This behavior can also increase transmission when infected birds mix with uninfected birds while they wait for favorable conditions to migrate. Additionally, for some subspecies of migratory cranes such as the Greater Sandhill Cranes (*Grus canadensis tabida*), they comingle with resident Florida Sandhill Cranes (*Grus canadensis pratensis*) during the winter.

Severe fulminant, clinically significant, or fatal DVC has not been reported in wild cranes. Two captive-reared Mississippi Sandhill Cranes (*Grus canadensis pulla*) that were released in the field for about 5 months died from DVC, but also had other contributing concurrent diseases (N. Thomas and J. C. Franson, personal communication). In a few cases, young birds that were shedding oocysts were treated successfully in veterinary clinics, but it is not known if they had extraintestinal lesions. The relatively rare occurrence of disease in wild birds may be because they are less likely to ingest more than a few food items that are contaminated with sporulated oocysts. Alternatively, sick cranes may be killed by predators before the disease runs it course. Fatal wild infections, particularly in highly susceptible young chicks, may be underestimated because predators or scavengers may consume carcasses before they can be recovered for necropsy.

In one of the most intensive studies of the epizootiology of DVC, captive-reared and released Whooping Cranes were monitored in Florida for the presence of DVC by fecal analysis, oral examination, and necropsy. These birds had access to a coccidiostat during the time of the release and for the time that they still remained near the release pen. This treatment reduced the number of Whooping Cranes that were shedding fecal oocysts by an order of magnitude relative to wild Sandhill Cranes at the release site (Table 9.1). Treatment with coccidiostats had no effect on the prevalence of oral granulomas in the Whooping Cranes, indicating that merogony was still taking place in spite of exposure to these drugs. When Whooping Cranes older than 1 year and no longer on coccidiostat therapy were examined, there was no significant change in the prevalence of fecal oocysts, indicating that the lack of therapy may be balanced by the decrease in exposure as birds moved away from a confinement situation. At necropsy, 32 of 74 (43%) Whooping Crane carcasses that were of sufficient quality for histological examination had characteristic lesions of DVC. Oocysts and gamonts were seen only in the lungs of two birds, while most of the remaining birds had hepatic lymphohistiocytic phlebitis. Asexual stages were frequently not visible, possibly due to exposure to coccidiostats, but

tissues with grossly visible nodules were positive for *Eimeria* by polymerase chain reaction (PCR) . Lesions in two Whooping Cranes were severe enough to predispose the cranes to predation. Primary acute DVC has not been observed in wild Whooping Cranes (Forrester and Spalding 2003; M. G. Spalding, unpublished data).

CLINICAL SIGNS

Sandhill crane chicks with experimental infections of both *E. reichenowi* and *E. gruis* exhibit progressive weakness, emaciation, greenish diarrhea, and recumbency (Novilla et al. 1989). Little information is available about clinical signs in Whooping Cranes other than observations of lethargy and severe diarrhea in an adult bird (Carpenter et al. 1980).

Although there are anecdotal reports of serum enzyme changes in birds infected with species of *Eimeria* (Carpenter et al. 1979; M. G. Spalding, unpublished data), there are no good clinical markers of acute disseminated coccidiosis. Birds may die prior to shedding oocysts. Infected cells in the peripheral circulation are rare and observed occasionally in only the most severe cases. Oral granulomas and fecal oocyst shedding can demonstrate infection but tell little about the stage of disease or prognosis. Such information, however, can be very useful for monitoring and managing captive populations.

Clinical signs in wild birds are limited to the presence of oral granulomas and the shedding of oocysts. Oral granulomas are relatively common in cranes and can also be caused by several less common conditions (see Diagnosis section).

PATHOGENESIS AND PATHOLOGY

Clinical signs and mortality in Sandhill Crane chicks with experimental infections of *E. reichenowi* appear to be associated with widespread merogony, with mortality occurring at 10–11 days PI (Novilla et al. 1989). Sporozoites are first observed in the intestines by 6 h PI and subsequently appear in the liver, spleen, and lungs (Augustine et al. 1998). Merozoites in mononuclear cells are seen by 9 days PI and oocysts appear in feces by 12–14 days PI. A granulomatous inflammatory reaction to the presence of infected or ruptured cells or to necrosis of parenchymal cells results in the formation of granulomas in various tissues. The exact cause of the necrosis that presumably initiates the granulomatous response is not known. In acute cases, cranes die with mild to severe bronchointerstitial pneumonia, granulomatous inflammation of the trachea, esophagus, gastrointestinal tract, liver, heart, kidney, spleen, thymus, bursa, and many other extraintestinal tissues. In these cases, mortality often precedes gametogony.

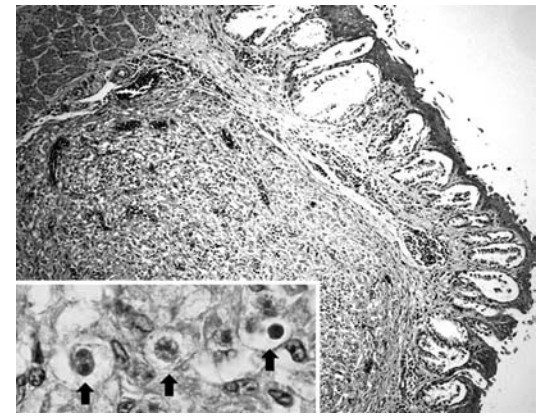


Figure 9.6. Well-circumscribed oral granuloma from a wild Sandhill Crane (*Grus canadensis*) from Florida. Note the chronic nature of the granulomatous nodule in the submucosa and the meronts (arrows) within parasitophorous vacuoles (inset).

The more chronic form of DVC, and the form seen most often in wild birds, is characterized by widely disseminated lymphohistiocytic nodules. These nodules tend to lack the more active inflammation and necrosis found in the more acute phases of the disease (Figure 9.6).

In severe acute cases of DVC in juvenile Whooping Cranes and experimentally infected Sandhill Cranes, birds may have turgid intestines containing fluid and greenish white mucoid material, hyperemic mucosa, congested consolidated lungs with airways that contain frothy fluid, enlarged mottled liver and spleen, and scattered orange-white nodules in many organs and tissues. Mild necrosis of the intestinal epithelium with epithelial cells containing coccidial oocysts and gametocytes is present. Asexual stages are present in the lamina propria and developing meronts are present within the cytoplasm of macrophages (Carpenter et al. 1980; Novilla et al. 1989).

Less severe and more chronic cases are characterized by white raised, 0.5–4.0-mm nodules scattered through a variety of tissues including oral mucosa, esophagus, feathered skin, eyelid, lung, air sac, liver, kidney, heart, spleen, adventitia of vessels, submucosa, and serosa of the intestinal tract (Figure 9.6). The most common locations for nodules are oral and esophageal mucosa, liver, and heart. In active cases, nodules in the liver are surrounded by a red rim of hemorrhage.

Microscopic lesions are well illustrated in the published literature (Carpenter et al. 1980, 1984; Novilla et al. 1981, 1989; Gardiner et al. 1988; Forrester and

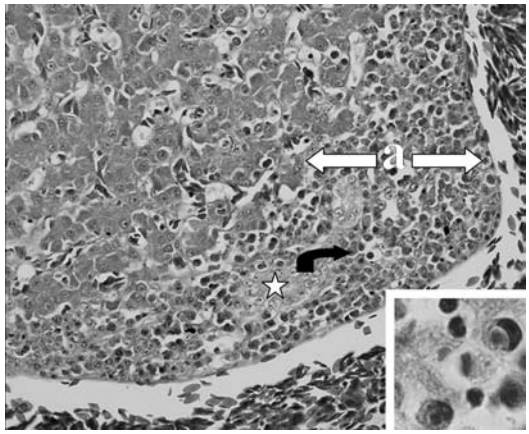


Figure 9.7. Photomicrograph of liver with mononuclear cells containing *Eimeria* sp. meronts (curved black arrow) infiltrating a vein and parenchyma (area delineated by white arrows) in a wild Sandhill Crane (*Grus canadensis*) from Florida. The white star is over a bile duct. The inset illustrates meronts within mononuclear cells in the liver at a higher magnification.

Spalding 2003) as is the ultrastructure of *Eimeria* sp. (Carpenter et al. 1979; Novilla et al. 1981, 1989; Parker and Duszynski 1986; Augustine et al. 2001; Dorrestein and Van Den Brand 2006).

Granulomatous nodules are most commonly associated with veins, especially in the liver, resulting in phlebitis that may protrude into the lumen of the vein (Figure 9.7). The granulomas consist predominantly of lymphocytes and macrophages that displace the normal tissue architecture. In liver, heart, and blood vessels, the granulomas may invade into surrounding parenchyma. In liver, the periphery of the lesion frequently consists of an area of hemorrhage, hepatocellular necrosis, and disruption of hepatic cords. Basophilic cytoplasmic inclusions displace the nuclei of mononuclear cells (Figure 9.7, inset). Depending on the age of the lesion, variable amounts of necrotic cellular debris can be present at the center. Nodular granulomas within other tissues, such as the oral mucosa, esophagus, lungs, and skin, are usually more discrete and circumscribed with little evidence of necrosis and inflammatory reaction. At the light microscope level, it is sometimes difficult to discern protozoan parasites within mononuclear cells, possibly due to low numbers of parasites.

In a survey of Greater (*Grus canadensis tabida*) and Lesser Sandhill Cranes from New Mexico, granulomatous nodules were most prevalent in the oral mucosa (67%), followed by liver (41%), small intestine (12%), heart (10%), esophagus (3%), peritoneum (2%), and mesenteries (2%). Although protozoa were not always seen within granulomas, they were present in 67% of oral granulomas, 38% of nodules in the liver, and 14% of nodules in the small intestine. The authors reported macrogamonts in the lung of only one crane (Parker and Duszynski 1986).

In experimentally infected Sandhill Cranes and captive Whooping Cranes, more generalized changes were noted such as bronchointerstitial pneumonia and parasitemia. Gamonts and oocysts were seen in bronchial epithelium (Figure 9.4). Oocysts were present in airways and the esophagus of chicks of both experimentally infected Sandhill Cranes and naturally infected captive Whooping Cranes (Carpenter et al. 1984).

Captive-reared Whooping Cranes released into the wild in Florida had lesions that differed from the typical chronic granulomas, possibly because of treatment with coccidiostats during the release process. Protozoa were frequently difficult to see at the light microscope level and almost every bird had hepatic phlebitis. It was later demonstrated by PCR that *Eimeria* was present in these lesions (see Diagnosis section). Intestinal lesions were rare in wild Sandhill and Whooping Cranes that were a part of this study (M. G. Spalding, unpublished data). When present, lesions were very mild.

DIAGNOSIS

A presumptive diagnosis of DVC is made based on finding extraintestinal nodules that contain protozoal meronts or oocysts. The disease can be confirmed if eimerian oocysts are present in fecal flotation tests and there are gamonts and oocysts in the intestinal tract and/or lungs. In the cases of fulminant disease, birds may die before oocysts are produced, making identification of the protozoan more difficult. Demonstration of *Eimeria* within the granulomas by PCR has proved useful in the cases when merozoites are not seen by light microscopy (Terrell et al. 1999). Indirect immunofluorescence microscopy with monoclonal antibodies has also been used to study the sporozoite stage (Augustine et al. 1998, 2001). Electron microscopy may also be useful for documenting infection. Shedding of *Eimeria* oocysts in feces alone is not necessarily diagnostic for the disseminated form of the disease.

Differential diagnoses for DVC based on gross lesions include avian tuberculosis and neoplasia, especially cholangiocarcinoma. Oral lesions can also be caused by bacteria, *Candida* sp., *Capillaria* sp., avian pox, and vitamin A deficiency. At the light microscope level, intracellular protozoa must be differentiated from other protozoans such as *Leucocytozoon*,

Sarcocystis, *Toxoplasma*, and *Isospora*. This is especially true when birds die prior to development of oocysts. Clusters of parasitized cells in tissues such as liver and spleen can superficially resemble lymphosarcoma.

IMMUNITY

Very little is known about how parasites interact with the host immune system in DVC. Among other species of *Eimeria*, infection with one species greatly reduces the severity of disease when birds are reinfected by the same species (Augustine 1999). Generally, reinfection with a second species of *Eimeria* results in a diminution of the reproductive potential of the second species. In chickens, T-cell-mediated immunity by intestinal lymphocytes is the predominant mechanism of protection against *Eimeria* (Lillehoj and Lillehoj 2000). There are both synergistic and immunosuppressive interactions between coccidiosis and other infectious diseases (reviewed by McDougald and Reid 1997). It is likely that naïve, young chicks are more susceptible to the severe consequences of exposure than are older birds.

Environmental contamination with oocysts and the possibility of increased transmission may explain the severity of disease in crowded conditions. However, immunosuppression related to poor diet, stress, loss of genetic diversity, and drug therapy may also be an important factor that affects severity of infection.

PUBLIC HEALTH AND DOMESTIC ANIMAL CONCERNS

Infections with species of *Eimeria* are generally species specific, although the causative agents of DVC appear to have a much wider host range among multiple species of cranes. There have been no reports of this disease in humans or domestic animals. Broiler chicks, ducks, and dogs inoculated with pooled oocysts of *E. reichenowi* and *E. gruis* are refractory to infection (Novilla et al. 1981; M. N. Novilla and J. W. Carpenter, unpublished data).

WILDLIFE POPULATION IMPACTS

The impact of DVC on wild crane populations is probably best understood for the Florida Sandhill Crane and the introduced Whooping Crane. The disease is common, but seldomly fatal in juvenile and adult cranes of both species. The role of DVC as a cause of mortality in young chicks is less well known. Mortality of wild, young crane chicks is relatively high during the first few weeks after hatching and poorly documented because of the difficulty in finding carcasses.

Mortality from DVC in wild Whooping Cranes has never been documented; however, severe lesions in birds killed by predators suggest that ill birds may be predisposed to predation. At the time of release, cranes congregate around feeders and receive pellets that contain a coccidiostat to counteract the increased risk of DVC associated with crowding. Thus, our understanding of the importance of DVC to the Florida Whooping Crane population is confounded by these unnatural factors. Fledged chicks in Florida have oral granulomas and the few that have died have disseminated granulomas, but they have not been severe enough to be considered detrimental to health.

TREATMENT AND CONTROL

Although treatment of wild cranes is not feasible, it is recommended for cranes in captive collections, breeding facilities, or for those being released into pens.

Because DVC is a significant clinical problem in young cranes, a coccidiostat should be used in the food or water. Among coccidiostats that have been tested, monensin is the only one that provides protection against experimentally induced DVC in Sandhill Cranes (Carpenter et al. 1992, 2005). Coccidian infections in individual birds have also been treated with some success with trimethoprim-sulfamethoxazole, ormetoprim-sulfa, sulfadimethoxine, or amprolium. There is increasing evidence that coccidia can develop resistance to coccidiostats. Therefore, alternating treatment between monensin and amprolium or newer generation coccidiostats may be advisable.

Captive cranes should also be monitored for oocysts, and treated as appropriate to reduce contamination of pens and potential exposure of chicks. In addition to the use of coccidiostats, reducing crowding in pens, rotation of pens, facility hygiene, quarantine, prophylactic or therapeutic treatment of new birds, and separating birds by age are integral components for controlling DVC in captivity.

When birds are being released into the wild, control may still be possible because they continue to forage at the release site. Inclusion of a coccidiostat in feed is recommended. Failure to do this may lead to development of significant lesions in cranes that are being released. For example, a Whooping Crane that escaped from a pen earlier than intended developed significant lesions of DVC, most likely because it did not have access to treated feed (M. G. Spalding, unpublished data).

Use of a coccidiostat during the vulnerable period when birds are young and crowded together may aid in development of immunity to infection, by suppressing multiplication of the parasite while allowing exposure to the parasite. Gradual withdrawal from the

coccidiostat, as birds learn to forage on natural foods, may also allow birds to develop immunity while they are still partly protected.

MANAGEMENT IMPLICATIONS

Because concentrations of the oocysts of *Eimeria* spp. in the soil may increase substantially where cranes use feeding stations, DVC is an important consideration for the management of cranes in captivity and during reintroduction into the wild. All cranes kept in close quarters and allowed to feed from soil that is contaminated with feces should receive feed treated with a coccidiostat.

The significance of DVC to wild populations is probably low based on the high morbidity and low mortality observed in subadult and adult Sandhill Cranes and reintroduced Whooping Cranes in North America. Dead chicks, however, are rarely found and since they are probably more susceptible to infection, the impact of DVC on young wild birds may be greater than realized.

Many endangered species of cranes are being intensively propagated around the world to help prevent their extinction. Disseminated visceral coccidiosis is an excellent example of how important disease can be when management activities that involve population manipulation in the wild or in captivity are undertaken.

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10
Cryptosporidium

David S. Lindsay and Byron L. Blagburn

INTRODUCTION

Members of the Genus *Cryptosporidium* belong to the protozoan Phylum Apicomplexa. They are coccidial-like parasites that develop in the microvillus border of epithelial cells in the digestive, respiratory, and urinary tracts of vertebrates. They have asexual and sexual reproductive stages in their life cycle and are excreted as fully sporulated oocysts. Molecular studies indicate that they are more closely related to the gregarine parasites of invertebrates than to the true coccidial parasites of vertebrates (Carreno et al. 1999). *Cryptosporidium* has been recognized as an increasingly important disease of commercial poultry, but has only been identified on an individual basis in wild birds, usually from fecal evaluation. There have been no recognized die-offs in wild bird populations from cryptosporidiosis.

SYNONYMS

Cryptosporidiosis.

HISTORY

The Genus *Cryptosporidium* was first described by Dr E. E. Tyzzer. He was also the first to report cryptosporidial infection in an avian species and described a species in the ceca of domestic chickens (*Gallus gallus*) (Tyzzer 1929) that was structurally similar to *Cryptosporidium parvum* from mice (*Mus musculus*). However, he did not name or describe this parasite. A species of *Cryptosporidium* was subsequently reported in the ileum of turkey poults (*Meleagris gallopavo*) suffering from enteritis (Slavin 1955). Slavin (1955) named the parasite *Cryptosporidium meleagridis* and partially described its endogenous life cycle. The first complete life cycle for an avian species was described for *Cryptosporidium baileyi*, a species that was isolated from broiler chickens (Current et al. 1986).

ETIOLOGY AND HOST RANGE

Five different species of *Cryptosporidium* have been described from birds: *Cryptosporidium meleagridis*, *C. baileyi*, *Cryptosporidium galli*, and *Cryptosporidium tyzzeri* from chickens and turkeys, and *Cryptosporidium anserinum* from domestic geese. *Cryptosporidium baileyi* is considered to be distinct from *C. meleagridis* from turkeys because it differs in oocyst structure, is only moderately infectious for turkeys, and differs in site of endogenous development in the intestine. Both molecular and morphological data have confirmed that *C. galli* Pavlasek, 1999, from chickens is a valid species (Ryan et al. 2003). This species develops in the proventriculus and has oocysts that are larger than those of *C. baileyi*. The name *Cryptosporidium blagburni* was given to oocysts from three types of finches, the Gouldian Finch (*Chloebia gouldiae*), the Red-faced Pytilia (*Pytilia hypogrammica*), and the Plum-headed Finch (*Neochmia modesta*) (Morgan et al. 2001), but it is no longer considered to be a valid species (Ryan et al. 2003). The two remaining species, *C. anserinum* from domestic geese and *C. tyzzeri* from domestic chickens, were not adequately described and are considered *nomina nuda*. Neither of the original reports gave adequate descriptions of the oocysts or provided other useful information that would support their status as new species. Ng et al. (2006) examined the genetic diversity of oocysts collected from the feces of birds from Western Australia and the Czech Republic. They found four nondescribed genotypes plus *Cryptosporidium andersoni*, a species reported from adult cattle (Lindsay et al. 2000), *Cryptosporidium muris*, *C. baileyi, C. galli*, and *C. meleagridis*, suggesting that the diversity of species of *Cryptosporidium* in avian hosts may be even higher.

The status of a species of *Cryptosporidium* that infects Northern Bobwhite (*Colinus virginianus*) is currently being investigated. The oocysts are structurally distinct from those of *C. baileyi* and resemble those of *C. meleagridis* (Lindsay et al. 1989). Unlike *C. meleagridis*, parasites from the Northern Bobwhite produce a generalized small intestinal infection that is associated with extreme morbidity and mortality in captive quail. Studies of the molecular genetics and host specificity of this parasite are needed before its status as a distinct

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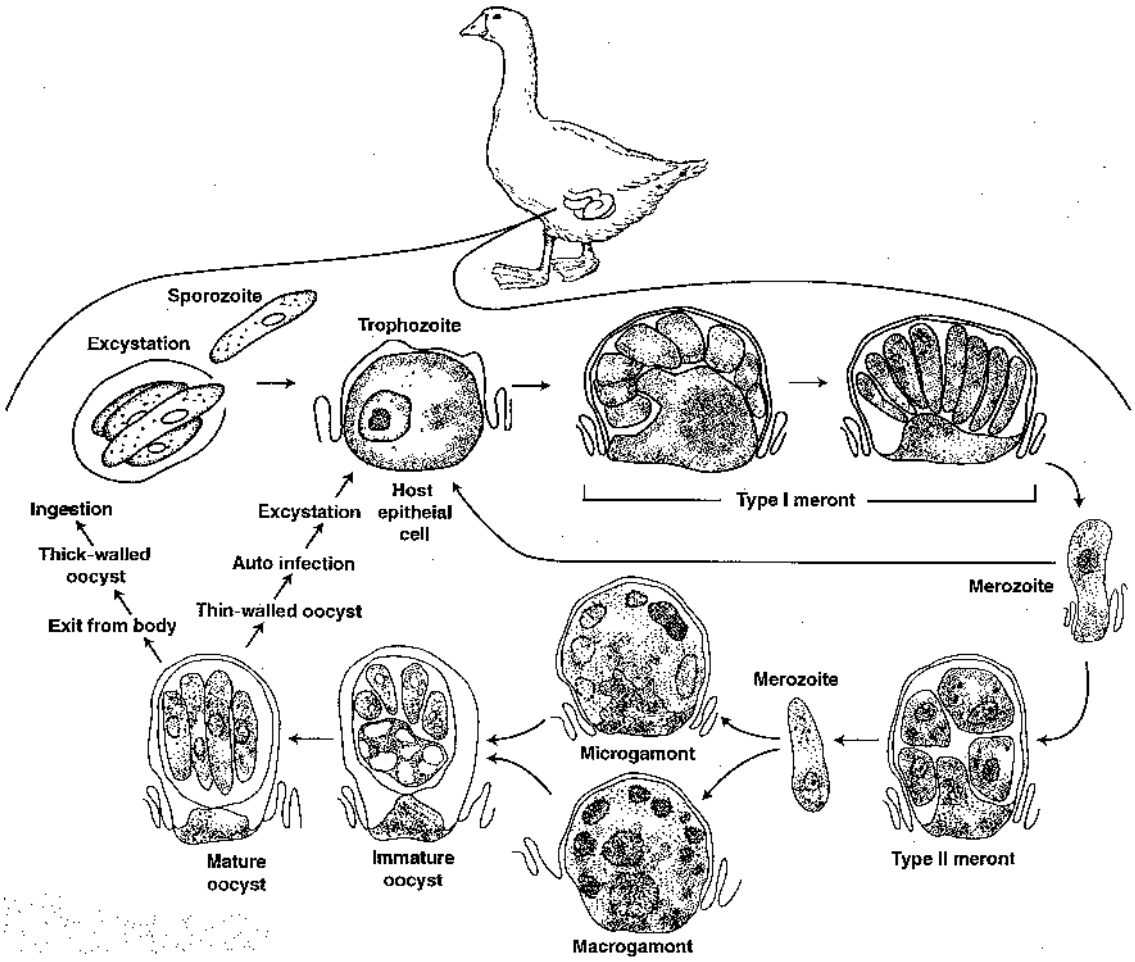


Figure 10.1. Generalized life cycle of a typical species of *Cryptosporidium* that infects birds. Adapted from Fayer et al. (1990).

species can be determined. The status of a species of *Cryptosporidium* from ostriches (*Struthio camelus*) is not fully understood. This undescribed species is not infectious for suckling mice, chickens, turkeys, or Japanese Quail (*Coturnix japonica*) (Gajadhar 1994), and molecular studies indicate that it is closely related, but distinct from, *C. baileyi* (Meireles et al. 2006).

It is clear that many genotypes and species of *Cryptosporidium* can be found in the feces of wild birds. Studies of life cycles, molecular genetics, and host specificity are needed before we can accurately determine their true number.

EPIZOOTIOLOGY

The life cycles of *C. baileyi* (Current et al. 1986) and *C. meleagridis* (Slavin 1955; Pavlasek 1994) are known in detail (Figure 10.1). Sporulated oocysts are ingested in contaminated food or water. Oocysts excyst in the digestive tract and sporozoites penetrate the microvilli of epithelial cells at locations in the intestinal tract that are specific for different species of *Cryptosporidium*. The sporozoite rounds up and becomes a trophozoite and undergoes asexual reproduction or merogony to become a multinucleated type I meront. These produce eight type I merozoites (Figure 10.2) that invade additional epithelilial cells and either initiate an additional cycle of type I merogony or develop into type II meronts. Type II meronts produce four type II merozoites that penetrate microvilli and become sexual stages. The male stages are microgamonts and they produce nonflagellated microgametes. The female stage is the macrogamont (Figure 10.3). Fertilization occurs and oocysts are produced. Two types of

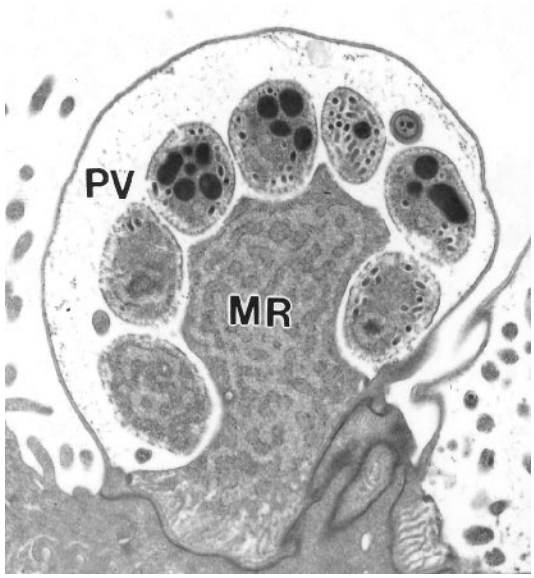


Figure 10.2. Transmission electron micrographs of *Cryptosporidium baileyi* in the respiratory tract of domestic chickens. Type II meront. Note the meront residuum (MR) and parasitophorous vacuole (PV). Bar = 1 μm. Courtesy of M. A. Cheadle.

oocysts are produced and both types sporulate endogenously (Cheadle et al. 1999). Both types contain four sporozoites, an oocyst residuum, and no sporocysts. Thin-walled oocysts are autoinfective (Figure 10.4).

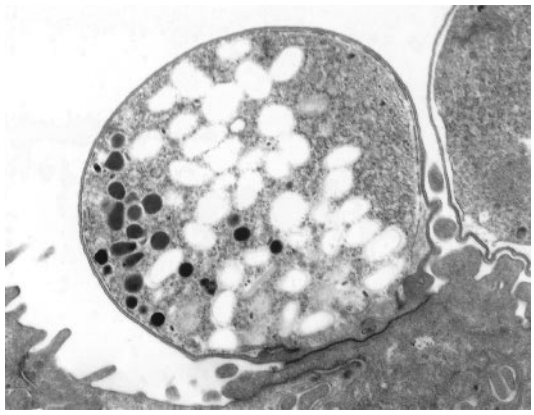


Figure 10.3. Transmission electron micrographs of *Cryptosporidium baileyi* in the respiratory tract of domestic chickens. Macrogamont. Bar = 1 μm. Courtesy of M. A. Cheadle.



Figure 10.4. Transmission electron micrographs of *Cryptosporidium baileyi* in the respiratory tract of domestic chickens. Thin-walled oocyst. Note the oocyst residuum (OR) and the thin oocyst wall (arrowhead). Bar = 1 μm. Courtesy of M. A. Cheadle.

The thick-walled oocysts (Figure 10.5) are excreted in the feces (Lindsay et al. 1986b).

If sporozoites or merozoites reach epithelial cells in the respiratory, conjunctival, or urinary tracts, then development can occur in these locations. Infections of these nonintestinal sites are not due to transport of infective stages by the blood (Lindsay et al. 1987b). Oocysts from the environment or oocysts currently being excreted probably come in direct contact with respiratory or conjunctival tissues, excyst, and initiate new infections on these surfaces.

CLINICAL SIGNS

Cryptosporidiosis in birds manifests itself as enteritis, respiratory disease, or renal disease. Usually only one condition is present in an outbreak, but combinations of the three forms have been observed. Clinical signs of intestinal cryptosporidiosis include nonbloody diarrhea. In respiratory infections, birds may suffer from rales, coughing, sneezing, and dyspnea (Hoerr et al. 1978; Ranck and Hoerr 1987; Goodwin et al. 1988a). Prolapses of the phallus and cloaca have been reported in Ostrich chicks (Penrith et al. 1994).

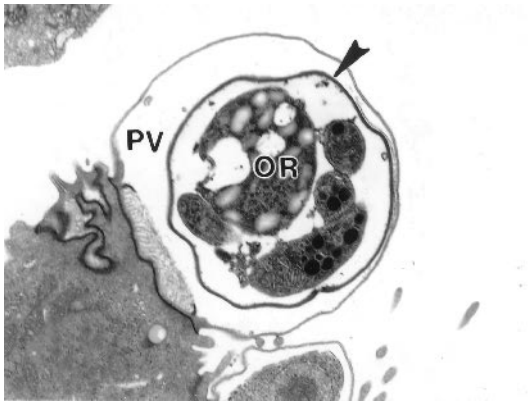


Figure 10.5. Transmission electron micrographs of *Cryptosporidium baileyi* in the respiratory tract of domestic chickens. Thick-walled oocyst. Note the oocyst residuum (OR), presence of the oocyst in a parasitophorous vacuole (PV), and thick oocyst wall (arrowhead). Bar = 1 μm. Courtesy of M. A. Cheadle.

Because cryptosporidiosis in birds manifests itself as enteritis, respiratory disease, or renal disease, it is difficult to diagnose. Many other agents can cause clinical signs that are similar to those of birds with cryptosporidiosis. There is no one clinical sign that indicates cryptosporidiosis.

PATHOLOGY

Intestinal Cryptosporidiosis

Nonbloody diarrhea is associated with intestinal cryptosporidiosis. Gross lesions are confined to the intestinal tract (Hoerr et al. 1986; Goodwin et al. 1988b). The small intestine may be distended with mucoid intestinal contents and gas. Similar lesions may be seen in the ceca. Microscopic lesions consist of villous atrophy, villous fusion, and crypt hyperplasia (Hoerr et al. 1986; Goodwin et al. 1988b). Infiltrates of lymphocytes, heterophils, macrophages, and plasma cells may be present. Cryptosporidia generally are on the distal 2/3 of the villi and are usually not seen in the ceca. Nonbloody diarrhea is also associated with cryptosporidial infection confined to the proventriculus (Blagburn et al. 1990). Lesions in the proventriculus consist of focal cuboidal metaplasia of glandular epithelial cells and deposition of amyloid in the perivascular interstitial tissues at the base of the glands (Blagburn et al. 1990)

Respiratory Cryptosporidiosis

Excess mucous may be present in the trachea and nasal cavities of birds with respiratory cryptosporidiosis. Infection of nasal tissues is associated with "swollen head syndrome" (Goodwin and Waltman 1994). Air sacculitis may be present. Microscopic lesions generally consist of hypertrophy and hyperplasia of infected epithelial surfaces (Mason and Hartley 1980). Infiltrates of macrophages, heterophils, lymphocytes, and plasma cells usually are present. Cilia generally are reduced or are absent on ciliated epithelial surfaces.

Renal Cryptosporidiosis

Infected kidneys are often pale and grossly enlarged (Abbassi et al. 1999). Urate distention may be seen in surface tubules. The epithelial cells of the collecting ducts, collecting tubules, distal convoluted tubules, and ureters are hypertrophic and hyperplastic in response to the infection (Abbassi et al. 1999; Trampel et al. 2000). Interstitial tissues may be infiltrated by lymphocytes, macrophages, heterophils, and plasma cells. Fibrotic areas may be present (Abbassi et al. 1999; Trampel et al. 2000).

DIAGNOSIS

Examination of feces for oocysts or of intestinal tissues for developmental stages is the method used for documenting *Cryptosporidium* infections in birds and other animals. The small size of the parasite makes it difficult to detect. Oocysts of *Caryospora*, *Isospora*, and *Eimeria*, and sporocysts of *Sarcocystis* can frequently be found in the feces of wild birds. The oocysts of *Caryospora*, *Isospora*, and *Eimeria* are usually excreted nonsporulated. Oocysts of *Sarcocystis* spp. sporulate in the intestinal mucosa and are excreted as sporocysts that may be confused with oocysts of *Cryptosporidium*. The sporocysts of *Sarcocystis* contain four sporozoites, are surrounded by a sporocyst wall, and are identical in structure to the oocysts of *Cryptosporidium* oocyst. They can be distinguished by size and morphology of residual material. Sporocysts of *Sarcocystis* are 10–12 by 4–7 μm and contain several granular residual granules. Oocysts of *Cryptosporidium* are 4–8 by 5–6 μm and contain a compact residual body.

Microscopic examination of fresh preparations from birds with respiratory signs is useful in obtaining a diagnosis (Ranck and Hoerr 1987; Goodwin et al. 1988a). *Cryptosporidium* oocysts can be observed in standard fecal flotations. Sheather's sugar solution is the best flotation medium. Oocysts are difficult to see

because of their small size. They will float in a plane higher than helminth ova and other protozoan cysts. Oocysts are observable using the 40× objective of a light microscope. They are often light pink to greenish in color with brightfield microscopy depending on the microscope objective lens. A central residual body is usually visible in *Cryptosporidium* oocysts in fecal flotations.

More than 10 different types of staining methods have been developed to detect *Cryptosporidium* oocysts in fecal smears with standard light microscopy (Arrowood 1997). The Ziehl-Neelsen acid-fast-staining technique is used most often and produces red-stained oocysts against a blue-green background of fecal material.

Immunodetection of *Cryptosporidium* oocysts in feces was pioneered by the human medical community. Fluorescently labeled monoclonal antibodies that bind to the oocysts of *C. parvum* are used in a direct immunofluorescent antibody test. Most species of avian *Cryptosporidium* will cross-react with the reagents in commercial immunofluorescent antibody tests that were developed for *C. parvum* (Graczyk et al. 1996a). Serum from birds infected with avian species of *Cryptosporidium* also cross-reacts with *C. parvum* in enzyme-linked immunosorbent assays, making these tests useful in detecting the presence of cryptosporidial antibodies in infected birds.

Detection of cryptosporidial stages in avian tissues is readily done using routine histological procedures and hematoxylin and eosin staining of tissues. The wide variety of locations within the avian host where *Cryptosporidium* can develop makes it important that tissues from many different anatomical sites be collected from birds at necropsy. For example, *C. baileyi* is usually found in the bursa of Fabricius and cloaca, *C. meleagridis* is usually found in the small intestine, and *C. blagburni* is usually found in the proventriculus. Failure to collect these tissues could result in false negative findings. Tissue should be taken from the head, trachea, lungs, kidneys, proventriculus, duodenum, jejunum, ileum, bursa of Fabricius, and cloaca to insure that all potential sites of development are examined.

IMMUNITY

Most studies on immunity to avian species of *Cryptosporidium* have been done with experimental infections of *C. baileyi* in chickens. Some partial immunity, as measured by oocyst excretion, may be passed in the eggs of hens infected with oocysts of *C. baileyi* (Hornok et al. 1998). It is not known if this occurs with other species of *Cryptosporidium* that infect birds. Younger chickens are more susceptible to infection with *C. baileyi* (Lindsay et al. 1988) and will produce more oocysts over a longer period of time than older birds. Severity of clinical signs, respiratory disease, and magnitude of oocyst production are also greater in younger chickens when they are inoculated with oocysts in the respiratory tract.

Birds develop serum antibodies following exposure to oocysts of *C. baileyi* (Current and Snyder 1988). Studies using chemical bursectomy and treatment with cyclosporin A indicate that cell-mediated immunity (CMI) is more important in resistance to *C. baileyi* than circulating antibody. Hatkin et al. (1993) found that bursectomy altered serum antibody production, but not CMI, as measured by the delayed-type hypersensitivity skin reaction. They also reported that chickens treated with cyclosporin were more susceptible to respiratory disease than untreated controls. Sréter et al. (1996) found that immunity to reinfection was inhibited in thymectomized chickens infected with oocysts of *C. baileyi*, further indicating the role of CMI in resistance to *Cryptosporidium*. Infection with *Eimeria* species does not induce immunity to *Cryptosporidium* in chickens; however, experimental infection with *C. parvum* does induce some cross-resistance (Sréter et al. 1997).

Infection with *C. baileyi* may inhibit development of antibodies to other infectious agents. Rhee et al. (1998a, 1998b) have demonstrated decreased antibody production to *Brucella abortus* vaccine and sheep red blood cells in chickens infected with *C. baileyi*. Secondary infections with bacteria and viruses may also be present in natural cases and add to the severity of disease.

PUBLIC HEALTH CONCERNS

Birds as a Source of *C. parvum* and *Cryptosporidium hominis*

In the early 1980s, *C. parvum* was identified as a major cause of intestinal disease in AIDS patients and it was later found in immunocompetent humans. It is now well recognized as a public health problem.

Viable oocysts of *C. parvum* can pass undamaged through the digestive system of several avian hosts (Graczyk et al. 1996b, 1997) and the oocysts of *C. parvum* have been detected in feces of wild Canada Geese (*Branta canadensis*) (Graczyk et al. 1998). These oocysts are infectious for mice, indicating that migratory waterfowl can disseminate infectious oocysts. Oocysts comprising a minimum of five different genotypes of *Cryptosporidium* can be found in the feces of Canada Geese (Jellison et al. 2004), including oocysts of *C. hominis* (Zhou et al. 2004).

Wild birds can contaminate water with fecal droppings containing oocysts of *Cryptosporidium*. Water is a major source of oocysts and outbreaks can occur in developed countries including North America, the United Kingdom, and Japan (Fayer 2004). Surveys of surface water, groundwater, estuaries, and seawater indicate that contamination of water with *Cryptosporidium* is common and not isolated to specific geographical regions (Fayer 2004). Migratory birds are the likely source of oocysts of *Cryptosporidium* because environmental samples are more likely to be positive when birds are present (Jellison et al. 2007).

C. meleagridis Infections in Humans

C. meleagridis is reported frequently from humans. In a large survey in England of human cases with diarrhea and oocysts, 0.9% of 2,414 cases were due to *C. meleagridis* (Leoni et al. 2006). Oocysts of *C. meleagridis* have been reported from humans (Xiao et al. 2001). These findings have all been based on molecular-based diagnostic tests for oocysts and not on actual recovery of oocysts from fecal material. These reports indicate that *C. meleagridis* may pose a public health risk. Most human infections with *C. meleagridis* have been in children or immunocompromised individuals, although diarrhea can also occur in individuals that have no identifiable immune deficiency (Pedraza-Díaz et al. 2001). Oocysts of *C. meleagridis* that were isolated from turkeys were infectious to immunosuppressed mice (Sréter et al. 2000), lending evidence to the potential zoonotic threat posed by this species.

C. baileyi Infections in Humans

C. baileyi is not infectious for laboratory mammals under experimental conditions (Lindsay et al. 1986a), although *C. baileyi*-like parasites have been found in human feces on a few occasions (Ditrich et al. 1991, 1993). There is one report of transmission from humans to chickens (Ditrich et al. 1993).

DOMESTIC ANIMAL HEALTH CONCERNS

Since migratory birds can carry infectious oocysts of *C. parvum* in their feces (Graczyk et al. 1998), they may serve as a source of infection for domestic livestock. Because of the confinement of domestic birds, wild birds do not appear to serve as a significant source of cryptosporidial oocysts. Wild birds may serve as a source of oocysts for free range poultry because they can contaminate the environment with oocysts.

WILDLIFE POPULATION IMPACTS

No clinical outbreaks of avian cryptosporidiosis have been reported in wild flocks of birds. European Herring Gulls (*Larus argentatus*) and Black-headed Gulls (*Larus ridibundus*) have been reported to be naturally infected with *Cryptosporidium* based on the presence of oocysts in the feces (Smith et al. 1993; Pavlasek 1993), but infections did not cause morbidity or mortality.

TREATMENT AND CONTROL

There is presently no proven treatment for avian cryptosporidiosis. Commonly used ionophorous anticoccidials are not effective (Lindsay et al. 1987a; Varga et al. 1995). The addition of the antioxidant, duokvin, to feed that contains ionophors increases their efficacy, but the combination is toxic (Varga et al. 1995). Diclazuril and toltrazuril are also not effective (Sréter et al. 1999). Enrofloxacin is marginally effective and paromomycin causes a reduction in oocyst output by 67 to 82% (Sréter et al. 2002). Paromomycin has a positive effect on weight gain.

Control methods may be useful in limiting or preventing avian cryptosporidiosis in zoos or rehabilitation centers. Several commonly used disinfectants were evaluated for the ability to kill oocysts of *C. baileyi* in an excystation assay (Sundermann et al. 1987). None of the disinfectants was effective at concentrations recommended by the manufacturers. Commercially available ammonia compounds were effective when used at 50% (v/v) concentration, with less than 5% of oocysts remaining viable. A similar concentration of commercial bleach (5.25% sodium hypochlorite) was somewhat effective with less than 15% of oocysts remaining viable.

Treatment of metal brooders, feeders, and waterers with a bleach solution followed by exposure to direct sunlight for 3 days, cleaning concrete floors, and replacing wood shavings, were effective methods for controlling an outbreak of cryptosporidiosis in young Northern Bobwhite (Hoerr et al. 1986).

Like most coccidial infections, cryptosporidiosis is a disease of confined birds or birds that are present in an area in large numbers. Any management program that decreases the numbers of birds in an area will decrease the probability of transmission of the parasite.

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11
Toxoplasma

J. P. Dubey

INTRODUCTION

Toxoplasma gondii is a protozoan parasite of worldwide distribution. It infects virtually all warm-blooded animals, including birds and humans (Dubey and Beattie 1988; Dubey 1993; Remington et al. 1995; Tenter et al. 2000; Dubey and Odening 2001). It can cause serious disease in many hosts, especially Australasian marsupials, new world monkeys, and those with immunodeficiencies. Severe toxoplasmosis has been reported in endangered Hawaiian Crows (*Corvus hawaiiensis*), canaries (*Serinus* spp.), and finches (Fringillidae).

HISTORY

Toxoplasma gondii was first discovered in a Tunisian rodent, *Ctenodactylus gundi*, by Nicolle and Manceux (1908, 1909). At about the same time, Splendore (1909) independently described a similar parasite in a laboratory rabbit in São Paulo, Brazil. The complete life cycle was not discovered until 1970 when cats were found to be the only definitive hosts (reviewed by Dubey and Beattie 1988 and Dubey 2007).

Toxoplasma-like parasites were first observed in birds by Carini (1911) in smears prepared from the liver and spleen of a Rock Pigeon (*Columba livia*) in São Paulo, Brazil. Previously, there were reports of *Toxoplasma*-like parasites in sparrows and other birds, but they were believed to be hemoprotozoans. *Toxoplasma* was subsequently reported from several species of birds (Dubey 2002), but these identifications may not have been accurate because there were no *T. gondii*-specific serologic or immunohistochemical techniques available prior to 1950. The development of the dye test by Sabin and Feldman (1948) provided a reliable serological method for evaluating and comparing presumed infections with *T. gondii* among various animal species. In the 1950s and 1960s, it became clear that there were no morphologic or serologic differences among various isolates of *T. gondii* from avian or mammalian hosts.

DISTRIBUTION AND HOST RANGE

Toxoplasma gondii has a worldwide distribution. Because there are several *T. gondii*-like parasites in birds (*Atoxoplasma*, *Isospora*, *Sarcocystis*) (Dubey 2002; Chapter 5), true hosts of *T. gondii* are only those where *T. gondii* has been demonstrated by bioassays. Reports of isolation of viable *T. gondii* from tissues of various avian species without clinical signs are summarized in Table 11.1. For bioassays, tissue homogenates from naturally exposed animals are inoculated into laboratory animals or cell cultures to observe the development of *T. gondii*; outbred Swiss albino mice are the animals most commonly used. Detection of *T. gondii* DNA is not sufficient to prove the presence of the parasite; rather, development of the organisms in cell culture or subinoculated mice is the most definitive assay. Demonstration of antibodies to *T. gondii* only indicates exposure to the organism but does not provide any information whether the host harbors live parasites. Serologic tests, in most cases, do not provide information about whether infections are recent or whether they are latent (Table 11.2).

ETIOLOGY

Toxoplasma gondii is a coccidian parasite with cats as the definitive host and warm-blooded animals as intermediate hosts. Current classifications place it in the phylum Apicomplexa Levine, 1970; class Sporozoasida Leukart, 1879; subclass Coccidiasina Leukart, 1879; order Eimeriorina Leger, 1911; and family Toxoplasmatidae Biocca, 1956. There is only one species, *T. gondii*, but genetic differences exist among isolates of *T. gondii*, even within a given host. For example, both pandemic strains and strains specific to different continents were recently identified among over 200 isolates of *T. gondii* from free-range chickens (Lehmann et al. 2006). Isolates of *T. gondii* have been classified by biological characteristics as mouse virulent or avirulent and by molecular methods into three main lineages (Types I, II, and III). Type I lineages are

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Table 11.1. Isolation of *Toxoplasma gondii* from tissues of naturally infected wild birds.

Order	Species	Scientific name	Country	*N*	Infected (%)	Reference
Anseriformes	Mallard	*Anas platyrhynchos*	Czech Republic	184	12.0	Literák et al. (1992)
			Egypt	19	5	Dubey et al. (2003)
	Common Pochard	*Aythya ferina*	Czech Republic	8	12.5	Literák et al. (1992)
	Tufted Duck	*Aythya fuligula*	Czech Republic	25	28.0	Literák et al. (1992)
	Northern Pintail	*Anas acuta*	Kazakhstan	57	1.8	Pak (1976)
	Gadwall	*Anas strepera*	Kazakhstan	93	1.1	Pak (1976)
	domestic goose	*Anser anser*	USA	1	100	Dubey et al. (2007)
	Canada Goose	*Branta canadensis*	USA	1	100	Dubey et al. (2004)
Falconiformes	Northern Goshawk	*Accipiter gentilis*	Czech Republic	10	10.0	Literák et al. (1992)
	Cooper's Hawk	*Accipiter cooperi*	USA	4	25	Lindsay et al. (1993)
	Eurasian Buzzard	*Buteo buteo*	Czech Republic	123	8.1	Literák et al. (1992)
			Kazakhstan	12	8.3	Pak (1976)
	Eurasian Kestrel	*Falco tinnunculus*	Czech Republic	1	100	Literák et al. (1992)
	American Kestrel	*Falco sparverius*	USA	3	33.3	Lindsay et al. (1993)
	Pallid Harrier	*Circus macrourus*	Kazakhstan	3	33.3	Pak (1976)
	Cinereous Vulture	*Aegypius monachus*	Kazakhstan	4	25.0	Pak (1976)
	Red-tailed Hawk	*Buteo jamaicensis*	USA	27	41.1	Lindsay et al. (1993)
	Red-shouldered Hawk	*Buteo lineatus*	USA	12	66.7	Lindsay et al. (1993)
Galliformes	Gray Partridge	*Perdix perdix*	Czech Republic	16	18.7	Literák et al. (1992)
	Ring-necked Pheasant	*Phasianus colchicus*	Czech Republic	590	2.4	Literák et al. (1992)
			Slovakia	1	100	Čatár (1974)
	Wild Turkey	*Meleagris gallopavo*	USA	16	50	Lindsay et al. (1994)
Gruiformes	Eurasian Coot	*Fulica atra*	Czech Republic	43	4.6	Literák et al. (1992)
			Kazakhstan	29	3.4	Pak (1976)
Charadriiformes	Black-headed Gull	*Larus ridibundus*	Czech Republic	61	16.4	Literák et al. (1992)
			Kazakhstan	84	1.2	Pak (1976)
	Common Tern	*Sterna hirundo*	Kazakhstan	14	7.1	Pak (1976)
			USSR	3	33.3	Pak (1970)
Columbiformes	Eurasian Collared-Dove	*Streptopelia decaocto*	Czech Republic	60	5.0	Literák et al. (1992)
			Slovakia	12	50	Čatár (1974)
	Common Wood-Pigeon	*Columba palumbus*	Czech Republic	12	8.3	Literák et al. (1992)
	Rock Pigeon	*Columba livia*	Czech Republic	606	1.0	Literák et al. (1992)
			Denmark	3	100	Siim et al. (1963)
			Slovakia	16	12.5	Čatár (1974)
			USA	1	100	Feldman and Sabin (1949)
			USA	50	2	Manwell and Drobeck (1951)

(*continues*)

Table 11.1. (*Continued*)

Order	Species	Scientific name	Country	*N*	Infected (%)	Reference
			USA	80	5	Jacobs et al. (1952)
			USA	16	6	Gibson and Eyles (1957)
	Laughing Dove	*Streptopelia senegalensis*	Kazakhstan	20	5.0	Pak (1976)
	Ruddy Ground-Dove	*Columbina talpacoti*	Panama	79	3	Frenkel et al. (1995)
Psittaciformes	Budgerigar	*Melopsittacus undulatus*	Switzerland	2	100	Galli-Valerio (1939)
Strigiformes	Ferruginous Pygmy-Owl	*Glaucidium brasilianum*	Costa Rica	1	Not given	Holst and Chinchilla (1990)
	Little Owl	*Athene noctua*	Kazakhstan	15	6.7	Pak (1976)
	Great Horned Owl	*Bubo virginianus*	USA	5	20	Lindsay et al. (1993)
	Barred Owl	*Strix varia*	USA	15	26.7	Lindsay et al. (1993)
	Tawny Owl	*Strix aluco*	France	1	100	Aubert et al. (2008)
Passeriformes	Northern Shrike	*Lanius excubitor*	Czech Republic	1	100	Literák et al. (1992)
	Yellowhammer	*Emberiza citrinella*	Czech Republic	5	20	Hejlíček et al. 1981
			Czech Republic	185	0.5	Literák et al. (1992)
	Chaffinch	*Fringilla coelebs*	Czech Republic	133	0.7	Literák et al. (1992)
			Czech Republic	152	0.7	Literák et al. (1992)
	House Sparrow	*Passer domesticus*	Costa Rica	106	16	Ruiz and Frenkel (1980)
			Czech Republic	(1,907)	0.5	Literák et al. (1992)
			Czech Republic	40	17.5	Hejlíček et al. 1981
			Kazakhstan	177	1.7	Pak (1976)
			Slovakia	5	40	Čatár (1974)
			USSR	412	0.5	Pak (1972)
	Eurasian Tree Sparrow	*Passer montanus*	Czech Republic	4	25	Hejlíček et al. 1981
			Czech Republic	316	0.6	Literák et al. (1992)
			Kazakhstan	178	0.6	Pak (1976)
	Eurasian Jay	*Garrulus glandarius*	Czech Republic	43	2.3	Literák et al. (1992)
	European Starling	*Sturnus vulgaris*	Czech Republic	69	1.4	Literák et al. (1992)
			Kazakhstan	430	0.5	Pak (1976)
	Palm Tanager	*Thraupis palmarum*	Panama	3	33.3	Frenkel et al. (1995)
	Eurasian Blackbird	*Turdus merula*	Czech Republic	54	1.9	Literák et al. (1992)
			Slovakia	4	25	Čatár (1974)

(*continues*)

Mistle Thrush	*Turdus viscivorus*	Slovakia	1	100	Čatár (1974)
Song Thrush	*Turdus philomelos*	Slovakia	7	71.4	Čatár (1974)
European Robin	*Erithacus rubecula*	Slovakia	8	37.5	Čatár (1974)
Great Tit	*Parus major*	Czech Republic	215	1.4	Literák et al. (1992)
		Slovakia	5	40	Čatár (1974)
Eurasian Nuthatch	*Sitta europaea*	Slovakia	6	33	Čatár (1974)
Eurasian Treecreeper	*Certhia familiaris*	Slovakia	1	100	Čatár (1974)
European Greenfinch	*Carduelis chloris*	Slovakia	1	100	Čatár (1974)
American Crow	*Corvus brachyrhynchos*	USA	82	1.2	Finlay and Manwell (1956)
Carrion Crow	*Corvus corone*	Kazakhstan	58	1.7	Pak (1976)
		Slovakia	4	50	Čatár (1974)
Eurasian Jackdaw	*Corvus monedula*	Czech Republic	5	20.0	Literák et al. (1992)
Rook	*Corvus frugilegus*	Czech Republic	495	18.0	Literák et al. (1992)

Source: Modified from Dubey (2002).

Table 11.2. Serologic prevalence of antibodies to *Toxoplasma gondii* in wild birds.

Order	Species	Scientific name	Country	*N*	Positive (%)	Test	Cutoff	Reference
Struthioniformes	Ostrich	*Struthio camelus*	Canada	973	2.9	MAT	1:25	Dubey et al. (2000)
Ciconiiformes	Cattle Egret	*Bubulcus ibis*	USA	40	2.5	IHAT	1:64	Burridge et al. (1979)
Anseriformes	Wood Duck	*Aix sponsa*	USA	16	6	IHAT	1:64	Burridge et al. (1979)
	Magpie Goose	*Anseranas semipalmata*	USA	11	10.8	MAT	1:25	Dubey et al. (2001)
	Barnacle Goose	*Branta leucopsis*	Norway	149	7	MAT	1:40	Prestrud et al. (2007)
Falconiformes	White-backed Vulture	*Gyps africanus*	Nigeria	240	64.8	MAT	1:25	Arene (1999)
	Turkey Vulture	*Cathartes aura*	USA	2	50	IHAT	1:64	Franti et al. (1975)
	Eurasian Buzzard	*Buteo buteo*	France	14	79	MAT	1:25	Aubert et al. (2008)
Galliformes	Wild Turkey	*Meleagris gallopavo*	USA	130	10	MAT	1:25	Quist et al. (1995)
			USA	16	71	MAT	1:25	Lindsay et al. (1994)
Gruiformes	American Coot	*Fulica americana*	USA	38	3	IHAT	1:64	Franti et al. (1976)
Charadriiformes	Ring-billed Gull	*Larus delawarensis*	USA	13	15.3	IHAT	1:64	Burridge et al. (1979)
	Laughing Gull	*Larus atricilla*	USA	33	6	IHAT	1:64	Burridge et al. (1979)
Columbiformes	Rock Pigeon	*Columba livia*	Belgium	220	3.18	MAT	1:64	Cotteleer and Famerée (1978)
			Germany	49	2	DT	1:16	Niederehe (1964)
			Italy	108	3	DT	1:50	Mandelli and Persiani (1966)
			South Africa	16	100	IHAT	1:64	Mushi et al. (2001)
			Taiwan	665	4.7	LAT	1:32	Tsai et al. (2006)
			USA	20	10	DT	1:16	Feldman and Sabin (1949)
			USA	15	6	DT	1:16	Gibson and Eyles (1957)
			USA	80	8.7	DT	1:16	Jacobs et al. (1952)
			USA	34	5.9	MAT	1:40	Kirkpatrick et al. (1990)
			USA	322	8.6	IHAT	1:16	Pendergraph (1972)
	Spotted Dove	*Streptopelia chinensis*	USA	134	8.2	DT	1:16	Wallace (1973)
	Ruddy Ground-Dove	*Columbina talpacoti*	Panama	79	12.6	MAT	1:5	Frenkel et al. (1995)
Strigiformes	Barn Owl	*Tyto alba*	USA	38	27.3	MAT	1:40	Kirkpatrick et al. (1990)
			USA	80	22.5	MAT	1:40	Kirkpatrick et al. (1990)
			USA	28	10.7	MAT	1:40	Kirkpatrick et al. (1990)
			France	18	11	MAT	1:25	Aubert et al. (2008)
	Tawny Owl	*Strix aluco*	France	12	50	MAT	1:25	Aubert el al. (2008)
Passeriformes	Plain Wren	*Thryothorus modestus*	Panama	1	100	MAT	1:5	Frenkel et al. (1995)
	Northern Mockingbird	*Mimus polyglottos*	USA	133	0.75	IHAT	1:64	Burridge et al. (1979)
	American Robin	*Turdus migratorius*	USA	23	8.6	IHAT	1:64	Franti et al. (1975)
			USA	20	5	IHAT	1:64	Franti et al. (1976)
	Clay-colored Robin	*Turdus grayi*	Panama	12	16.6	MAT	1:5	Frenkel et al. (1995)

(*continues*)

Crimson-backed Tanager	*Ramphocelus dimidiatus*	Panama	8	12.5	MAT	1:5	Frenkel et al. (1995)
Blue-gray Tanager	*Thraupis episcopus*	Panama	15	33	MAT	1:5	Frenkel et al. (1995)
Palm Tanager	*Thraupis palmarum*	Panama	3	33	MAT	1:5	Frenkel et al. (1995)
Common Grackle	*Quiscalus quiscula*	USA	27	37	IHAT	1:64	Burridge et al. (1979)
Great-tailed Grackle	*Quiscalus mexicanus*	Panama	33	33	MAT	1:5	Frenkel et al. (1995)
Red-winged Blackbird	*Agelaius phoeniceus*	USA	31	6.4	IHAT	1:64	Franti et al. (1975)
Brewer's Blackbird	*Euphagus cyanocephalus*	USA	4	25	IHAT	1:64	Franti et al. (1975)
House Sparrow	*Passer domesticus*	Czech Republic	227	12.3	IFAT	1:10	Literák et al. (1997)
Eurasian Tree Sparrow	*Passer montanus*	Czech Republic	41	4.9	IFAT	1:10	Literák et al. (1997)
European Starling	*Sturnus vulgaris*	USA	563	4.8	IHAT	1:64	Haslett and Schneider (1978)
American Crow	*Corvus brachyrhynchos*	USA	74	14	IHAT	1:64	Franti et al. (1976)

Source: Modified from Dubey (2002).

Note: It is not accurate to compare distribution, prevalence, and host distribution of *Toxoplasma gondii* based on information in this table because different serological tests were used, different cutoff values were employed, and different numbers of birds were tested. None of the serological tests have been validated for use in wild birds using isolation of *T. gondii* from naturally infected animals as the standard. Most of the information gathered in these reports is from opportunistic samples rather than planned surveys. DT, dye test; IFAT, indirect fluorescent antibody test; IHAT, indirect hemagglutination test; LAT, latex agglutination test; MAT, modified agglutination test.

considered more virulent in mice than are Types II and III. However, there are no firm data indicating whether pathogenicity in mice reflects pathogenicity in other hosts, including birds (Dubey 2006). In the only outbreak of acute toxoplasmosis in avian hosts where associated *T. gondii* strains were genotyped, isolates from five Blacked-winged Lories (*Eos cyanogenia*) were identified as Type III (Dubey et al. 2004). It is likely that all *T. gondii* isolates in nature are capable of causing illness under appropriate conditions.

The name *Toxoplasma* (toxon = arc and plasma = form) is derived from the crescent shape of the tachyzoite stage (Figure 11.1a). There are three infectious stages of *T. gondii* that are linked in a cycle: tachyzoites (in groups), bradyzoites (in tissue cysts), and sporozoites (in oocysts) (Figure 11.2).

The tachyzoite is often crescent-shaped with a pointed anterior end and a rounded posterior end and measures approximately 2 × 6 μm in size in smears (Figure 11.1a). It has a pellicle (outer covering) and several organelles including subpellicular microtubules, a mitochondrion, endoplasmic reticulum, a Golgi apparatus, an apicoplast, ribosomes, a micropore, and a well-defined nucleus (Dubey and Beattie 1988). The nucleus is usually situated toward the central area of the cell. Tachyzoites vary in shape and size, depending on the stage of division or plane of section. Dividing tachyzoites are often globular in shape

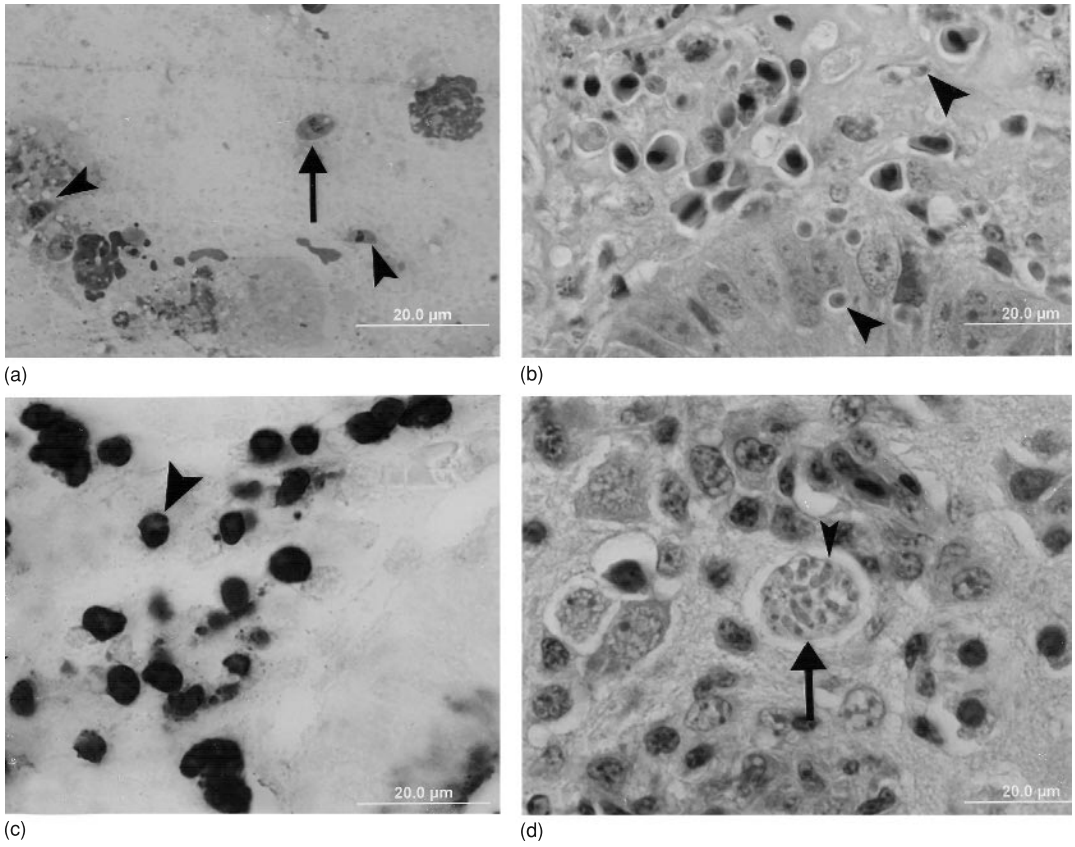


Figure 11.1. Developmental stages of *Toxoplasma gondii* from experimentally infected Budgerigars (*Melopsittacus undulatus*). (a) Tachyzoites in impression smear of intestine. Note crescent-shaped individual tachyzoites (arrowheads) and dividing tachyzoite (arrow). Giemsa stain. (b) Section of small intestine showing tachyzoites (arrowheads) and necrosis of the lamina propria cells and enterocytes. Note faint staining of tachyzoites. Hematoxylin and eosin stain. (c) Section of small intestine after immunohistochemical labeling with antibodies to *T. gondii*. Numerous tachyzoites (arrowhead) are darkly stained. Note differences in sizes of tachyzoites in parts (a)–(c). (d) Section of cerebrum showing granulomatous inflammation surrounding a tissue cyst. Note the thin cyst wall (arrow) and terminal nuclei in bradyzoites (arrowhead). From Dubey and Hamir (2002).

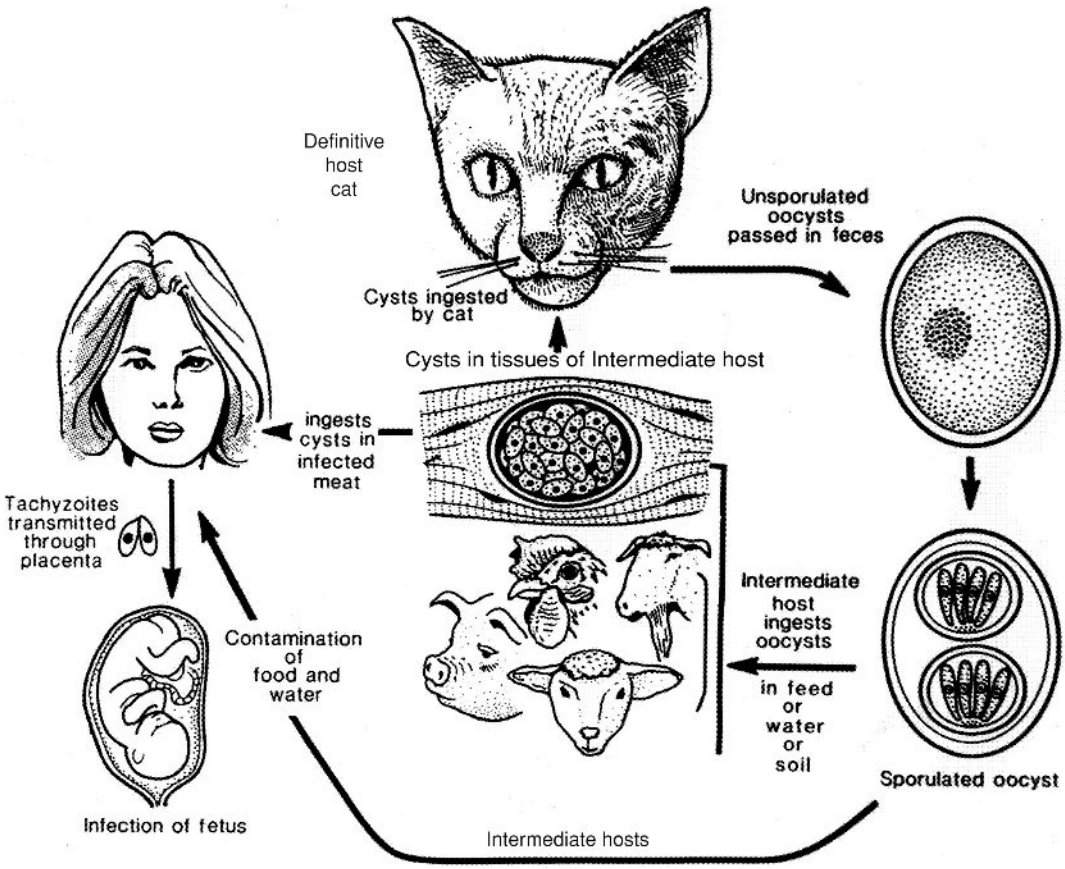


Figure 11.2. Life cycle of *Toxoplasma gondii*. From Dubey and Beattie (1988).

and bigger in size than undividing ones (Figure 11.1a). In histological sections stained with hematoxylin and eosin, tachyzoites are often globular and only about 2 μm in diameter. They are difficult to distinguish from degenerating host cells (Figure 11.1b). Tachyzoites in tissue sections labeled with *T. gondii*-specific antibodies are often larger than those in hematoxylin and eosin-stained sections (Figure 11.1c).

Bradyzoites are a slowly growing developmental stage of *T. gondii* and are enclosed in an elastic membrane. This entire structure is called a tissue cyst. Tissue cysts vary in size from 5 to 70 μm (Figure 11.1d). Although tissue cysts may develop in visceral organs, including lungs, liver, and kidneys, they are more prevalent in muscular and neural tissues, including the brain (Figure 11.1d) eye, skeletal, and cardiac muscle. Intact tissue cysts probably persist for the life of the host.

A tissue cyst may enclose hundreds of the bradyzoites that are approximately 7 × 1.5 μm in size. Bradyzoites differ structurally only slightly from tachyzoites. They have a nucleus that is situated toward the posterior end of the cell, whereas the nucleus in tachyzoites is more centrally located. The rhoptries in well-developed bradyzoites are electron-dense, whereas in tachyzoites they are electron-lucent (Dubey et al. 1998a).

EPIZOOTIOLOGY

Birds may acquire infections with *T. gondii* by ingestion of oocysts from the environment or by ingestion of tissue-inhabiting stages of the parasite in their prey. The oocyst is the environmentally resistant stage of the parasite and is excreted only by cats. In freshly passed feline feces, oocysts are unsporulated and noninfective. Unsporulated oocysts are subspherical to spherical and measure 10 × 12 μm in diameter. They sporulate and become infectious outside the cat within 1–5 days depending on aeration and temperature. Sporulated oocysts contain two ellipsoidal sporocysts. Each sporocyst contains four sporozoites that measure 2 × 6–8 μm in size.

Carnivorous birds are more likely to become infected by ingesting infected tissues from their prey and

are expected to have a high prevalence of *T. gondii* (Table 11.1). By contrast, ground-feeding avian species are most likely to become infected by ingesting oocysts from contaminated soil. Contamination of the environment by oocysts is widespread as oocysts are shed by all members of the Felidae. Domestic cats are probably the major source of environmental contamination as oocyst formation is greatest in these hosts and they are extremely common. While as few as 1% of cats may be shedding *T. gondii* oocysts at any given time, a cat may excrete millions of oocysts after ingesting only one bradyzoite or one tissue cyst. Since many tissue cysts may be present in one infected mouse or bird (Dubey 2001), numbers of excreted oocysts may be even higher. Congenital infection can also occur in cats, and congenitally infected kittens can excrete oocysts, thus providing another common source for contamination.

Sporulated oocysts survive for long periods under most environmental conditions. They can survive in moist soil, for example, for months and even years (Dubey and Beattie 1988; Dubey 2004) and can be mechanically spread by flies, cockroaches, dung beetles, and earthworms. Environmental resistance of oocysts and the enormous numbers that are shed in the feces of domestic cats assure widespread contamination (Dubey 2004).

Cats are thought to become infected by eating both birds and rodents. Hence, prevalence of infection in cats is determined by prevalence of infection in the local avian and rodent populations (Ruiz and Frenkel 1980). As environmental contamination with oocysts increases, prey animals are more likely to be infected, leading to higher rates of infection in cats.

CLINICAL SIGNS

Clinical signs of avian toxoplasmosis are nonspecific and cannot be used to make a definitive diagnosis. These signs include anorexia, depression, dull ruffled feathers, diarrhea, and dyspnoea. Unusual clinical signs of toxoplasmosis have been observed in Island Canaries (*Serinus canaria)*, including cataracts and blindness (Vickers et al. 1992; Lindsay et al. 1995; Gibbens et al. 1997; Williams et al. 2001). In ocular cases, the eyes became dull, sightless, closed, and sunken into the head, but birds were otherwise alert and continued to feed (Vickers et al. 1992; Lindsay et al. 1995; Gibbens et al. 1997; Williams et al. 2001). In one outbreak, half of the affected canaries had evidence of central nervous system involvement, including head twitch and disoriented walking in circles.

PATHOGENESIS AND PATHOLOGY

After ingestion, sporozoites from oocysts penetrate intestinal epithelial cells and multiply as tachyzoites in cells of the lamina propria. *Toxoplasma gondii* may spread to distant organs via lymphatics or blood. A host may die of acute toxoplasmosis because of necrosis of the intestine and associated lymphoid tissues before other organs are severely damaged (Figure 11.3), but more often recovers. Focal areas of necrosis may develop in many organs. By about the third week after infection, tachyzoites begin to disappear from visceral tissues and may localize as tissue cysts in neural and muscular tissues during recovery. Inflammatory lesions may persist in the central nervous system (Figure 11.1d).

Toxoplasma gondii causes tissue necrosis by active destruction of host cells; it does not produce a toxin. Tachyzoites can be found in lesions (Figure 11.3), often at the periphery of necrotic foci. In chronic lesions, tissue cysts may be found. The presence of tissue cysts in the absence of lesions indicates only persistent infection and not the clinical disease.

In naturally infected animals, lesions predominate in the liver, lungs, spleen, brain, and eyes (Figures 11.4–11.6) (Table 11.3), and occasionally in adrenal glands and bursa of Fabricius. Necrosis of hepatocytes and mononuclear cell infiltrations in periportal areas and sinusoids are characteristic of hepatic lesions (Figure 11.7). Many tachyzoites and occasionally tissue cysts are present among lesions. In lung tissue, pneumonitis is characterized by necrosis of pulmonary parenchyma and infiltrations of mononuclear cells. Tachyzoites are often present in pulmonary macrophages. Necrosis of splenic parenchyma is the primary lesion in spleen tissue. Neural lesions consist of necrosis of neuropils, gliosis, and perivascular infiltrations of mononuclear cells.

Ocular lesions in canaries are characterized by acute, severe diffuse choroiditis and retinal necrosis (Figure 11.6), panophthalmitis, optic neuritis, cataracts, and osseous replacement of the globe. Tachyzoites have been found in the choroid, retina, vitreous, and even in the lens (Vickers et al. 1992).

Many factors may determine the outcome of toxoplasmosis, including stage of the parasite ingested, dose, and host species. Oocyst-acquired infections are generally thought to be more clinically severe in birds than infection from the ingestion of infected tissues. The number of organisms ingested may not be the determining factor because hundreds of bradyzoites are contained in a tissue cyst versus only eight sporozoites in an oocyst. Currently, there is no test that can determine whether a host was infected with oocysts or tissue cysts.

The following information is derived from avian species with experimental oral infections with oocysts or tissue cysts; data derived from birds infected by parenteral routes are not comparable. Oral infection of birds with tissue cysts has been reported in only one

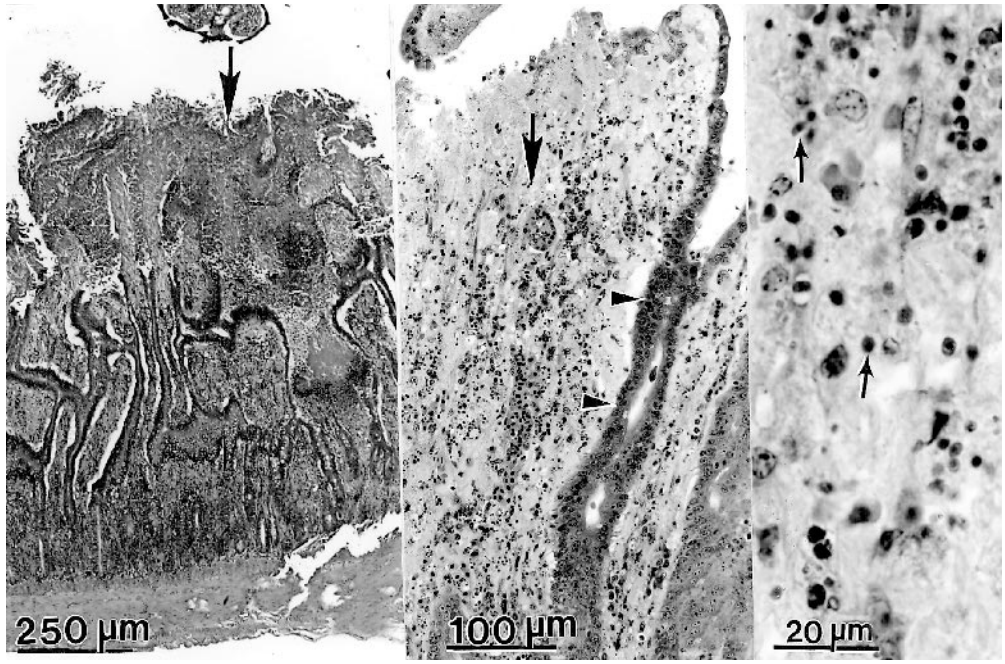


Figure 11.3. Sections of small intestines from Rock Partridges (*Alectoris graeca*) that were fed *Toxoplasma gondii* oocysts. Hematoxylin and eosin stain. (a) Desquamation (arrow) of intestinal contents into the lumen at 10 days postinfection. (b) Necrosis of lamina propria (arrow) with intact epithelium (arrowheads) at 7 days postinfection. (c) Higher magnification of part (b). Note tachyzoites (arrows). From Dubey et al. (1995).

study. Great Horned Owls (*Bubo virginianus*), Barred Owls, (*Strix varia*), and Eastern Screech-Owls (*Megascops asio*) fed tissues of rats containing many tissue cysts of the GT1 and CT1 (Type I, mouse virulent strains of *T. gondii*) became infected but did not develop clinical signs (Dubey et al. 1992).

Rock Partridges (*Alectoris graeca*) fed 10,000 oocysts of the GT1 strain (the strain fed to owls) died of peracute toxoplasmosis 4–6 days postinoculation, whereas clinical signs were less severe in Rock Partridges fed the Me-49 (Type II, mouse avirulent) strain of *T. gondii*. However, even 10 oocysts of the Me-49 strain killed 2 of 6 Rock Partridges 12 and 13 days postinoculation (Dubey et al. 1995). It is interesting that Red-legged Partridges (*Alectoris rufa*) are less susceptible than Rock Partridges. Red-legged Partridges fed 10,000 oocysts of the OV-51/95 (genotype unknown) became infected but did not develop clinical signs (Martínez-Carrasco et al. 2004, 2005, 2006). Japanese Quail (*Coturnix japonica*), Northern Bobwhite (*Colinus virginianus*), and domestic turkeys were more susceptible to toxoplasmosis than Ring-necked Pheasants (*Phasianus colchicus*) (Dubey et al. 1993a, b, 1994a, b). Although severe toxoplasmosis has been reported in some psittacine species (Table 11.3), Budgerigars (*Melopsittacus undulatus*) are relatively resistant to clinical toxoplasmosis (Dubey and Hamir 2002; Kajerová et al. 2003).

DIAGNOSIS

Serologic, histopathologic, immunohistochemical, and molecular methods can aid diagnosis, and this subject has been discussed in detail elsewhere (Dubey and Beattie 1988; Dubey 1993; Dubey and Odening 2001). Although the dye test is the most specific test for the detection of antibodies to *T. gondii* in humans, it does not work with sera from most avian species (Frenkel 1981; Dubey 2002). The latex agglutination test (LAT), indirect hemagglutination test (IHAT), and the modified agglutination test (MAT) have been evaluated in experimentally infected, domestic Wild Turkeys (*Meleagris gallopavo*), Ring-necked Pheasants, Rock Partridges, Red-legged Partridges, Northern Bobwhites, Japanese Quail, Great Horned Owls, Barred Owls, and Eastern Screech-Owls. The MAT is the most specific and sensitive test (Dubey et al. 1992, 1993a, b, 1994a, b, 1995; Martinez-Carrasco et al. 2004) and is simple,

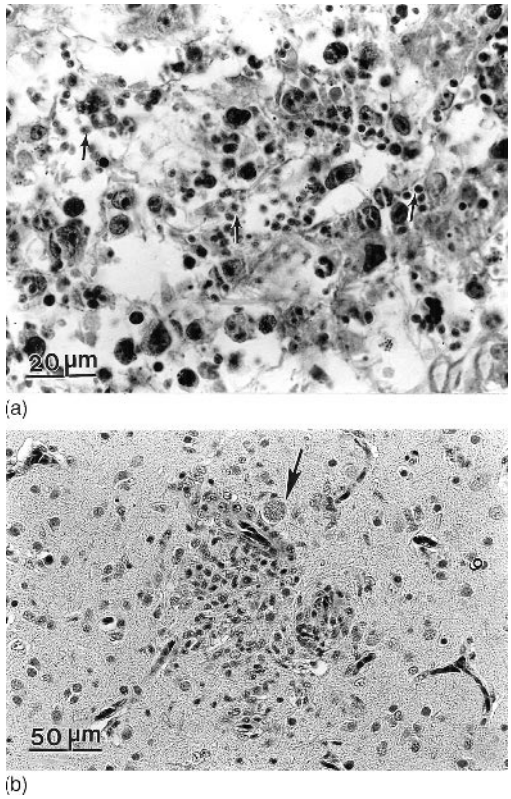


Figure 11.4. Sections of brain of birds fed *Toxoplasma gondii* oocysts. Hematoxylin and eosin stain. (a) Cerebrum of a Japanese Quail (*Coturnix japonica*) at 16 days postinfection. Note mononuclear cell infiltrate and necrosis of neutrophils associated with tachyzoites (arrows). From Dubey et al. (1994b). (b) Cerebrum of a Budgerigar (*Melopsittacus undulatus*) at 35 days postinfection. Tissue cyst (arrow) is located at the periphery of the lesion in a glial nodule. From Dubey and Hamir (2002).

reliable, does not require specific reagents, and works well with plasma (Dubey et al. 1992). A titer of 1:25 is considered indicative of infection, but titer intensity does not reflect clinical status. The LAT also works well with avian sera but titers can decline to undetectable levels (Kajerová et al. 2003).

Hematologic values are unaffected and of little use for diagnosing infection with *Toxoplasma* (Kajerova et al. 2003), although enzymes indicative of tissue necrosis (e.g., lactic dehydrogenase) may help indicate involvement of specific organ systems.

Toxoplasma gondii DNA can be detected by polymerase chain reaction methodology in fresh, frozen, and sometimes in fixed tissue by using *T. gondii*-specific primers. Fixation in formalin for a long period may denature *T. gondii* DNA. In most cases, however, diagnosis is made by histologic examination of tissues submitted for necropsy that may have already been fixed in buffered neutral 10% formalin. A preliminary diagnosis can be made by examining Giemsa-stained impression smears of affected tissues (Figure 11.1a). *T. gondii* tachyzoites are crescentic to globular in smears, depending on the stage of division. However, in histologic sections, tachyzoites are globular to oval and about half the size of those in smears (Figure 11.1b).

Toxoplasma gondii tissue cysts are often globular, have a thin cyst wall (<0.5 µm), and enclose small (5 µm), slender bradyzoites (Figure 11.1d). The bradyzoites are periodic acid Schiff positive and there are no intracystic septa (Dubey and Beattie 1988). Presence of tissue cysts in the absence of lesions generally indicates latent infection. Immunohistochemical staining with *T. gondii*-specific antibodies can aid diagnosis (Figure 11.1c). Polyclonal antibodies raised against whole parasites are often superior to monoclonal antibodies for immunohistochemical diagnosis of toxoplasmosis in tissue sections. Preservation of tissues in 10% formalin does not interfere with the immunohistochemical reaction.

Atoxoplasma and *Sarcocystis* are two parasite genera that should be considered in the differential diagnosis of avian toxoplasmosis. Proliferative stages (merozoites) of *Atoxoplasma* sp. are smaller than *T. gondii* tachyzoites, both in smears and in histologic sections (Figure 11.8).

Other parasites related to *Atoxoplasma* may also be found in birds (Baker et al. 1996; Speer et al. 1997). *Sarcocystis falcatula* and *S. falcatula*-like infections can cause generalized disease in birds, especially in passerines and psittacines (Smith et al. 1989; Hillyer et al. 1991). Some unidentified species of *Sarcocystis* can cause neural and myocardial sarcocystosis in association with development of merents in affected tissues (Gustafsson et al. 1997; Dubey et al. 2001). Neural sarcocystosis can simulate toxoplasmosis and has been reported in a Northern Goshawk (*Accipiter gentilis atricapillus*), Wild Turkeys, Eurasian Capercaillie (*Tetrao urogallus*), a Straw-necked Ibis (*Threskiornis spinicollis*), Golden Eagle (*Aquila chrysaetos*), and a Bald Eagle (*Haliaeetus leucocephalus*) (Aguilar et al. 1991; Dubey et al. 1991, 1998b, 2000, 2001; Teglas et al. 1998; Olson et al. 2007).

PUBLIC HEALTH AND DOMESTIC ANIMAL CONCERNS

Toxoplasma gondii infection is widespread among humans and its prevalence varies widely from place to

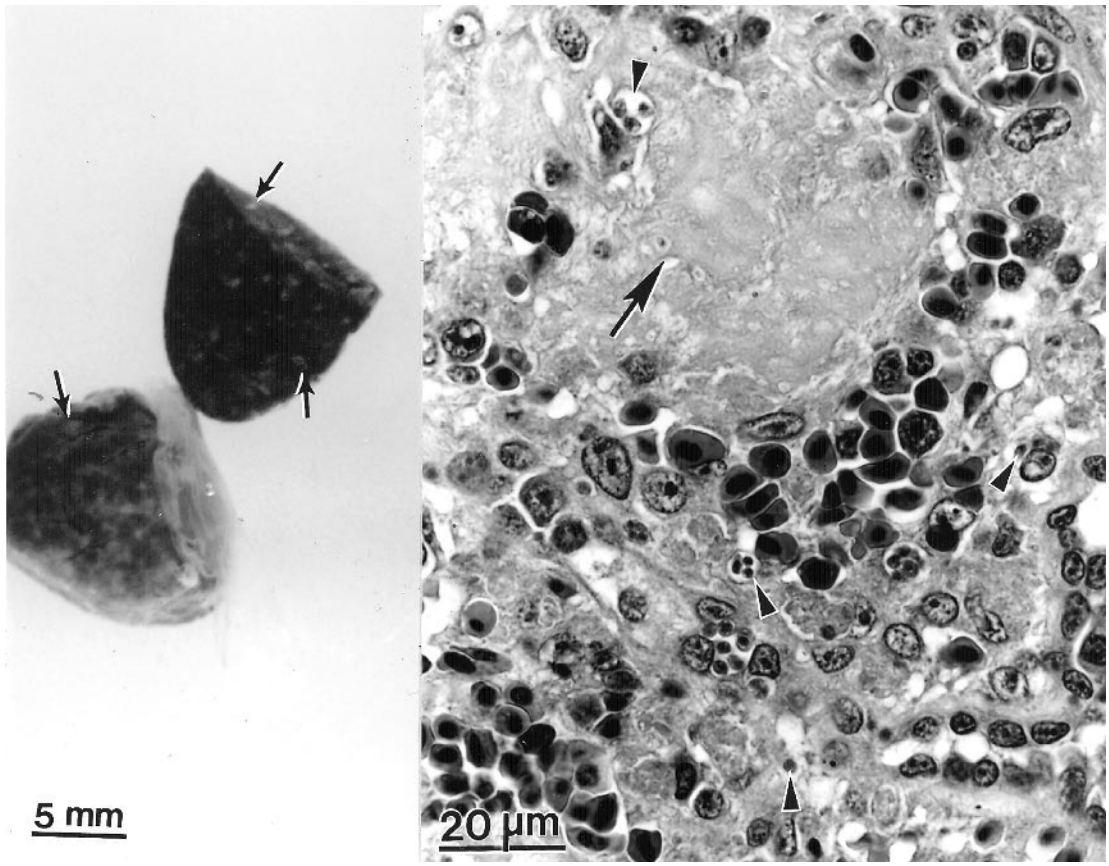


Figure 11.5. Spleen of a Rock Partridge (*Alectoris graeca*) 10 days after the bird was fed *Toxoplasma gondii* oocysts. (a) Note pale foci (arrows) on the surface and in the parenchyma. Unstained. (b) Spleen section stained with hematoxylin and eosin. Note coagulative necrosis (arrow) and tachyzoites (arrowheads). From Dubey et al. (1995).

place. Most infections in humans are asymptomatic, but at times the parasite can produce devastating disease. *Toxoplasma gondii* is capable of causing severe disease in animals other than humans (Dubey and Beattie 1988; Tenter et al. 2000). Toxoplasmosis causes great losses in sheep and goats and may cause embryonic death and resorption, fetal death and mummification, abortion, stillbirth, and neonatal death in these animals. Wild birds probably play a minor role as a source of infections for both humans and domestic animals. Most cases originate from exposure to oocysts and consumption of raw or undercooked meat.

WILDLIFE POPULATION IMPACTS

Virtually any species of bird can be an intermediate host for *T. gondii* and a source of infection for cats. There are no firm data on the impact of *T. gondii* on decline or mortality of birds in the wild, but this organism can pose a significant threat to small populations of critically endangered species. Approximately 20% of the wild population of the Hawaiian Crow died from toxoplasmosis in the late 1990s during attempts to restore this species in former habitat on the island of Hawaii (Work et al. 2000).

TREATMENT, CONTROL, AND PREVENTION

Sulfadiazine and pyrimethamine (Daraprim) are two drugs widely used for therapy of toxoplasmosis. These drugs are effective during acute stages of the disease when there is active multiplication of the parasite, but will not usually eradicate infection. Sulfadiazine and pyrimethamine have little effect on subclinical

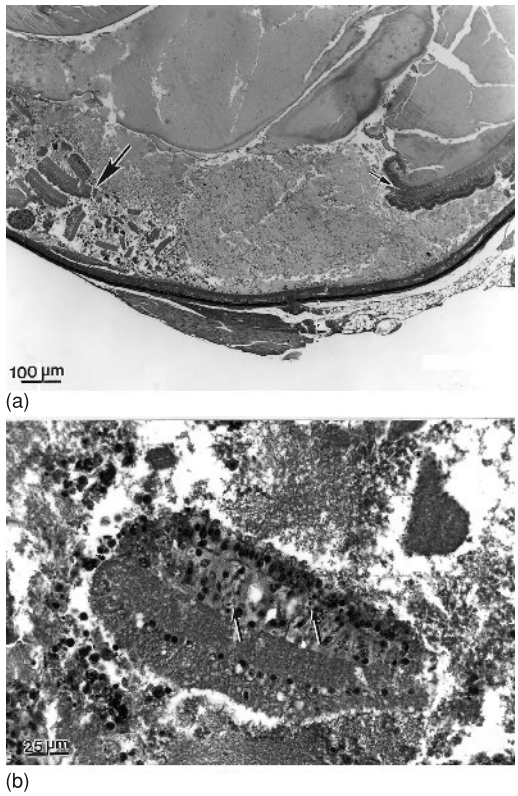


Figure 11.6. Chorioretinitis in a naturally infected Island Canary (*Serinus canaria)* with fatal toxoplasmosis. Hematoxylin and eosin stain. (a) Fragmentation (large arrow) and detachment (small arrow) of retina in vitreous humor. (b) Higher magnification of a portion of retina in the vitreous humor. Note tachyzoites (arrows). From Vickers et al. (1992).

infections, but the growth of tissue cysts in mice has been restrained with sulfonamides (Beverley 1958).

Lindsay et al. (1995) successfully treated *T. gondii*-associated blindness in canaries with sulfadiazine and trimethoprim. Diclazuril, a benzene acetonitride, was effective when used to treat toxoplasmosis in Hawaiian Crows (Work et al. 2000).

To prevent infection with *T. gondii*, aviaries should be made cat-proof. Feed should be stored in covered containers to prevent contamination with cat feces, and meat fed to birds should be cooked thoroughly to 67°C. If cooking is not practical, meat should be frozen at −12°C for at least 24 h. Freezing meat in a household freezer kills most, if not all, *T. gondii*. There is no direct transmission of *T. gondii* from birds to humans or other birds other than by eating infected meat.

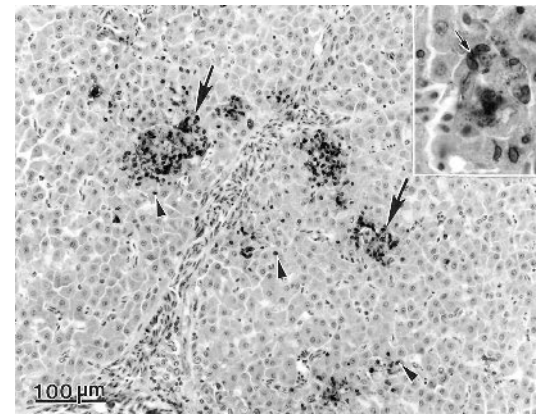


Figure 11.7. Sections of liver from a Barred Owl (*Strix varia*) naturally infected with *Toxoplasma gondii* and labeled by immunohistochemical staining with anti-*T. gondii* antibodies. Note foci of necrosis (arrows) and numerous black tachyzoites (arrowheads). Inset: Higher magnification view of tachyzoites (arrow). From Mikaelian et al. (1997).

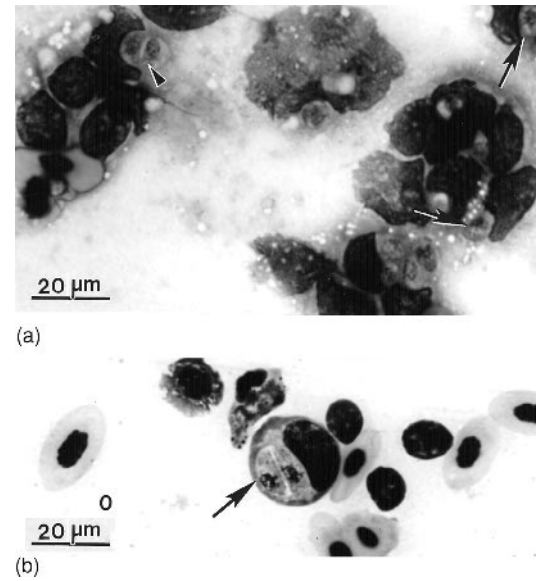


Figure 11.8. *Atoxoplasma* sp. merozoites (a) and *Toxoplasma gondii* tachyzoites (b) in impression smears of avian tissues stained with Giemsa. (a) Individual (arrow) and dividing (arrowheads) *Atoxoplasma* in spleen of a Bali Mynah (*Leucopsar rothschildi*). (b) Two *T. gondii* tachyzoites (arrow) within a mononuclear cell in the lung of a Rock Partridge (*Alectoris graeca*). From Dubey (2002).

Table 11.3. Reports of toxoplasmosis at necropsy from birds that died in captivity (C) or in the wild (W).

Order	Species	Scientific name	Country	*N*	Died	Method	Reference
Sphenisciformes	Humboldt Penguin	*Spheniscus humboldti*	USA	4	C	H	Ratcliffe and Worth (1951)
	Magellanic Penguin	*Spheniscus magellanicus*	USA	2	C		Ratcliffe and Worth (1951)
	Jackass Penguin	*Spheniscus demersus*	USA	1	C		Ratcliffe and Worth (1951)
	Little Penguin	*Eudyptula minor*	Australia	1	C	H, IHC	Mason et al. (1991)
Pelecaniformes	Red-footed Booby	*Sula sula*	USA	1	C	H, IHC	Work et al. (2002)
Anseriformes	Magpie Goose	*Anseranus semipalmata*		2	C	H, IHC	Dubey et al. (2001)
	Hawaiian Goose	*Branta sandvicensis*	USA	2	C	H, IHC	Work et al. (2002)
	Mallard	*Anas platyrhynchos*	Argentina	Many	C	H	Boehringer et al. (1962)
Galliformes	Wild Turkey	*Meleagris gallopavo*	USA	1	W	H, TEM	Howerth and Rodenroth (1985)
			USA	1	W	H, IHC	Quist et al. (1995)
	Gray Partridge	*Perdix perdix*	Czech Republic	3	C	H	Pokorny (1955)
	Erckel's Francolin	*Francolinus erckelii*	USA	1	W	H, IHC	Work et al. (2002)
Columbiformes	Western Crowned-Pigeon	*Goura cristata*	Belgium	1	C	H, I	Kageruka and Willaert (1971)
			Belgium	1	C	H	Tackaert-Henry and Kageruka (1977)
			The Netherlands	3	C	H, I	Poelma and Zwart (1972)
	Victoria Crowned-Pigeon	*Goura victoria*	USA	1	C	H	Ratcliffe and Worth (1951)
			Belgium	4	C	H	Tackaert-Henry and Kageruka (1977)
			The Netherlands	2	C	H, I	Poelma and Zwart (1972)
	Pied Imperial-Pigeon	*Ducula bicolor*	The Netherlands	1	C	H, I	Poelma and Zwart (1972)
	Southern Crowned-Pigeon	*Goura scheepmakeri*	The Netherlands	4	C	H, I	Poelma and Zwart (1972)
	Torresian Imperial-Pigeon	*Ducula spilorrhoa*	Australia	3	C	H, IHC	Hartley and Dubey (1991)
	Wonga Pigeon	*Leucosarcia melanoleuca*	Australia	1	C	H, IHC	Hartley and Dubey (1991)
	Luzon Bleeding-heart	*Gallicolumba luzonica*	The Netherlands	1		H, I	Poelma and Zwart (1972)
Strigiformes	Barred Owl	*Strix varia*	Canada	1	C	H, IHC	Mikaelian et al. (1997)
Psittaciformes	Regent Parrot	*Polytelis anthopeplus*	Australia	1	C	H, IHC	Hartley and Dubey (1991)
	Superb Parrot	*Polytelis swainsonii*	Australia	1	C	H, IHC	Hartley and Dubey (1991)
	Red Lorry	*Eos bornea*	USA	1	W	H, IHC, TEM	Howerth et al. (1991)
			USA	5	C	H, I	Howerth et al. (1991)

(*continues*)

Table 11.3. (*Continued*)

Order	Species	Scientific name	Country	*N*	Died	Method	Reference
	Black-winged Lorry	*Eos cyanogenia*					Dubey et al. (2004)
	Rainbow Lorikeet	*Trichoglossus haematodus moluccanus*	The Netherlands	1	C	H, I	Poelma and Zwart (1972)
	Crimson Rosella	*Platycercus elegans*	Australia	1	C	H, IHC	Hartley and Dubey (1991)
	Red-fronted Parakeet	*Cyanoramphus novaezelandiae*	Australia	3	C	H, IHC	Hartley et al. (2008)
	Yellow-fronted Parakeet	*Cyanoramphus auriceps*	Australia	3	C	H, IHC	Hartley et al. (2008)
Piciformes	Red-bellied Woodpecker	*Melanerpes carolinus*	USA	1	C	H, IHC	Gerhold and Yabsley (2007)
Passeriformes	Hawaiian Crow	*Corvus hawaiiensis*	USA	5		A, H, I, IHC	Work et al. (2000)
	Satin Bowerbird	*Ptilonorhynchus violaceus*	Australia	1	C	H, IHC	Hartley and Dubey (1991)
	Regent Bowerbird	*Sericulus chrysocephalus*	Australia	1	C	H, IHC	Hartley and Dubey (1991)
	Red-whiskered Bulbul	*Pycnonotus jocosus*	Australia	1	C	H, IHC	Hartley and Dubey (1991)
	Island Canary	*Serinus canaria*	Australia	12/24*	C	H, I, IHC	Vickers et al. (1992)
			Australia	2/40*	C	H	Lindsay et al. (1995)
			United Kingdom	2/44*	C	H	Gibbens et al. (1997)
			United States	2/9*	C	H	Williams et al. (2001)

Note: Methods of diagnosis include antibodies to *Toxoplasma gondii* in serum (A), histology (H), immunohistochemical staining with anti-*T. gondii* antibodies (IHC), isolation in mice (I), and transmission electron microscopy (TEM). In most cases, birds died from pneumonia, hepatitis, splenitis, encephalitis, and/or ophthalmitis. In addition to data compiled in this table, clinical toxoplasmosis has been reported in unspecified species of pigeons as well as Rock Pigeons (*Columba livia*) from Brazil (Carini, 1911; Pires and Dos Santos 1934; Reis and Nóbrega 1936; Springer 1942), Democratic Republic of Congo (Wiktor 1950), Ecuador (Rodriguez 1954), Italy (de Mello 1915; Alosi and Iannuzzi 1966), Mexico (Paasch 1983), Panama (Johnson 1943), Scandinavia (Siim et al. 1963), Venezuela (Vogelsang and Gallo 1954), Uruguay (Cassamagnaghi et al. 1952, 1977), and from an unspecified source (Hubbard et al. 1986). Toxoplasmosis has also been reported from canaries and finches from Uruguay (Cassamagnaghi et al. 1952, 1977) and Italy (Parenti et al. 1986) and from Budgerigars (*Melopsittacus undulatus*) from Switzerland (Galli-Valerio 1939). The number of birds affected and the methods of diagnosis were not detailed in several of these reports.

*Fraction of total that were examined by histopathology.

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Section III: Helminths

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12
Trematodes

Jane E. Huffman

INTRODUCTION

Trematodes are flat, leaflike parasitic helminthes that are classified in the phylum Platyhelminthes—a diverse group of free-living and parasitic worms that also includes the cestodes (Chapter 14). Current taxonomy places trematodes into two classes—the monogenetic trematodes that are primarily ectoparasites of fish and possess a posterior attachment organ or haptor armed with hooks and the digenetic trematodes that are common endoparasites of a variety of vertebrate hosts and have muscular oral and/or ventral suckers.

The digenetic trematodes have life cycles with both sexual and asexual phases of reproduction—the former in their vertebrate definitive hosts and the latter in molluscan intermediate hosts. With the exception of the schistosomes (Chapter 13), trematodes are hermaphroditic and generally have one or two large, sometimes branched, testes, a comparatively small ovary, an often long and looping uterus, and a single common genital pore. They typically have a bifurcated intestine and blind ceca that exit the body through an anus or via an excretory vesicle. Unlike nematodes, and similar to both cestodes and acanthocephalans, the tegument lacks a cuticle (Marquardt et al. 2000).

A wide range of digenetic trematodes occur in wild birds. However, the majority of these species are not associated with significant disease. Trematodes that cause large-scale epizootics in birds include *Sphaeridiotrema globulus* (Psilostomatidae), *Cyathocotyle bushiensis* (Cyclocoelidae), and *Leyogonimus polyoon* (Lecithodenriidae). Epizootics with these three parasites usually occur in the spring and fall, often when large numbers of birds stop over at particular areas during migration. The success of these parasites depends on the presence of first, second, and definitive hosts in the environment. Large late-summer mortalities of waterfowl attributable to these trematodes have been reported since the 1960s, when thousands of ducks were apparently killed by mixed infections of the digeneans *C. bushiensis* and *S. globulus* along the St. Lawrence River south of Quebec, Canada.

SYNONYMS

Flukes, flatworms.

HISTORY

Much of the early history of the digenetic trematodes of birds was summarized by Dawes (1946). It seems likely that the larger flukes that are parasitic in mammals, for example, *Fasciola*, have been recognized since at least the fourteenth century. Their occurrence in birds was noted by Goeze (1782, cited in Dawes 1946) who published a work on the natural history of parasitic worms. Zeder in 1800 developed a systematic classification of parasitic worms and described *Cyclocoelum mutabile*, a trematode of the Black Scoter (*Melanitta nigra*) and Common Moorhen (*Gallinula chloropus*). Rudolphi broadened the foundations of our knowledge of parasitic worms with two works in 1810 and 1819, and coined the term "trematode" to replace the term "sucking worm." Rudolphi described a number of trematode species from British birds. Jagerskiold published a series of papers between 1896 and 1908 on the trematode parasites of birds (Dawes 1946).

In the US, the first report of an epizootic in waterfowl was reported by Price (1934) in Lesser Scaup (*Aythya affinis*) in the Potomac River in Washington, DC. The causative agent was *S. globulus*.

ETIOLOGY

McDonald (1981) listed 536 species of digenetic trematodes from 125 genera and 27 families of birds. A detailed treatment of this diverse group of parasites is well beyond the scope of this chapter, although they all have a number of similarities in life cycles, morphology, and development within the gut and other tissues of their definitive hosts. The most significant trematodes of birds occur in 6 of the 10 orders of digenetic trematodes listed by Brooks and McLennan (1993). These trematodes are distinguished by morphological features of both adult and immature forms. Details about their morphology and taxonomy can be found

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in Dawes (1946), Yamaguti (1971), and McDonald (1981).

Ventral suckers are generally about the same size as the oral suckers, and located centrally on the anterior, ventral surface. Often, the ventral suckers are quite close behind the oral sucker. The location, number, and morphology of the suckers are used to separate members of this group. A distome is a digenetic trematode with an anterior oral sucker and the posterior sucker located on the ventral surface and a monostome possesses a single sucker, oral or ventral, rather than both. A holostome is a type of adult digenetic trematode having a portion of the ventral surface modified as a complex adhesive organ.

HOST RANGE AND DISTRIBUTION

The geographical distribution of trematodes is influenced by environmental conditions that affect the distribution of their intermediate hosts. These conditions include biotic variables such as vegetation cover and abiotic variables of the lentic environment such as size, average depth, salinity, and characteristics of the sediments. For example, a trematode that uses a particular species of mollusk as an intermediate host may only occur where that mollusk is found. The hosts of *Uvulifer ambloplitis*, a parasite of kingfishers (*Megaceryle* spp.), include snails, fish, and birds. The distribution of this trematode is a combination of the ranges of all three hosts. Nonetheless, the distribution of a particular trematode can span large areas, particularly if definitive and intermediate host species live in a broad range of habitats or if the definitive host migrates over vast regions.

Some species of trematodes occur with great frequency in a large variety of avian hosts, others seem to be rarer, even specific for one or more hosts. Monostomes such as *Notocotylus attenuatus* and *Catatropis verrucosa* occur in a large variety of hosts. Holostomes such as *Strigea* spp. are common parasites of birds of prey, ducks, and gulls. *Prosthogonimus ovatus* likewise occurs in numerous birds, more than half of which are passerines of wading birds. The species *P. cuneatus* shows an even greater preference for passerines. At the other extreme, the two most common echinostomes—*Echinostoma revolutum* and *Hypoderaeum conoideum*—are almost entirely confined to ducks and their close relatives.

Table 12.1 summarizes the distribution and host range of *L. polyoon*, *C. bushiensis*, and *S. globulus*, the cause of current epizootics in the US. The exotic Faucet Snail (*Bithynia tentaculata*), originally native to Europe, can serve as the intermediate host, and the American Coot (*Fulica americana*) can serve as the definitive host for all three parasites. In Europe, the only definitive hosts for *L. polyoon* are Eurasian Coots (*Fulica atra*) and Common Moorhens; in the US, the only known host currently is the American Coot. *S. globulus* and *C. bushiensis* share similar waterfowl hosts in both Europe and the US. Coinfections with *S. globulus* and *C. bushiensis* have been reported (Hoeve and Scott 1988). All three parasites can produce mortality in the avian host within 3–8 days after infection.

Annual migrations can disseminate avian trematodes and of special importance is the cross migration and movement of birds following the breeding season and preceding the beginning of fall migration. This can allow for widespread dissemination of these parasites over the breeding grounds as long as the intermediate hosts are present.

EPIZOOTIOLOGY

Digenetic trematodes produce eggs in their definitive hosts which pass with feces either into water or onto land. The egg of most forms is oval and has a lid-like hatch on one end called an operculum. When eggs reach freshwater, the operculum opens and a ciliated free-swimming larva called a miracidium swims out. The miracidium will then use chemotactic cues to find a suitable intermediate host, which is usually a snail. The miracidium penetrates the snail, loses its cilia, and develops into a sporocyst. Sporocycsts reproduce asexually to form either more sporocysts or a number of rediae. Rediae reproduce asexually to form more rediae or tailed forms called cercariae. The cercariae emerge from the snail and penetrate a second intermediate host (either a mollusk, amphibian, or fish), the final host, or encyst on vegetation where they transform into metacercariae. Adult worms develop from metacercariae when they are ingested by a definitive host (Figure 12.1) (Marquardt et al. 2000).

Trematodes of birds generally develop in specific locations in the body of the host. The most common site is the intestine. Both holostomes and echinostomes show a preference for the lower end of the intestine. Other sites include the bursa of Fabricii (Prosthogonimus) and the cloaca (Leucochloridium). Most monostomes inhabit the air sacs and *Collyriculum faba* forms cysts under the skin. Species of Opisthorchiidae and Dicrocoeliidae infect the liver (Table 12.2).

A number of trematodes develop in very specific and unusual sites. Both *Lyperosomum longicauda*, a common fluke of Carrion Crows (*Corvus corone*) in Europe, and *Athesmia heterolecithodes* from the Ruffed Grouse (*Bonasa umbellus*) develop specifically in the liver. *Clinostomum complanatum* is found in the buccal cavity and the upper ends of the esophagus and trachea. *Renicola pinguis* (Troglotrematidae) and *Eucotyle nephritica* (Eucotylidae) inhabit the kidney. Some birds are exceptional in harboring two or more species of trematodes that

Table 12.1. Summary of host range and distribution information for *Sphaeridiotrema globulus* (family Psilostomatidae), *Cyathocotyle bushiensis* (family Cyathocotylidae), and *Leyogonimus polyoon* (family Lecithodendriidae).

Trematode*	Intermediate hosts	Avian hosts (USA)	Location	Avian hosts (Europe)
Sphaeridiotrema globulus	*Bithynia tentaculata*†	American Coot (*Fulica americana*)	Potomac River	Tufted Duck (*Aythya fuligula*)
	Elimia virginica	Mute Swan (*Cygnus olor*)	NJ, NY, WI, OR, USA	Greater Scaup (*Aythya marila*)
	Fluminicola virens	Tundra Swan (*Cygnus columbianus*)		Northern Pintail (*Anas acuta*)
	Oxytrema silicula	Lesser Scaup (*Aythya affinis*)		Long-tailed Duck (*Clangula hyemalis*)
		Canvasback (*Aythya valisineria*)		Razorbill (*Alca torda*)
		Common Goldeneye (*Bucephala clangula*)		Common Merganser (*Mergus merganser*)
		Ruddy Duck (*Oxyura jamaicensis*)		
		Long-tailed Duck (*Clangula hyemalis*)		Whooper Swan (*Cygnus cygnus*)
				Red-breasted Merganser (*Mergus serrator*)
Cyathocotyle bushiensis	*Bithynia tentaculata*	American Coot (*Fulica americana*)	St. Lawrence River, Canada	Long-tailed Duck (*Clangula hyemalis*)
		American Black Duck (*Anas rubripes*)	Great Lakes Basin, WI, USA	European Shag (*Phalacrocorax aristotelis*)
		Blue-winged Teal (*Anas discors*)		
		Green-winged Teal (*Anas carolinensis*)		
Leyogonimus polyoon	*Bithynia tentaculata*	American Coot (*Fulica americana*)	WI	Eurasian Coot (*Fulica atra*)
				Common Moorhen (*Gallinula chloropus chloropus*)

Note: All three trematodes were introduced from Europe. Epizootics caused by *Sphaeridiotrema* were documented in 1928, while those caused by *Cyathocotyle bushiensis* occurred in the 1960s. *Leyogonimus polyoon* was identified as a cause of eipizootics in Wisconsin in 1996. These trematodes occur in the lower intestine (*Sphaeridiotrema*, *Cyathocotyle*) or upper and middle intestines (*Leyogonimus*) of their hosts.

*References for avian hosts can be found in Dawes (1946), Gower (1939), and Yamaguti (1971).

†*Bithynia tentaculata* was introduced from Europe into Lake Michigan in the 1970s.

belong to the same genus. The Black Scoter has been reported to be parasitized by 7 species of *Gymnophallus* (Microphallidae) (Dawes 1946). Skrjabin (1926) described an example of a bird parasitized by 17 species of helminths.

The larval stages of trematodes are influenced by density-independent factors such as temperature and moisture. Their populations may fluctuate dramatically over time as a result of environmental changes, possibly leading to local extinctions (Bush et al. 2001). Trematodes that use hosts that are also strongly influenced by density-independent factors and parasites living at the edges of their geographic ranges may also be strongly influenced by density-independent factors (Bush et al. 2001). In contrast, density-dependent effects primarily occur within vertebrate and intermediate hosts and can reduce trematode survival or fecundity and ultimately regulate parasite abundance. Two of the factors most likely to be important in exerting density-dependent effects are host immune responses

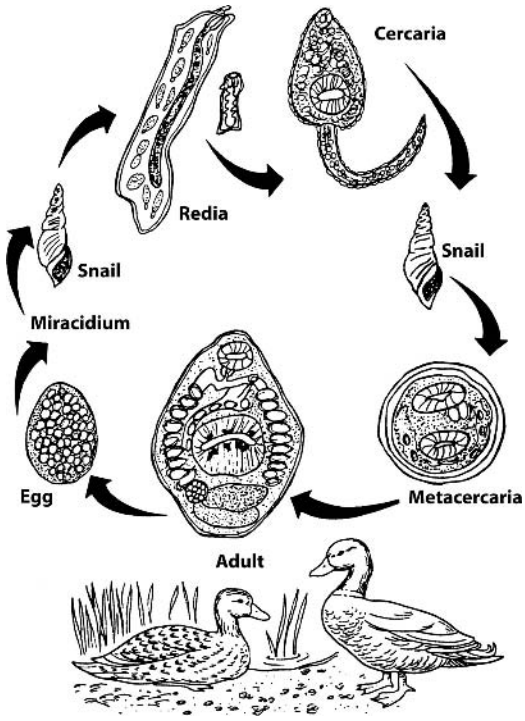


Figure 12.1. Typical life cycle of a digenetic trematode. Eggs are released by adult worms and pass with feces of their avian hosts either into water or onto land. When eggs reach freshwater, a ciliated free-swimming miracidium is released which penetrates a snail, loses its cilia, and develops into a sporocyst. Sporocycsts reproduce asexually to form either more sporocysts or a number of rediae. Rediae reproduce asexually to form more rediae or tailed forms called cercariae. The cercariae emerge from the snail and penetrate a second intermediate host (either a mollusk, amphibian, or fish), the final host, or encyst on vegetation where they transform into metacercariae. Adult worms develop from metacercariae when they are ingested by a suitable definitive host.

and competitive interactions either within or between trematode species. Both these factors may lead to host mortality as trematode density increases, with ultimate effects on overall abundance of the parasites (Bush et al. 2001).

Pathogenicity of a trematode may differ among species of birds as well as different populations of the same species. The effects of trematodes on individual birds are well documented in the literature. The identification of multiple concurrent disease problems in individual birds in poor body condition suggests that gradual debilitation of a wild bird from a specific disease may predispose it to several other potential pathogens within its environment. A statistically significant association between poor body condition and high numbers of intestinal trematodes has been reported in Common Loons (*Gavia immer*) from maritime Canada (Daoust et al. 1998). Factors involved in susceptibility of a host population include density of infective stages of the parasite in the environment, rate of exposure between host and infective stage, and host susceptibility. Density and nutritional status of avian populations and the interaction with avian trematodes may have consequences that result in epizootics.

CLINICAL SIGNS

Clinical signs of trematode infections vary and depend on the number of parasites, species of trematode involved, and the organs and organ systems affected. Signs seen in gastrointestinal infections include watery blood-stained diarrhea and pericloacal feathers stained with blood (Roscoe and Huffman 1982), weakness (i.e., wing droop) (Huffman and Roscoe 1989), leg weakness (van Haitsma 1931), inability to fly (Kocan and Kocan 1972), unsteady gait, disorientation, and a weak raspy call (Huffman and Roscoe 1989). Emaciation (Poonswad et al. 1992) and diarrhea (Dedrick 1965; Patnaik et al. 1970; Graczyk and Schiff 1993) also occur in wild birds. A high intensity of infection with *Paratanaisia bragai* in the kidney can cause apathy, loss of weight, diarrhea, and death in pigeons (Portugal et al. 1972; Arnizaut et al. 1992). Cloacal discharges have been reported in waterfowl infected with gastrointestinal trematodes (Annereaux 1940; Biester and Schwarte 1959). Increases in cloacal temperature have also been noted (Gagnon et al. 1993). Anemia can be pronounced in infected birds (Kocan and Kocan 1972; Huffman and Roscoe 1989; Luppi et al. 2007). Increases in hemoglobin and packed cell volume have been reported (Gagnon et al. 1993). Mallards (*Anas platyrhynchos*) experimentally infected with *S. globulus* develop increased prothrombin time. Excysted metacercariae were shown to produce beta hemolysis on blood agar (Tabery et al. 1988). Approximately 25 proteins have been isolated from the excretory/secretory products of *S. globulus* (Babu 2000). These have an effect on the coagulation factors Xa and IIa (Isopi 2000)—causing a 54% inhibition of factor Xa and a 17% inhibition of IIa. How the excretory/secretory products inhibit the factors has not been determined.

PATHOLOGY

Trematodes can cause lesions in their hosts by a number of different mechanisms. The most pathogenic species of trematodes are described in this section.

Table 12.2. Some trematodes and their anatomical location in avian hosts. As, air sacs; Ac, abdominal cavity; B, gall bladder; Bd, bile ducts; BF, bursa Fabricii; Bv, blood vessels; C, cloaca; c, conjunctiva; Ca, ceca, E, eye; Es, esophagus; I, intestine; IO, infra-orbital sinus; K, kidney, L, liver; M, buccal cavity; N, nasal cavity; n, nictitating membrane; Od, oviduct; PV, proventriculus; s, subcutaneous cysts; T, trachea; Tb, trachea and bronchi; Tc, thoracic cavity (Dawes 1946; Yamaguti 1971).

Trematode	As	Ac	B	Bd	BF	Bv	C	c	Ca	E	Es	I	IO	K	L	M	N	N	Od	PV	Sc	T	Tb	Tc
Brachylaemidae																								
Leucochloridiomorpha					+																			
Cathaemasiidae																								
Cathaemasia											+													
Clinostomidae																								
Clinostomum											+					+								
Cortrematidae																								
Cortrema					+																			
Cyathocotylidae																								
Cyathocotyle												+												
Cyclocoelidae																								
Bothrigaster	+																						+	
Cyclocoelum	+																+							+
Typhlocoelum																	+						+	
Hyptiasmus													+				+							
Ophthalmophagus													+											
Wardianum	+																							
Dicrocoeliidae																								
Athesmia															+									
Lyperosomum			+												+									
Oswaldoia			+																					
Platynosomum															+									

(*continues*)

Table 12.2. (*Continued*)

Trematode	As	Ac	B	Bd	BF	Bv	C	c	Ca	E	Es	I	IO	K	L	M	N	N	Od	PV	Sc	T	Tb	Tc
Diplostomatidae																								
Diplostomum												+												
Neodiplostomum												+												
Posthodiplostomum												+												
Uvulifer												+												
Echinostomatidae																								
Chaunocephalus												+												
Echinostoma							+					+												
Echinoparyphium												+												
Echinochasmus												+												
Himasthla																								
Hypoderaeum							+																	
Parorchis					+		+																	
Stephanoprora												+												
Eucotylidae																								
Eucotyle														+										
Paratanaisia														+										
Eumegacetidae																								
Eumegacetes							+																	
Heterophyidae																								
Ascocotyle												+												
Cryptocotyle												+												
Phagicola												+												
Lecithodendriidae																								
Leyogonimus												+												
Macyella												+												
Leucochloridiidae																								
Leucochloridium					+		+																	
Urotocus					+																			

| | | |

Table 12.2. (*Continued*)

Trematode	As	Ac	B	Bd	BF	Bv	C	c	Ca	E	Es	I	IO	K	L	M	N	N	Od	PV	Sc	T	Tb	Tc
Psilostomatidae																								
Psilostomum												+												
Psilochasmus												+												
Ribeiroia												+												
Sphaeridiotrema												+								+				
Schistosomatidae																								
Austrobilharzia						+																		
Gigantobilharzia						+																		
Bilharziella						+																		
Stomylotrematidae																								
Laterotrema					+																			
Stomylotrema							+					+												
Strigeidae																								
Cotylurus					+							+												
Apatemon												+												
Parastrigea												+												
Strigea												+												
Thapariellidae																								
Thapariella												+												
Troglotrematidae																								
Collyriclum																					+			
Renicolla																					+			

Pathology may be due to a direct host–parasite interaction, mechanical insult resulting in tissue damage, or by the ingestion of host tissue. Host immune responses cause inflammation and immune-mediated pathology. The lesions are closely related to the anatomical location of the parasite.

Liver, Bile Ducts, and Gall Bladder

Gross lesions caused by trematodes that develop within the bile ducts and gall bladder of their avian hosts include irritation and inflammation of the bile duct epithelium, cholangiectasis, enlargement of the bile duct lumen, blockage of the duct, and back flow of bile into the liver, leading to dystrophic and atrophic changes in the liver. Lesions have been reported from infections of *Metorchis bilis* in White-bellied Sea-Eagles (*Haliaeetus leucogaster*) (Krone et al. 2006), and infections of *Opisthorchis* sp. in Western Marsh Harriers (*Circus aeruginosus*) and Northern Harriers (*Circus cyaneus*) (Averikhin et al. 1984). Intensity of infection likely plays a role in severity of lesions. For example, over 200 individuals of an *Opisthorchis* sp. were recovered from the bile ducts of infected Western Marsh-Harriers, causing inflammation of the duct walls, enlargement of the lumen, and stasis of the contents, leading to dystrophic and atrophic changes in the liver.

Fatal hepatic trematodiasis caused by *Amphimerus elongatus* has been diagnosed in Double-crested Cormorants (*Phalacrocorax auritus*), Common Loons, and Bald Eagles (*Haliaeetus leucocephalus*). Most birds had enlarged livers with irregular capsular surfaces and numerous white, dark green, or black foci and tracts. Microscopic lesions consisted of granulomas composed of multinucleated giant cells with fibrous connective tissue and other inflammatory cells surrounding necrotic debris, trematode eggs, trematode pigment, and, occasionally, bacterial colonies. Gravid trematodes were associated with compression of adjacent hepatocytes in portal areas.

Amphimerus heterolecithodes infects the bile ducts of the liver of Wild Turkeys (*Meleagris gallopavo*). The ducts can be occluded and there can be hyperplasia or complete desquamation of the epithelium of the duct walls. In areas that contain numerous parasites, there is extensive fibrosis. The parasite has been reported free in the liver parenchyma. Trematode eggs have been found in the kidney and pancreas of Double-crested Cormorants, suggesting that the parasite migrates via the bile duct, pancreatic duct, and ureter to reach these organs (Kuiken et al. 1999).

Kidneys

Gross lesions caused by trematodes that develop within the kidney of birds include distention of the collecting tubules and a thickening of their walls and extensive cellular infiltration of the parenchyma (dos Santos 1934).

At necropsy, Blue- and Yellow-Macaws (*Ara ararauna*), Blue-winged Macaws (*Primolius maracana*), White-eared Parakeets (*Pyrrhura leucotis*), and Ring-necked Pheasants (*Phasianus colchicus*) infected with *P. bragai* had enlarged kidneys with brown-yellow discoloration and irregular cortical surfaces. Microscopic lesions consisted of granulomatous nephritis and included an interstitial, multifocal to coalescent, lymphoplasmacytic infiltrate with some epithelioid macrophages and a few heterophils. Adult worms and eggs were observed within dilated tubules and in the renal pelvis. In one bird, some parasite eggs were located interstitially and associated with an intense adjacent granulomatous reaction (Luppi et al. 2007).

Air Sacs

Bothrigaster variolaris (Cyclocoelidae) infects the air sacs of Snail Kites (*Rostrhamus sociabilis*) (Cole et al. 1995). Grossly, the air sacs were opaque and tan granular deposits had accumulated in the folds and angles of the tissues. Primary microscopic lesions included moderate pyogranulomatous bronchitis and peribronchitis, with mild squamous metaplasia of the epithelium near intrabronchial trematodes. Mild granulomatous airsaculitis composed exclusively of large, pigment-laden macrophages was also noted.

Gastrointestinal Tract

Lesions associated with gastrointestinal trematodes can be mild to severe depending on the number of parasites and species. The character of the lesions also depends on the species of trematode. Lesions are generally confined to the gastrointestinal tract and can range from mild enteritis to severe ulcerative hemorrhagic enteritis. *Sphaeridiotrema globulus*, *C. bushiensis*, and *L. polyoon* are three gastrointestinal parasites that cause epizootics in waterfowl in the US. Infections with *S. globulus* cause ballooning of the jejunum and ileum and the affected intestine may have a generalized cyanotic appearance. Foci of hemorrhage circumscribe trematodes and are visible through the serosa. Ulcers in the jejunum and ileum may penetrate the mucosa to the circular muscle layer.

The intensity of fatal infections appears to be host species dependent. American Coots and Mute Swans (*Cygnus olor*) can die from an infection of *S. globulus* with as few as 20 parasites, whereas the fatal worm burden for Muscovy Ducks (*Cairina moschata*), Mallards, and Canada geese (*Branta canadensis*) ranges from 100 to 3,300 (Trainer and Fischer 1963; Campbell and Jackson 1977; Roscoe and Huffman 1982).

Cyathocotyle bushiensis infects the lower intestine and most commonly the cecae of ducks (Table 12.3). Both ceca can be affected and may externally appear to be dark, elongated, and regularly distended. Internally, the ceca have numerous hemorrhagic areas and whitish caseous plaques. Ulceration, generalized mucosal necrosis, and firm, irregular cores may be present (Gibson et al. 1972). Intensity of infection in Blue-winged Teals (*Anas discors*) ranged from 1 to 649 worms per individual, with an average of 260 worms. Intensity of infection in American Black Ducks (*Anas rubripes*) was 180 worms per individual (Gibson et al. 1972). Hoeve and Scott (1988) reported that as few as seven parasites could cause mortality in experimentally infected ducks. Lesions from low-intensity infections may heal rapidly once the worms complete their life span. Mortality is probably attributable to the effects of severe infections, including hemorrhage and fluid loss.

Leyogonimus polyoon infects primarily the upper and middle areas of the small intestine. Gross lesions include severe enteritis characterized by thickening of the intestinal wall and a fibrinous to caseous core of necrotic debris that blocks the lumen of the intestine (Cole and Friend 1999).

Echinostoma spp. can cause mild to severe enteritis in birds (Griffiths et al. 1976; Hossain et al. 1980). However, a slight abrasion of the mucosal surface at the site of attachment was the only damage noted in Mallards infected with *E. trivolvis* (Mucha et al. 1990).

Enlargement of the proventriculus and reddening around the orifices of the glands has been observed with infection with *Ribeiroia.* In heavy infections, grayish exudates on the surface and superficial ulceration have been reported. Histologically, the mucosal surface is covered with a fibrinous exudate and the outer portion is necrotic with a polymorphonuclear leukocytic infiltration (Kocan and Locke 1974).

Eyes

Noticeable irritation of the eyes and a retracted nictitating membrane can be observed in chickens experimentally infected with *Philophthalmus gralli* (West 1961). Waterfowl infected with *P. gralli* have swollen and hyperemic nictitating membranes (Schmidt and Toft 1981). Erosion and ulceration of the conjunctival membrane and an intense inflammatory response were evident in histological sections of infected areas of the eye. Diffuse conjunctivitis was present adjacent to the attachment site of *P. gralli* in a Swan Goose (*Anser cygnoides*) (Schmidt and Hubbard 1987).

Oviduct

Infection of the oviduct in White-throated Sparrows (*Zonotrichia albicollis*) and Wild Turkeys with *P. marcrochis* results in distention and the accumulation of considerable amounts of exudates and egg material. The oviduct may have varying degrees of inflammation, depending on the number of parasites. A catarrhal to a fibrinous exudate or a caseous mass may be present in the oviduct lumen, where broken yolks and frequently large concentrations of yolk and albumen will also be found. If the oviduct ruptures, albumen and yolk material will be present in the body cavity and peritonitis with possible organ adhesions will result (Biester and Schwarte 1959).

DIAGNOSIS

Anatomical location of adult trematodes is an important clue for their identification (Table 12.2). Birds should be closely examined for the presence of conjunctival discharges that may indicate infection with eye flukes such as *Philophthalmus* sp. or the presence of cloacal prolapse or soiling around the vent that may indicate infection with intracloacal or intestinal flukes.

The diagnosis of a trematode infection may be based on the microscopic identification of eggs in the stool. Trematode eggs are relatively small, typically have an operculum, and contain either an embryo or, in mature eggs, a ciliated miracidium. By contrast, nematode eggs usually have thin shells and contain either a morula in unembryonated eggs or a recognizable larval worm. Nematode eggs do not have an operculum, but some species may have unusual but well-defined structural modifications. Cestode eggs have thickened walls and contain a larva called an onchosphere, which possesses six hooklets. Acanthocephalan eggs contain a partially developed embryo or acanthor. The eggs of schistosomes (blood-dwelling trematodes) do not have an operculum, but do possess terminal or lateral spines (Chapter 13).

If fecal samples are examined within less than 72 h, no preservatives are needed. However, some eggs may embryonate or hatch during this time unless air is excluded from the container. To maintain fecal samples longer than 72 h, the fecal sample should be fixed in 10–15 volumes of 10% formalin.

Determination of the genus and species of the trematode can be done after fixing and staining adult worms by using traditional morphological methods (Pritchard and Kruse 1982). Trematodes that are recovered at necropsy or passed in the feces require relaxation before fixation. Chilling the worms, either in saline or in tap water overnight in the refrigerator, relaxes them with the least handling. Another method is to place them into 5–10% ethyl alcohol at room temperature. The relaxed worms can be fixed in 10% formalin or preferably alcohol–formalin–acetic acid fixative (Pritchard and Kruse 1982).

Table 12.3. The major parasite families and species of trematodes that can cause disease in wild birds.

Host order	Host species	Parasite family	Parasite	Geographic local	Lesion	References
Sphenisciformes	Little Penguin (*Eudyptula minor*)	Prosthogonimidae	*Mawsonotrema eudyptulae*	Australia	Liver necrosis	Harrigan (1992)
Pelecaniformes	Brown Pelican (*Pelecanus occidentalis*)	Heterophyidae Cyathocotylidae	*Phagicola longa* *Mesostephanus appendiculatoides*	Florida, USA	Villus atrophy	Greve et al. (1986)
	Double-crested Cormorant (*Phalacrocorax auritus*)	Opisthorchiidae	*Amphimerus elongatus*	Canada	Mutifocal hepatitis Bile duct hyperplasia	Kuiken et al. (1999)
Ciconiiformes	Great Blue Heron (*Ardea herodias*)	Clinostominae	*Clinostomum attentuatum*	North America	Esophageal obstruction	Forrester and Spalding (2003)
	White Stork (*Ciconia ciconia*) Black Stork (*Ciconia nigra*)	Cathaemasiidae	*Cathaemasia hians*	Europe	Esophageal obstruction	Stoskopf et al. (1982), and Merino et al. (2001)
	Asian Openbill (*Anastomus oscitans*)	Echinostomatidae	*Chaunocephalus ferox*	Thailand	Catarrhal enteritis	Patnaik et al. (1970), Poonswad et al. (1992), and Hofle et al. (2003)
	Cattle Egret (*Bubulcus ibis*)	Echinostomatidae	*Pegosomum* sp.	Japan	Cholangitis and cholecystitis	Murata et al. (1998)
Anseriformes	American Black Duck (*Anas rubripes*) Blue-winged Teal (*Anas discors*) Green-winged Teal (*Anas carolinensis*)	Cyathocotylidae	*Cyathocotyle bushiensis*	North America	Typhlitis	Gibson et al. (1972)
	Northern Pintail (*Anas acuta*) Northern Shoveler (*Anas clypeata*) Canvasback (*Aythya valisineria*) Mallard (*Anas platyrhynchos*) Green-winged Teal (*Anas carolinensis*) Blue-winged Teal (*Anas discors*) Greater Scaup (*Aythya marila*) Lesser Scaup (*Aythya affinis*) Mottled Duck (*Anas fulvigula*)	Cyclocoelidae	*Typhlocoelum cucumerium*	Cosmopolitan	Tracheal obstruction	Gower (1937), Town (1960), Cornwell and Cowan (1963), Taft (1971), Kinsella and Forrester (1972), Broderson et al. (1977), Mahoney and Threlfall (1978), and Scott et al. (1980)

(*continues*)

Table 12.3. (*Continued*)

Host order	Host species	Parasite family	Parasite	Geographic local	Lesion	References
	Mallard (*Anas platyrhynchos*) Northern Pintail (*Anas acuta*)	Echinostomatidae	*Echinoparyphium recurvatum*	Cosmopolitan	Enteritis	Soulsby (1965)
	Mallard (*Anas platyrhynchos*) Canada Goose (*Branta canadensis*)		*Echinosotma* sp. *Echinosotma trivolvis*	Cosmopolitan	Enteritis	Huffman (2000)
	Anas spp.	Notocotylidae	*Notocotylus attenuatus*	USA	Enteritis	Griffiths et al. (1976)
	Anas spp.	Paramphistomatidae	*Zygocotyle lunata*	North America	Typhlitis	Mettrick (1959)
	American Black Duck (*Anas rubripes*)	Microphallidae	*Maritrema acadiae*	Nova Scotia	Intestinal ulceration	Swales (1933)
	Blue-winged Teal (*Anas discors*)		*Maritrema* sp.	Canada	Intestinal enteritis	Hoeve and Scott (1988)
	Mute Swan (*Cygnus olor*) Tundra Swan (*Cygnus columbianus*) Whooper Swan (*Cygnus cygnus)*	Psilostomatidae	*Sphaeridiotrema globulus*	North America	UHE*	Roscoe and Huffman (1982, 1983)
	Lesser Scaup (*Aythya affinis*)		*Sphaeridiotrema globulus*	Washington, DC, USA	UHE	Price (1934)
	Greater Scaup (*Aythya marila*)		*Sphaeridiotrema globulus*	North Central Minnesota, USA	UHE	Minnesota Department of Natural Resources (2007)
	Ruddy Duck (*Oxyura jamaicensis*) Blue-winged Teal (*Anas discors*) Mallards (*Anas platyrhynchos*)		*Sphaeridiotrema globulus*	Wisconsin, USA	UHE	United States Geological Survey (1997)
	Dabbling Ducks (*Anas* spp.)		*Sphaeridiotrema globulus*	Canada	UHE	Hoeve and Scott (1988)
	Wood Duck (*Aix sponsa*)	Philophthalmidae	*Philophthalmus* sp.	North America	Conjunctivitis	Schmidt and Toft (1981)
Falconiformes	Osprey (*Pandion haliaetus*)	Renicolidae	*Renicola lari*	North America	Nephritis	Kennedy and Frelier 1984
		Psilostomatidae	*Ribeiroia ondatrae*	North America	Hyperplasia of the proventriculus	Kocan and Locke (1974); Kinsella et al. (1996)
	Florida Snail Kite (*Rostrhamus sociabilis plumbeus*)	Cyclocoelidae	*Bothrigaster variolaris*	Florida, USA	Air sacculitis Pyogranulomatous bronchitis	Cole et al. (1995)
	Prairie Falcon (*Falco mexicanus*)	Strigeidae		North America	Diarrhea	Dedrick (1965)

	Bald Eagle (*Haliaeetus leucocephalus*)	Strigeidae	*Strigea falconis*	North America	Hyperplasia of the Proventriculus	Kinsella et al. (1998)
		Heterophyidae	*Cryptocotyle lingua*	North America	Emaciation	Smith (1978)
	White-tailed Eagle (*Haliaeetus albicilla*)	Opisthorchiidae	*Metorchis bilis*	Finland	Cholangiectasis	Krone et al. (2006)
	Western Marsh-Harrier (*Circus aeruginosus*)		*Opisthorchis* sp.	North America	Dystrophic and atrophic liver damage	Averikhin et al. (1984)
	Northern Harrier (*Circus cyaneus*)					
	Red-shouldered Hawk (*Buteo lineatus*)	Strigeidae	*Parastrigea tulipoides*	North America	None described	Miller and Harkema (1965)
	Cooper's Hawk (Accipiter cooperii)	Psilostomatidae	*Ribeiroia ondatrae*	North America	Granuloma	Beaver (1939)
	American Kestrel (*Falco sparverius*)	Dicrocoeliidae	*Athesmia jolliei*	North America	Fibrosis of the bile ducts	Schell (1957)
	Gyrfalcon (*Falco rusticolus*)	Plagiorchiidae	*Plagiorchis elegans*	North America	Enteritis	Clausen and Gudmundsson (1981)
Galliformes	Wild Turkey (*Meleagris gallopavo*)	Brachylaemidae	*Postharmostomum gallinum*	North America	Typhlitis	Soulsby (1965)
		Opisthorchiidae	*Amphimerus heterolecithodes*	North America	Obstruction and fibrosis of bile ducts	Kingston (1984), and Davidson and Wentworth (1992)
		Prosthogonimidae	*Prosthogonimus macrorchis*	North America	Oviduct inflammation	Davidson and Wentworth (1992)
	White-throated Sparrow (*Zonotrichia albicollis*)	Prosthogonimidae	*Prosthogonimus macrorchis*	Canada	None described	Brooks et al. (1993)
	Spot-winged Wood Quail (*Odontophorus capueira*)	Eucotylidae	*Paratanaisia bragai*	South America	Nephritis	Travassos et al. (1969), Costa et al. (1975), Silva et al. (1990), Menezes et al. (2001), and Pinto et al. (2004)
	Wild Turkey (*Meleagris gallopavo*)					
	Ring-necked Pheasant (*Phasianus colchicus*)		*Paratanaisia bragai*	Brazil	Nephritis	Gomes et al. (2005)

(*continues*)

Table 12.3. (*Continued*)

Host order	Host species	Parasite family	Parasite	Geographic local	Lesion	References
Gruiformes	American Coot (*Fulica americana*)	Lecithodendriidae	*Leyogonimus polyoon*	North America Europe	Enteritis	Cole and Friend (1999), and Cole and Franson (2006)
		Psilostomatidae	*Sphaeridiotrema globulus*	North America	Enteritis	Trainer and Fischer (1963)
		Cyclocoelidae	*Cyclocoelum mutabile*	North America	Hemopericardium, blood-filled air sacs, biliary congestion	McLaughlin (1976, 1977, 1983)
Charadriiformes	Red Knot (*Calidris canutus*) Greater Yellowlegs (*Tringa melanoleuca*)	Cyclocoelidae	*Cyclocoelum mutabile*	North and South America	Retardation of moult	Underhill et al. (1994), and Branton et al. (1985)
	Royal Tern (*Thalasseus maximus*) Laughing Gull (*Larus atricilla*) Yellow-crowned Night-Heron (*Nyctanassa violacea*)	Philophthalmidae	*Philophthalmus hegeneri*	North America	Conjunctivitis	Nollen and Kanev (1995)
	European Herring Gull (*Larus argentatus*)		*Philophthalmus gralli*	North America	Conjunctivitis	Schmidt and Toft (1981)
Columbiformes	Ruddy Ground-Dove (*Columbina talpacoti*)	Eucotylidae	*Paratanaisia bragai*	South America	Renal medullary collecting ducts and ureters	Pinto et al. (2004)
Coraciiformes	Belted Kingfisher (*Megaceryle alcyon*)	Opisthorchiidae	*Amphimerus elongates*	North America	Bile duct hyperplasia	Boyd and Fry (1971)
	Ringed Kingfisher (*Megaceryle torquatus*)	Cathaemasiidae	*Pulchrosoma pulchrosoma*	Peru	Lung granulomas	Merino et al. (2003)
Passeriformes	Wood Thrush (*Hylocichla mustelina*)	Troglotrematidae	*Collyriclum faba*	North and Central America	Wasting and anemia, obstruction of the cloaca	Farner and Morgan (1944), and Kirmse (1987)
	American Robin (*Turdus migratorius*)	Dicrocoeliidae	*Brachylecithum mosquensis*	North America	Obstruction of bile ducts	Schell (1957)

*UHE, Ulcerative hemorrhagic enteritis.

In studies designed to clarify relationships between morphologically similar species, molecular techniques are being developed to identify trematodes (Galazzo et al. 2002). Adult specimens of the opisthorchiid liver flukes *Opisthorchis felineus* from the Western Marsh-Harrier and *M. bilis* found in White-tailed Eagles (*Haliaeetus albicilla*) can be identified by using species-specific primers based on a part of the mitochondrial cytochrome *c* oxidase I gene (Pauly et al. 2003). To better understand the systematics and biogeography of *Ribeiroia* sp. from Great Blue Herons (*Ardea herodias*), the intertranscribed spacer region 2 of the ribosomal gene complex has been sequenced to determine differences between species (Wilson et al. 2005).

IMMUNITY

There is experimental evidence of acquired and age-related immunity in wild birds with trematode infections. Acquired resistance to infection with *S. globulus* has been reported in experimentally infected Mallards (Huffman and Roscoe 1986). When exposed to a moderate dose of metacercariae of *S. globulus*, Mallards can develop resistance to subsequent reinfection. Host cell-mediated immunity and wound healing in Mallards experimentally infected with *S. globulus* has been evaluated (Mucha and Huffman 1991). An increase in mast cells and eosinophils occurred in intestinal tissue of infected ducks, but not in controls. Antibodies that were reactive with antigens of *S. globulus* have been demonstrated in Mallards (Jones 1993). Immunity to reinfection with *Zygocotyle lunata* has also been reported (Willey 1941).

The age of the host at the time of infection may be a factor in the number and size of eye flukes (*Philophthalmus* sp.) recovered from laboratory infected chickens or geese (Nollen 1971). No protection to a challenge infection was provided by a 10-day initial infection with *Philophthalmus megalurus.* An initial infection with *Philophthalmus hegeneri* failed to protect chickens against a homologous challenge 12–14 days later (Fried 1963). It appears from these studies that there is little host immunity after infection with *Philophthalmus*, although higher antibody titers were reported in infections with *P. megalurus* infection than those from *P. gralli* (Snyder 1991).

There appears to be age-specific immunity to reinfection with *Crytocotyle lingua*. Ducks have been reported to be refractory to reinfection with this species and older terns and gulls harbor relatively few mature worms and pass recently excysted metacercariae in their feces. In contrast, young birds are usually heavily parasitized (Willey and Stunkard 1942).

In a comparison of pairs of closely related species of birds that differ with respect to whether they are migratory or residents, the size of two immune defense organs (the bursa of Fabricius and the spleen) was consistently larger in the migratory species (Møller and Erritzøe 1998). Since the bursa is found only in juvenile, sexually immature birds, adaptations for immune defense appear to exist before the start of the first migration.

PUBLIC HEALTH CONCERNS

Nollen and Kanev (1995) documented several cases of human infections with preadult (prepatent) eye flukes. A human eye infection with *Philophthalmus* sp. was reported from Japan from a 67-year-old farmer (Mimori et al. 1982). However, most avian trematodes pose no threats to humans.

DOMESTIC ANIMAL HEALTH CONCERNS

Echinostomiasis (*Echinostoma* spp.) is a significant cause of mortality in commercial duck farms in Europe and Asia (Kishore and Sinha 1982). The parasite can be maintained within the domestic flock or brought in by wild birds. *Psilochasmus oxyurus* has been reported from domestic geese in Brazil where flocks are generally maintained under poor sanitary conditions (Fernandes et al. 2007).

Three types of integrated fish-cum-duck farming practices have been developed in China that can allow exposure of domestic waterfowl to trematode infections: (1) raising large groups of ducks in open rivers, lakes, and reservoirs during the day and confining the birds in pens at night, (2) raising ducks on the edge of ponds where a large duck pen is constructed on flat areas of the shore with appropriate cemented areas for dry and wet runs, and (3) embankment and fencing of ponds to form both dry and wet runs (Bao-tong and Hua-zhu 1984).

WILDLIFE POPULATION IMPACTS

Birds are hosts to a wide variety of trematodes, but with few exceptions the significance of these infections on wild populations is unknown. Mixed trematode infections are common and the effect of any one parasite species depends on other parasites, diseases, or stressors that may be present. When trematodes do not directly kill the host they may, however, affect behavior, reproduction, the assimilation of nutrients, and in other ways contribute to the ill health of birds (Threlfall 1986).

Severe and repeated epizootics in wild waterfowl have been caused by *S. globulus*, *C. bushiensis*, and *L. polyoon*. An epizootic in wild ducks was attributed to *Maritrema acadiae* by Swales (1933). This species has

only recently been reported again from Mar Chiquita coastal lagoon, Buenos Aires province, Argentina, when adults of *Maritrema bonaerensis* n. sp. were collected from the intestine of Brown-hooded Gulls (*Larus maculipennis*) and Olrog's Gulls (*Larus atlanticus*) (Etchegoin and Martorelli 1997).

TREATMENT AND CONTROL

Treatment and control measures for avian trematodes are few, especially for free-ranging waterfowl. The only practical solution is to remove birds from the source of infection. This can be done if the intermediate hosts are known. Control measures could involve reduction of snail intermediate hosts through the use of molluscicides or by draining snail habitat.

Good waste management practices help prevent infection in captive situations. Oxyclozanide has been used on duck farms in Poland for treatment of the trematode *N. attenuatus*. Treatment was successful in eliminating the worm and preventing contamination of the pasture (Robertson and Courtney 1995).

Birds infected naturally with *P. gralli* at the San Antonio, Texas, zoo were treated successfully with creoline (Nollen and Murray 1978), and the eyes were immediately flushed with sterile distilled water to remove the worms. Greve and Harrison (1980) reported that young Ostriches (*Struthio camelus*) raised in captivity were found to harbor large numbers of adult *P. gralli* in the orbital cavity between the nictitating membrane and outer eyelid. Persistent treatment with carbamate powder and antibiotics finally eliminated the worms.

In raptors, trematode infections are usually regarded as being of little clinical significance. If diagnosed and considered to be significant, they can be treated with praziquantel (Kollias et al. 1987).

MANAGEMENT IMPLICATIONS

Massive late-summer mortalities of American Black Ducks, Blue-winged Teal, and Mallards have been attributed to mixed infections of *C. bushiensis* and *S. globulus* in the St. Lawrence River south of Quebec. Infections were linked to ingestion of the invasive European gastropod *Bithynia tentaculata* (Hoeve and Scott 1988). In 1997, tens of thousands of American Coots were killed in Wisconsin's Shawano Lake by a third digenean trematode (*L. polyoon*) that was known formerly only from Europe. Once again, introduced *Bithynia* played a key role in transmission of the trematodes (USGS-NWHC Fact Sheet). Since 2002, all three worms have been implicated in massive waterfowl mortalities in Wisconsin (United States Geological Survey 2007). Waterfowl mortality attributable to *Cyathocotyle* and *Sphaeridiotrema* in Minnesota's Lake Winnibigoshish has been linked to *Viviparus georgianus* (Minnesota Department of Natural Resources 2007). This snail is a native of the American southeast and is much more widely distributed throughout the US than *Bithynia*. If these trematodes can infect *Viviparus*, they would also seem likely to be able to exploit other indigenous snails and in so doing expand their potential ranges nationwide.

As stopover sites for waterfowl become fewer, remaining refuges become more important in sustaining populations of migratory birds. When migrants become concentrated within refuges, the probability that epizootics may occur increases. The introduction of waterfowl into new continents may have led to the transfer and establishment of their parasites into these new locations. For example, *S. globulus* was first reported in 1927 in the US, most likely as a result of the importation and release of Mute Swans.

During migration, wild birds may carry trematodes over long distances to new areas and the resulting introduction of their helminth parasites can put naïve, native hosts at risk. Enhancing productivity of an aquatic habitat, through the impoundment of flowing water, eutrophication, and increased thermal inputs, can increase mollusk populations and other intermediate hosts of trematodes. This can increase the prevalence of trematode parasitism in birds using the area. Both migration and pollutants may stress avian hosts, suppress immune responses, and enhance vulnerability to parasitic disease. To better manage healthy populations of wild birds, continued research and surveillance are critical. Bird population monitoring programs should focus on identifying the foci, pathways, and intermediate hosts for trematodes; continue to develop methods for detecting new populations of intermediate hosts and parasites; and develop strategies and methods to control and manage populations of intermediate hosts. Hazing, or chasing waterfowl elsewhere, would not be effective at reducing losses and may aid in spread of the diseases to other lakes or wetlands.

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13
Schistosomes

Jane E. Huffman and Bernard Fried

INTRODUCTION

The avian schistosomes are a specialized group of trematodes that develop as adults within the circulatory system or nasal tissue of their avian hosts. They comprise the largest and most diverse clade of the family Schistosomatidae (Brant et al. 2006) and include nine genera: *Allobilharzia*, *Austrobilharzia*, *Bilharziella*, *Dendritobilharzia*, *Gigantobilharzia*, *Jilinobilharzia*, *Macrobilharzia*, *Ornithobilharzia*, and *Trichobilharzia.* Like other trematodes (Chapter 12), avian schistosomes have a two-host life cycle and use freshwater snails as intermediate hosts. *Trichobilharzia* is the most extensively studied genus (Horák and Koláová 2005).

Schistosomes are important pathogens of birds in areas where intimate contact with infected snails occurs. Pulmonary lesions may be evident for species that live in blood vessels of the visceral organs, while neurological signs may be evident for species of nasal schistosomes that undergo larval development within tissues of the central nervous system (CNS).

Avian schistosomes are frequently responsible for human cercarial dermatitis or "swimmer's itch"—a skin rash caused by penetration of the skin by free living cercarial stages of species of *Gigantobilharzia*, *Ornithobilharzia*, *Trichobilharzia*, and *Austrobilharzia.*

SYNONYMS

Trichobilharziasis, cercarial dermatitis, schistosomiasis, swimmer's itch.

HISTORY

Schistosomiasis has a very long history. The first description of the disease in humans occurs some 3,000 years ago in the Egyptian medical papyrus, the Papyrus Ebers, and clearly described the symptoms, including blood in the urine. Bilharz working in Egypt discovered the adult human parasites in 1852, but it was a further 60 years before the life cycle was elucidated in 1912 by Fujinami in Japan (Mahmoud 2004).

An 1855 description by LaValette of an erythemous maculopapular eruption is presumed to have been cercarial dermatitis, although its relationship to the avian schistosomes of waterfowl was not yet known. Karube, a maculopapular rash, was also common among rice farmers who spent a great deal of time in cercariae-infested water. The first avian schistosome life cycle was described by Oiso (1927) for *Bilharziella yokogawai* from domestic ducks. In the American scientific literature, Cort (1950) was the first to demonstrate that swimmer's itch was caused by the cercariae of non-human schistosomiasis in 1928. Prior to that, it was believed that all such eruptions were caused by human schistosomiasis.

HOST RANGE AND DISTRIBUTION

Migratory water birds, including shorebirds, ducks, and geese, are the most typical hosts for avian schistosomes and movements of infected birds along major migratory flyways may play an important role determining their distribution in North America (Jarcho and van Burkalow 1952), Asia and the Pacific basin (Chu 1958), and Europe and Africa (Moreau 1972).

Species of *Austrobilharzia*, *Ornithobilharzia*, *Bilharziella*, *Trichobilharzia*, *Gigantobilharzia*, and *Dendritobilharzia* are cosmopolitan in distribution, while others appear to be more regional. Nasal schistosomes appear to be frequent parasites of birds in central Europe (Rudolfová et al. 2002), but it is not clear if this is a sampling artifact. Their small size, threadlike shape, and cryptic life in the nasal mucosa may make them difficult to detect if not specifically searched for.

Schistosomes are associated primarily with freshwater habitats in all temperate and tropical regions of the world and typically mirror the distribution of their snail intermediate hosts (Table 13.1). Larval *Ornithobilharzia* and *Austrobilharzia* are both parasites of marine caenogastropods and are found as adults primarily

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Table 13.1. Known intermediate and final hosts of avian schistosomes (Horák et al. 2002).

Parasite genus	Geographic location	Intermediate host	Habitat	Avian host order
Allobilharzia	Iceland	Unknown	U	Anseriformes
Austrobilharzia	Worldwide	Prosobrachia	SW	Anseriformes Charadriiformes Ciconiiformes
Bilharziella	Northern Hemisphere	Pulmonata	FW	Anseriformes
Dendritobilharzia	Worldwide	Pulmonata Opisthobrachia	FW	Anseriformes Phoenicopteriformes
Gigantobilharzia	Worldwide	Pulmonata Opisthobrachia	FW, SW	Anseriformes Charadriiformes Ciconiiformes Passeriformes
Jilinobilharzia	China	Pulmonata	FW	Anseriformes
Macrobilharzia	Worldwide	Unknown	U	Pelecaniformes
Ornithobilharzia	Northern Hemisphere	Prosobrachia	SW	Charadriiformes Pelecaniformes
Trichobilharzia	Worldwide	Pulmonata	FW	Anseriformes Ciconiiformes Columbiformes Coraciiformes Galliformes Passeriformes Pelecaniformes

FW, larval development in fresh water; SW, larval development in sea water; U, Unknown.

in gulls. Larval *Bilharziella*, *Trichobilharzia*, and *Gigantobilharzia* are parasites of pulmonate snails and as adults are found in a broad diversity of birds, including ducks, geese, grebes, and passerine birds. *Dendritobilharzia* larvae are parasites of pulmonate snails, but as adults occur only in ducks and grebes.

ETIOLOGY

General descriptions of the family Schistosomatidae and taxonomic histories of these parasites can be found in Farley (1971) and Gibson et al. (2002). Members of the Schistosomatidae have common life histories and patterns of transmission. All have brevifurcate furcocercous or fork-tailed cercariae that develop to sexual maturity following direct penetration of the host (Yamaguti 1975) and typically occupy the circulatory system as adults. Schistosomes are venous specialists with the exception of *Dendritobilharzia pulverulenta*, which inhabits the mesenteric arteries of ducks (Vande Vusse 1979; Platt and Brooks 1997).

There are relatively few morphological distinctions between the male and female avian schistosomes when compared to other members of this family. Males are considerably larger than females and possess a gynecophoric canal. This canal is a ventral groove running the length of the male schistosome into which the threadlike female worm fits. Presence or absence of this canal, relative size of the canal, presence or absence of an oral sucker, and morphological characteristics of the intestinal ceca are important characters for distinguishing the genera of avian schistosomes (Gibson et al. 2002).

The Schistosome Group Prague has made significant advances in the systematics and biology of these worms in Europe. The genus *Trichobilharzia* is the most species-rich genus within the family, with over 40 recognized species. The entire mitochondrial genome of *Trichobilharzia regenti* has been recently sequenced and annotated (Webster et al. 2007). The gross features of the genome of *T. regenti* are similar to those of mammalian schistosomes that have been characterized, and the mitochondrial genome is identical to the human parasite, *Schistosoma japonicum*, in terms of gene order. Intrinsic properties of the mitochondrial genome of *T. regenti* include potentially useful markers, repeat regions and multiple individual genes that may be useful for development of molecular markers for diagnostic, epidemiological, and population level studies. Other studies of the phylogenetics of this group have used molecular markers, morphological characters, intermediate and definitive host associations, and

biogeography to investigate relationships among genera (Snyder and Loker 2000; Lockyer et al. 2003). Results of these studies indicate that the avian clade consists of six genera of exclusively avian parasites and two genera of mammalian flukes from North America. This study provides little evidence concerning the identity of ancestral molluscan or vertebrate schistosome hosts but does demonstrate that host switching has been an important feature of schistosome evolution. Evidence also indicates that the reduced sexual dimorphism characteristic of some avian schistosomes is derived evolutionarily.

EPIZOOTIOLOGY

Adult schistosomes usually reside in veins around the intestine of their avian hosts and release eggs that make their way into the digestive tract and then pass out of the host with the feces. If the eggs are deposited in water, they will hatch within an hour if conditions are right and release a ciliated, free-swimming, nonfeeding aquatic stage, the miracidium. The miracidium has enough energy to keep moving for about a day. Once the miracidium comes in contact with a suitable snail, either it will penetrate the integument or it may be ingested through the mouth.

Avian schistosomes exhibit a high specificity toward snail intermediate hosts. The molecules involved in attraction are soluble macromolecular miracidia-attracting glycoproteins, the carbohydrate moieties of which are responsible for signal specificity (Hass 2003). Within the snail, the miracidium will elongate to form a reproductive sac called the sporocyst. This germinating structure will produce a second generation of sporocysts. In approximately 30 days, the sporocysts produce cercaria. The cercariae are furcocercous (have a forked tail). After leaving the snail, the cercariae swim freely in a zigzag pattern. They are negatively geotropic, positively phototropic, and rest occasionally by grasping the water surface or debris with the ventral sucker (Rind 1991). Water temperature and exposure to sunlight are principal determinants of the life span of cercariae. Cercarial die-off increases during hot and sunny days (Mulvihill and Burnett 1990). The life span for the cercariae is variable but averages about 24 h.

Infective schistosome cercariae normally gain entry to a mammalian or avian host by attaching to the skin with the ventral sucker and using a number of proteolytic enzymes to digest a route to reach blood capillaries or lymphatic vessels (McKerrow and Salter 2002; Mountford and Trottein 2004). Cercariae can also be ingested and then enter the blood vessels in the walls of the pharynx or esophagus. Cercariae of *Trichobilharzia ocellata* exhibit low specificity in recognizing their definitive host species and will therefore penetrate mammals (Hass and van de Roemer 1998). Cercarial attachment to, and enduring contact with, the vertebrate skin can be stimulated by temperature and chemical signals (ceramides and cholesterol), whereas the penetration itself is triggered by fatty acids (Hass 2003). Upon registering relevant stimuli, the cercariae start to release the contents of their penetration glands. Several proteins with activities probably playing a role in penetration have been detected in cercarial homogenates and/or secretions (Horák and Kolářová 2005).

Once in the skin, schistosomula, the immature forms of a schistosome after they have entered the blood vessels of their host, need to navigate an appropriate route to the target tissue. They move toward deeper skin layers and search for a blood vessel. They are able to monitor concentration gradients of chemical stimuli (D-glucose and L-arginine) and exhibit chemotactic orientation (Grabe and Haas 2004a). The schistosomes may use negative photo-orientation to move away from the light source into deeper skin layers (Grabe and Haas 2004b).

Schistosomes traverse the skin of their primary host within days, and the vast majority enter the circulation and migrate to specific locations in the host to complete their development (Table 13.2). The CNS is the most likely route to the nasal cavity for *T. regenti* and other species that complete their development in this location.

Once sexual maturity is reached, adult worms produce large numbers of eggs that are placed precisely in the venous system because they are released against the blood flow. Eggs are sequestered usually within the portal system of the avian host, thus restricting egg dispersal. Male and female schistosomes are permanently paired while they inhabit the bloodstream of their vertebrate hosts. Female schistosomes produce eggs only when they are in intimate association with a male. The natural elasticity of the vessel serves to hold the eggs in place against the flow of blood (Basch 1991). Endothelial cells actively migrate over the eggs and passively transfer the egg to the perivascular space, where they are subject to the host immune response (File 1995). A significant number of eggs may escape into the external environment before a heavily infected host is incapacitated by or dies from the infection (Platt and Brooks 1997). Avian schistosomes usually complete their life cycle in 2 months; however, the specific time varies with each species.

Transmission of avian schistosomes depends on their ability to find, recognize, penetrate, and prosper within appropriate intermediate and final hosts. This requires relevant host signals monitored by the parasite for orientation and migration purposes, but also the ability of parasites to evade host immune reactions and

Table 13.2. Anatomical location of some common species of adult avian schistosomes.

Tissue	Parasite species
Veins of nasal fossae	*Trichobilharzia duboisi* *Trichobilharzia arcuata* *Trichobilharzia nasicola* *Trichobilharzia rodhaini* *Trichobilharzia spinulata* *Trichobilharzia regenti* *Trichobilharzia aureliani* *Trichobilharzia australis*
Dorsal aorta	*Dendritobilharzia pulverulenta*
Hepatic portal, mesenteric and intestinal veins	*Austrobilharzia* spp. *Bilharziella* spp. *Gigantobilharzia* spp. *Trichobilharzia* spp. *Ornithobilharzia* spp.
Renal veins	*Bilharziella* spp. *Trichobilharzia* spp.
Interlobular bile ducts	*Bilharziella* spp. *Trichobilharzia* spp.
Pulmonary arteries	*Bilharziella* spp. *Trichobilharzia* spp.
Central nervous system, spinal cord, and brain	*Trichobilharzia regenti*

manipulate other regulatory systems of the host (Horák and Kolářová 2005). A serine protease was characterized from *T. ocellata* by Bahgat and Ruppel (2002) which may aid in facilitating the migration of avian schistosomes through host skin. Environmental factors such as water temperature, degree of pollution, extent of algal growth/aquatic plant habitats, aquatic bird, and coincident appropriate snail intermediate host populations influence the distribution and incidence of avian schistosomes.

Most birds probably become infected in nesting areas; however, birds may also transport mature (within the host) or larval (within snails transported on legs or plumage) schistosomes to and from the wintering locations (Woodruff and Mulvey 1997; Wesselingh et al. 1999).

CLINICAL SIGNS

Clinical signs of avian schistosomiasis are nonspecific and include weight loss, lameness, and "ill-thrift" (Wojcinski et al. 1987). After experimental exposure to the cercariae of *T. ocellata*, American Black Ducks (*Anas rubripes*), Blue-winged Teal (*Anas discors*), Muscovy Ducks (*Cairina moschata*), and Rouen ducks (*Anas platyrhynchos*) develop inflammation of the feet marked by engorgement of blood vessels and petechial hemorrhages (Rau et al. 1974). Listlessness, compulsive swallowing, respiratory distress, and occasional mild pulmonary hemorrhage are evident at 2 and 5 days after exposure to cercariae. Severity of these signs vary, at times being detectable only when the ducks were excited or under stress. Most birds produce mucoid, blood-flecked feces when they excrete large numbers of eggs.

Neurological signs have been observed in domestic ducklings infected experimentally with *T. regenti*, including leg paralysis and problems with orientation and balance (Horák et al. 1999).

Infections with schistosomes can also affect host hematology. Total leukocytes increase significantly in chickens infected with *Austrobilharzia variglandis* and peak at day 21 days postinfection (PI) (Ferris and Bacha 1986). Leukocyte counts decline over the next 3 weeks, returning to normal by day 42 PI. Increases in heterophils and monocytes are related to schistosome egg burden.

PATHOGENESIS AND PATHOLOGY

Schistosome infections in mammals cause chronic proliferative vascular lesions associated with the presence of adult parasites in the lumen of mesenteric and portal veins. In birds, however, these lesions have never been reported. Lesions in avian hosts include obliterative endophlebitis associated with the presence of adult schistosomes in intestinal and portal veins, moderate to severe lymphocytic and granulocytic enteritis associated with release of eggs by adult worms (van Bolhuis et al. 2004), and inflammatory reactions associated with migration of larvae in a variety of tissues, including the CNS. Most of what is known about the pathogenesis of avian schistosome infections is based on experimental studies that have documented migration of larvae, maturation of adults, and their associated host reactions.

Neurological signs are associated with the pathogenesis of nasal schistosomes and related to development of migrating larval stages in the CNS. Larval schistosomes are evident in the thoracic spinal cord of domestic ducklings with experimental infections of *T. regenti* by day 3 PI and are present in the synsacral and cervical portions of the spinal cord by day 6–7 PI (Horák et al. 1999). Worms are present in the cerebellum, cerebral hemispheres, ocular lobes/nerves, and nasal lobes, between day 10 and 13 PI. Adults appear in the nasal region by day 13 PI, and eggs can be detected by day 14 PI. Eggs have never been observed in the CNS. In the CNS, the parasites develop outside the blood vessels, directly in the nerve tissue, and on

nerve-associated cells. An inflammatory reaction with lymphocytic infiltration and undetermined degenerative changes develops in response to parasites or their tracks during migration in the nerve tissue (Horák et al. 1999).

During development of *T. regenti* in the CNS, immature parasites are located either in meninges or in various parts of the spinal cord and brain (Kolářová et al. 2001). In the spinal cord, the submeningeal location causes a strong inflammatory reaction around migrating schistosomula, resulting in eosinophilic meningitis. In the white and gray matter of the spinal cord and in the white matter of the brain, a cellular infiltration and spongy tissue surrounds the immature parasites. Dystrophic and necrotic changes of neurons, perivascular eosinophilic inflammation in the spinal cord and brain, and cell infiltration around the central canal of the spinal cord have been observed (Kolářová et al. 2001). Both adults and eggs are eventually detected in the nasal mucosa of infected ducklings. As eggs age, various host reactions are evident, ranging from focal accumulation of inflammatory cells to the formation of granulomas.

Avian schistosomes that develop in blood vessels surrounding abdominal organs typically migrate through the lungs before reaching their final destinations. Following experimental infection of Muscovy Ducks (*Cairina moschata*) and American Black Ducks (*Anas rubripes*) with cercaria of *T. ocellata*, parasites are found in the lungs and kidneys at 19 h PI and in the liver at 24 h PI (Bourns et al. 1973). In the lung, schistosomula break free into the air spaces. They appear first in air capillaries and parabronchi and later in secondary bronchi where they reinvade the bronchial epithelium and gain entrance into veins. Worms in the liver are found in the sinusoids but mainly in hepatic portal veins. From here, worms move initially to peripheral veins of the small intestine, but later penetrate deep into the mucosa, sometimes approaching the tips of the villi. Adult worms tended to be evenly distributed between Meckel's diverticulum and the ceca while eggs are most abundant in or near Meckel's diverticulum and the lymphatic tissue immediately posterior to it (Bourns et al. 1973). Birds release viable eggs of *T. ocellata* as early as 2 weeks PI. Mallard ducklings (*Anas platyrhynchos*) experimentally infected with *T. ocellata* had liver damage, but the lesions were not discussed (McMullen and Beaver 1945). In Mallards, parasites reach the lungs within 24 h PI and remain at this site for at least 5 days. Considerable growth and development takes place in the lungs, and damage may be sufficient to cause death.

Among other species of schistosomes that migrate through the lungs, a host inflammatory reaction and development of nodules composed of infiltrated lymphocytes, heterophils, eosinophils, and macrophages may occur in association with migrating schistosomula. These structures form around the blood vessels and in the gas-exchange tissues of the parabronchial walls and, consequently, in the walls of secondary bronchi in domestic ducks with experimental infections of *Trichobilharzia szidati* (Chanová et al. 2007). Extensive inflammation of secondary bronchi and parabronchi may be evident.

Host responses to eggs released by adult worms are common in schistosome infections. In one detailed study of egg deposition by *A. variglandis* in the mesenteric veins of domestic chickens, paired adults were observed in the mesenteric veins, or branches, paralleling the mesenteric border of the intestine (Wood and Bacha 1983). Females traveled from vessels in the serosa through the muscularis, squeezed into the small veins of the mucosa, and then withdrew back to the serosa after releasing eggs. As the infection progressed, deposition of eggs occurred in more peripheral layers of the intestine as smaller vessels became constricted from edema and cellular infiltration. This led to edema of the lamina propria and longer villi and expanded crypts were noted in the intestinal mucosa (Wood and Bacha 1983). Granulomatous responses to the presence of eggs were observed from day 12 to 18 weeks PI. Granulomas contained combination of macrophages and lymphocytes, giant cells, epithelioid cells, plasma cells, fibroblasts, eosinophils, and heterophils. The granulomas ranged from dense accumulations of macrophages and lymphocytes to fully developed granulomas. Phagocytosis by giant cells and the Hoeppli phenomenon was reported.

Among wild birds with natural schistosome infections, the host response to infection depends both on schistosome and on host species. Most lesions are associated with release of eggs that become lodged in veins associated with adult worms. These include development of granulomas, infiltration of heterophils and leukocytes, proliferation of connective tissue, and calcification of parasite eggs. Depending on species, lesions may develop in the mesenteric and pelvic veins, intestinal mucosa, liver, lungs, pancreas, cerebellum, and gizzard (Table 13.3).

In abnormal hosts, adult schistosomes may be found in scattered locations within the arterial system of a wide range of tissues. Here they typically produce few eggs that usually fail to embryonate. Mortality may occur when birds are translocated into new habitats and exposed to schistosome species they would not normally encounter. The death of 36 Brant (*Branta bernicla hrota*) was attributed to infections with *D. pulverulenta* and *Trichobilharzia* spp. when birds were translocated from a marine environment to a freshwater

Table 13.3. Genera of avian schistosomes with the location of the parasite in the host, geographic location, vertebrate host order, and selected references.

Genus	Species	Tissue	Geographic location	Host order	Reference
Allobilharzia	*visceralis*	Intestinal blood vessels and mesenterium	Iceland	Anseriformes	Kolářová et al. (2006)
Austrobilharzia	*terrigalensis*	Mesenteric veins	Canada, continental US, Hawaii, and Australia	Charadriiformes Ciconiiformes	Rohde (1977)
	variglandis	Mesenteric veins	Worldwide	Anseriformes Charadriiformes Ciconiiformes	Barber and Caira (1995)
Bilharziella	*polonica*	Mesenteric and portal veins	North America, Europe	Anseriformes	Kolářová et al. (1997)
Dendritobilharzia	*pulverrulenta*	Visceral tissue, heart	North America, New Zealand	Anseriformes Pelecaniformes	Cheatum (1940), Kinsella et al. (2004), and Davis (2006)
Dendritobilharzia	sp.	Visceral tissue, pancreas, spleen, liver, kidneys	Chile	Phoenicopteriformes	Pare and Black (1999)
Gigantobilharzia	*acotylea*	Gastrosplenic vein	North America	Charadriiformes	Ulmer (1968)
	adami	Visceral tissue	North America	Charadriiformes	Farley (1963)
	ardeola	Visceral tissue	Africa	Ciconiiformes	Farley (1963)
	gyrauli	Visceral tissue	North America	Anseriformes Passeriformes	Guth et al. (1979)
	huronensis	Visceral tissue	Continental US	Passeriformes	Strohm et al. (1981)
	huttoni	Intestinal veins	North America	Anseriformes	Leigh (1955)
	lawayi	Visceral tissue	Manitoba, Canada	Charadriiformes	Farley (1963)
	nettapi	Visceral tissue	North America	NA	Farley (1963)
	plectropteri	Visceral tissue	North America	Charadriiformes	Farley (1963)
	sturniae	Intestinal veins	Japan	Passeriformes	Oshima et al. (1991, 1992)
	tantali	NA	Africa	Ciconiiformes	Fain (1955)
Jilinobilharzia	*crecci*	Portal veins	China	Anseriformes	Liu and Bai (1976)
Macrobilharzia	*pulverulenta*	Mesenteric veins	New Mexico, USA	Pelecaniformes	Price (1929), Fain (1955), Baugh (1963), and Kohn (1964)
Ornithobilharzia	*baeri*	Mesenteric veins	East Africa	Pelecaniformes	Fain (1955)
	canaliculata	Mesenteric veins	North America	Charadriiformes	Kolářová et al. (1997)
	emberizae	Mesenteric veins	Japan	Passeriformes	Uchida et al. (1991)
Trichobilharzia	*adamsi*	Visceral tissue, liver	North America	Anseriformes	McDonald (1969)

(*continues*)

Table 13.3. (*Continued*)

Genus	Species	Tissue	Geographic location	Host order	Reference
	alaskensis	Visceral tissue	North America	Anseriformes	Becker (1956)
	anatina	Visceral tissue	Central Africa	Anseriformes	Fain (1955)
	aureliani	Nasal tissue	Africa	Podicipediformes	Fain (1956)a
	australis	Nasal blood vessels	Australia	Anseriformes	Horák et al. (1998a)
	arcuata	Nasal tissue	Australia	Anseriformes	Islam (1986)
	berghei	Visceral tissue	Central Africa	Anseriformes	Fain (1955)
	brantae	Serosal and mesenteric veins	North America	Anseriformes	Farr and Blankemeyer (1956)
	brevis	Visceral tissue	Japan	Anseriformes	Uchida et al. (1991)
	burnetti	Visceral tissue, cloacal vein	North America	Anseriformes	McMullen and Beaver (1945)
	cameroni	Visceral tissue	North America	Anseriformes Passeriformes	Wu (1953)
	cerylei	Visceral tissue	Africa	Coraciiformes	Fain (1956)b
	corvi	Visceral tissue	Japan	Passeriformes Galliformes	Ito (1960)
	duboisi	Nasal tissue	Africa	Anseriformes	Fain (1956)b
	elvae	Visceral tissue, venules of the intestinal wall	North America	Anseriformes Passeriformes	McMullen and Beaver (1945)
	filiformis	Visceral tissue	North America, Europe	Anseriformes	McMullen and Beaver (1945); Rudolfová (2001)
	franki	Hepatic and enteric veins	Europe	Anseriformes Dwarf mallards*	Müller and Kimmig (1994)
	horiconensis	Visceral tissue	North America	Anseriformes	McMullen and Beaver (1945)
	indica	Visceral tissue	Asia	Anseriformes	Baugh (1963)
	jequitibaensis	Visceral tissue	South America, Japan	Anseriformes	Leite et al. (1978)
	jianensis	Visceral tissue	Asia	Anseriformes	Liu and Bai (1976)
	kegonsensis	Visceral tissue, cloacal vein	North America	Anseriformes	McMullen and Beaver (1945)
	kossarewi	Visceral tissue, hepatic vessels	Europe	Anseriformes	Rudolfová et al. (2005)

kowalewskii	Visceral tissue	Europe, Asia	Anseriformes	McMullen and Beaver (1945)
littlebi	Visceral tissue	North America	Passeriformes	Farley (1971)
maegraithi	Visceral tissue	Asia	Anseriformes	Kruatrachue et al. (1968)
nasicola	Nasal tissue	Africa	Anseriformes	Fain (1955)
ocellata†	Intestinal veins, lungs, liver, intestine	Europe, North America, Asia	Anseriformes	Loken et al. (1995) and DeGentile et al. (1996)
oregonensis	Visceral tissue, portal, cecal and intestinal veins	North America	Anseriformes	McDonald (1969)
paoi	Visceral tissue	China	Anseriformes	Hu et al. (1994)
parocellata	Visceral tissue	Australia	Anseriformes	Islam and Copeman (1986)
physellae	Liver, mesenteric veins	North America, Japan	Anseriformes, Columbiformes, Passeriformes	McDonald (1969) and Yokogawa et al. (1976)
polonica	Visceral tissue	Poland, Ukraine	Anseriformes	Żbikowska (2002)
querquedulae	Visceral tissue	North America	Anseriformes	McMullen and Beaver (1945)
regenti	Nasal tissue	Europe, Australia	Anseriformes	Horák et al. (1998)b
rodhaini	Nasal tissue	Central Africa	Anseriformes Ciconiiformes	Fain (1955)
salmanticensis	Visceral tissue	Europe	Anseriformes	Simon-Martin and Simon-Vicente (1999)
schoutedeni	Visceral tissue	Central Africa	Anseriformes	Fain (1955)
spinulata	Nasal tissue	East Africa	Anseriformes	Fain (1955)
stagnicolae	Visceral tissue, intestinal veins	North America	Charadriiformes Passeriformes	McMullen and Beaver (1945)
szidati	Lungs, hepatic-portal system	Europe	Anseriformes	Neuhaus (1952)
waubesensis	Visceral tissue, intestinal and cloacal veins	North America	Anseriformes	McMullen and Beaver (1945)

NA, Not available.

*Experimental hosts.

†The taxonomical validity of *Trichobilharzia ocellata* is doubtful. *Trichobilharzia ocellata* should be regarded as species *inquirenda* (Rudolfová et al. 2005). In this chapter, we have used the original terminology used by the cited authors.

pond. The birds became emaciated and dehydrated with reduced pectoral muscle mass and prominent keels. Pathological findings were attributed both to eggs and to adult schistosomes and included emaciation, thrombosis of the caudal mesenteric vein and its branches, fibrinohemorrhagic colitis, and in some birds, heptomegaly and pulmonary congestion. Gallbladders were distended with bile and gastrointestinal tracts were devoid of ingesta (Wojcinski et al. 1987).

DIAGNOSIS

Live birds can be readily checked for nasal schistosomiasis by making a smear of nasal mucous using a cotton swab soaked in 0.85% saline. Eggs can be recovered but usually only when infections are intense (Blair and Ottesen 1979).

Adult worms recovered from naturally infected definitive hosts may be identified by morphological characteristics (Farley 1971) and are the most valuable for making identifications to level of species (Blair and Islam 1983). However, adult schistosomes may be knotted together or fragmented and are difficult to remove intact from infected hosts (Basch 1966). Intact specimens of *Trichobilharzia* can be successfully collected from ducks by exsanguination and retrograde perfusion of the descending aorta (Li et al. 1999). Large numbers of living adult worms were collected by this method. Species of *Dendritobilharzia* inhabit the arterial system of their avian hosts. All other bird schistosomes live in the venous system (Platt and Brooks 1997).

Eggs, miracidia, and cercariae can also provide diagnostic characteristics, but are most useful when the life cycle, developmental stages, and host specificity of the parasites are known. Keys are available for adult males of the genus *Trichobilharzia* (McDonald 1981). Descriptions of eggs of eight different avian schistosomes from birds in South Africa are available and were used to successfully place parasites in one of four possible genera: *Austrobilharzia*, *Gigantobilharzia*, *Trichobilharzia*, or *Ornithobilharzia* (Appleton 1986).

Schistosome infections can also be detected in birds by allowing miracidia to hatch from eggs in the feces. It is possible to determine the relative intensity of infection by weighing the fecal content and then counting the number of miracidia that hatch from 1 g of feces.

Molecular methods are becoming increasingly important for diagnosing and identifying schistosome infections, both in avian and intermediate hosts and in environmental samples. Internal transcribed spacers (ITS1 and ITS2) and the 5.8s ribosomal RNA gene of three European species of *Trichobilharzia* have been used successfully for identification of species within this genus, and primers developed for these genes may be particularly useful for diagnosing infection with *T. regenti*, a potential neuropathogen (Dvoák 2001; Dvoák et al. 2002). Primers based on a 396 bp tandem repeated DNA sequence (T1323) that was cloned from DNA isolated from *T. ocellata* have made it possible to identify *Trichobilharzia franki*, *T. ocellata*, and *T. regenti* in both snails and plankton collections. The T1323 sequence represents between 1 and 2% (7,000–14,000 copies) of the genome of the three *Trichobilharzia* species. Polymerase chain reaction primers, based on the T1323 sequence, are much more sensitive and also highly specific. They are sensitive enough to identify as few as one cercaria in a 0.5 g plankton sample and two cercariae in a 0.5 g sample of snail (*Lymnea stagnalis*) tissue (Hertl et al. 2002).

Avian schistosomes also induce production of specific antibodies, which may provide a certain level of protection in birds and which can be used to identify infected individuals. Assays have been developed to detect antibodies specific to cercarial antigens (Kolářová et al. 1994) and to gut-associated antigens of immature and adult flukes (Kouřilová and Kolářová 2002). Isoforms of gut-associated cathepsin B have recently been isolated from schistosomula of *T. regenti* and may be a useful candidate antigen for diagnostic purposes. Sera collected from ducks experimentally infected with *T. regenti* have antibodies that bind histological sections of the gut surface of schistosomula (Dvoák et al. 2005) and Western blots of recombinant cathepsin B (Horák and Kolářová 2005).

IMMUNITY

Schistosomiasis has been referred to as an immunologic disease, and the pathogenesis of acute and chronic schistosomiasis appears to involve immunologic mechanisms, either humoral or cell mediated, that affect both the duration of the infection in birds and the severity of lesions. There are fundamental mechanisms of immune evasion that dictate whether schistosomes succeed in intravascular environments in humans and other vertebrate hosts that are not completely understood (Brant and Loker 2005).

Challenge infections lead to a stronger inflammatory response around migrating parasites (*T. ocellata* in the lungs of birds) (Bourns and Ellis 1975). Avian schistosomes also induce production of specific antibodies that may provide a certain level of protection in birds. This has been shown in ducks, where transfer of large amounts of immune serum from donor birds to recipients was followed by partial or complete reduction in the number of eggs of *T. ocellata* in the feces and retardation of worm growth (Ellis et al. 1975).

PUBLIC HEALTH CONCERNS

Schistosome dermatitis is known as swimmer's itch, clam digger's itch, cercarial dermatitis, Gulf Coast itch, and sea bather's eruption. It is caused by the penetration of cercariae of nonhuman schistosomes into the skin of humans. On first exposure, it produces a mild erythema and edema, but on repeated exposure a marked reaction occurs with pruritus, vesicle formation, and marked papule formation. Skin lesions may be accompanied by a systemic febrile response that runs for 5–7 days and resolves spontaneously (Hoeffler 1974). The life cycle of avian schistosomes also favors cercarial dermatitis—peak cercarial production occurs in the hottest months when bathing is most common (Cort 1950). A number of species of schistosomes have been implicated as the causative agents of cercarial dermatitis. Foci of infection can be found along migratory routes of waterfowl (Blair and Islam 1983). In Europe and North America, the species most commonly associated with cercarial dermatitis in freshwater habitats are *Trichobilharzia stagnicola*, *Trichobilharzia physellae*, *and T. ocellata*. *A. variglandis* is a cosmopolitan species and is associated with cercarial dermatitis in saltwater. Treatment may not be necessary when there are only a few itching spots. An antihistaminic or mild corticosteroid cream purchased over the counter in pharmacies can be beneficial. If the initial itching is severe, then scratching can cause abrasions and skin infections may develop.

DOMESTIC ANIMAL HEALTH CONCERNS

Farming of domestic ducks, geese, or swans on reservoirs with wild waterfowl and intermediate hosts that may contain avian schistosomes may leave these animals vulnerable to infection and disease. The *aigamo method* of rice farming relies on ducks to eat insects and weeds. This method was developed in 1989 by Takao Furuno, and is used in South Korea, China, Vietnam, the Philippines, Thailand, and Iran (Furuno 1996). In the Philippines, China, and Vietnam, duck pasturing has been implicated in paddy field dermatitis caused by *Trichobilharzia paoi* (Hu et al. 1994).

WILDLIFE POPULATION IMPACTS

Large die-offs of birds as the result of avian schistosomes are rare and most reports involve small mortality events or reports of disease in isolated individuals. The two largest documented die-offs involved 45 Ring-billed Gulls (*Larus delawarensis*) on the shore of the St. Lawrence River, Canada (Dallaire 2006) and 40 wild-caught Brant that were maintained in captivity on a freshwater pond (Wojcinski et al. 1987). An intense, mixed infection with *Trichobilharzia* and *Dendritobilharzia* was considered the primary cause of death in the Brant, while the schistosome infection in the Ring-billed Gulls was not identified to genus.

TREATMENT AND CONTROL

Control of avian schistosomes is difficult and depends on breaking cycles of transmission. This may require chemical, mechanical, and biological approaches to reduce or eliminate snail intermediate hosts (Horák et al. 2002). Niclosamide has been widely used in mollusk control programs since the 1960s (World Health Organization 1965) and is still the molluscicide of choice (Perrett and Whitfield 1996). It is highly effective at all stages of the life cycle of snails (Webbe 1987) and does not adversely affect economically important crop plants, although certain algae and aquatic plants are damaged and fish mortality may occur at concentrations used to control snails (Andrews 1983). Other effective molluscicides include B-2 (sodium 2,5-dichloro-4-bromophenol), copper sulfate, and sodium pentachlorophenate (Perrett and Whitfield 1996).

Mechanical elimination of snail habitat has been successfully used to control populations of snails close to roosting habitats for some avian hosts and may be an effective way to control cercarial dermatitis (Leighton et al. 2000). Mechanical disturbance of epilithic habitat with a boat-mounted rototiller or tractor and rake successfully eliminated almost all snails when done in shallow areas of high snail concentration during the breeding and early development of the mollusks.

Treatment of infected birds with praziquantel is also effective in interrupting transmission when used in baits for dabbling ducks (Blankespoor and Reimink 1988, 1991) or for treating dwarf Mallards and Mallards infected with *Trichobilharzia* (Müller et al. 1993; Reimink et al. 1995). This method has some drawbacks. When treated baits were used, it was necessary to capture the primary definitive host, *Mergus merganser*, a diving fish-eater, and inject each bird as part of an overall wildlife management scheme, because these hosts would not consume the bait (Blankespoor and Reimink 1988, 1991). Dosage may also affect efficacy of the methods. When dwarf Mallards and Mallards infected with *Trichobilharzia* were treated with praziquantel (Müller et al. 1993), only a threefold application of 200 mg/duck at 24-h intervals led to permanent reduction of detectable miracidia. Application of praziquantel in low doses (30 or 40 mg/duck) did not reduce the number of released miracidia. Medication with praziquantel led to a strong shift of adult worms located in the enteric veins of the ducks to the liver in as little as 3 h. During prepatency, doses of 22.5 mg praziquantel per duck per day, given for 1 week, were sufficient to completely stop the release of miracidia.

In spite of these drawbacks, treatment of wild birds can be effective. Treatment of natural populations of Mallards infected with avian schistosomes in Michigan, USA, was an effective therapeutic agent for reducing natural infections of the parasite. One year after treatment, prevalence in Mallards was 1.8% versus 14.6% in an untreated control group (Reimink et al. 1995).

MANAGEMENT IMPLICATIONS

Management of avian schistosomiasis at an effective scale is difficult because of difficulties in delivering treatments and their high costs. Three basic strategies are possible: (1) prevention of the introduction of disease, (2) control of existing disease, and (3) eradication. Host–parasite interactions may differ by habitat type, host specificity may vary, and identification of species of schistosomes may be difficult, making eradication and control difficult. While snails that harbor the larval stages of avian schistosomes may be destroyed by molluscicides or mechanical treatments, this method is cost-effective only in small areas. Development of better detection methods for identification of schistosome cercariae in reservoirs could help target eradication methods for the parasite (Graczyk and Shiff 2000).

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14
Cestodes

J. Daniel McLaughlin

INTRODUCTION

Cestodes or tapeworms (class Cestoda, phylum Platyhelminthes) are extremely common parasites of birds. Most species infect the intestine, but a few can be found in the ceca or under the gizzard lining. They are readily distinguished from other worm parasites (trematodes, nematodes, and acanthocephalans) by their segmented appearance. Birds have the most diverse cestode fauna of any vertebrate group. Over 1,700 of the approximately 4,000 nominal species listed by Schmidt (1986) infect birds, and that number continues to grow as new species are recognized and described (McLaughlin 2003). Wild birds are often infected with large numbers of cestodes and average prevalence can be quite high. As reported in 16 studies from North America and Eurasia, average prevalence ranged from 18 to 69% in samples of up to 3,089 birds from 232 avian species. In each study, prevalence of cestode infection exceeded that of any other helminth group (Rausch 1983). Depending on host species, apparently healthy birds may be infected with tens, hundreds, or, in some cases, thousands of cestodes (Cornwell and Cowan 1963; Bush and Holmes 1986; Stock and Holmes 1987; Bush 1990).

The literature on avian cestodes is replete with studies of the distribution, systematics, and life histories of these parasites, but few address other aspects of host–parasite relationships or disease. Many cestodes are large enough to detect without magnification, and because they are so common, they are often observed in sick or dying birds (e.g., Kinsella and Forrester 1999). It is likely that earlier authors may have erroneously implicated cestodes as causes of disease or mortality when no other agents were evident (Rausch 1983). Further, wild birds may be infected with several species of helminths, making it difficult to ascribe effects to a particular parasite (Rausch 1983; Chapter 1).

There is little evidence to suggest that adult cestodes have an adverse effect on animals or birds (Rausch 1983; Holmes 1994). Infected animals usually grow normally and exhibit few, if any, signs of disease (Arme et al. 1983). Similarly, infected birds seldom display clinical signs, and cestodes are not considered a problem unless present in massive numbers (Greve 1986) or in malnourished (Wobeser 1981) or otherwise debilitated hosts. For example, in waterfowl, perhaps the best-studied group of wild birds, only 16 of the 264 cestode species listed by McDonald (1969a) have been associated with disease or mortality.

Nevertheless, a number of reports have associated cestodes with disease or mortality in wild or captive birds. Species of *Gastrotaenia* are known pathogens of waterfowl (Wobeser 1981), and a number of other cestodes cause lesions at the site of attachment or damage the intestinal mucosa. Fatal infections have been reported in Common Eiders (*Somateria mollissima*) (Grenquist et al. 1972; Kulachkova 1973, Persson et al. 1974; Hario et al. 1992), various species of swans (*Cygnus* spp.) (Jennings et al. 1961; Czaplinski 1965; Maksimova 1972; Papazahariadou et al. 1994), Arctic Loons (*Gavia arctica*) (Bayle 1983), Houbara Bustards (*Chlamydotis undulata*) (Jones et al. 1996a), and Willow Ptarmigan (*Lagopus lagopus*) (Holmstad et al. 2005). In some of these studies, however, the interpretation may have been confounded by the presence of other helminth species in the affected hosts. Although rare, larval cestodes have also been implicated as causes of avian mortality (Raethel 1977; Toplu et al. 2006).

This chapter is not intended to be an exhaustive review of all reports of morbidity and mortality attributed to cestode infections in wild birds. Nor is it meant to document detailed pathological responses in all examples covered. These can be found in the works cited. Rather, the objective here is to summarize some of the general effects of cestode infection on wild or captive birds and to assess their significance in natural populations and their potential effects on domesticated hosts.

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SYNONYMS

Cestodiasis, hymenolepididiasis, drepanidotaeniasis, fimbriariasis, gastrotaeniasis.

HISTORY

Early references to mortality and disease in birds due to cestodes can be found in Sprehn (1932). Most of these studies refer to infections in domesticated species. Virtually all the existing information on wild birds is based on incidental observations made during studies that had other goals. Experimental studies on avian cestodes are rare and most data come from studies of domesticated species. Even here, few controlled studies exist (Reid 1983).

Few species that cause visible pathology in wild birds have been studied either by experimental methods or by detailed studies of naturally infected birds. Some of the best examples include *Gastrotaenia dogieli* and *Gastrotaenia cygni*, two species that live under the gizzard lining of waterfowl (Wolffhugel 1938; Heck 1969; Egizbaeva and Erbolatov 1975; Egizbaeva and Basyvekova 1978; Kulukbaeva 1985), and *Schistotaenia tenuicirrus*, a species that causes intestinal diverticulae in Pied-billed Grebes (*Podilymbus podiceps*) and Horned Grebes (*Podiceps auritus*) (Boertje 1974).

ETIOLOGY

Cestodes belong to the phylum Platyhelminthes. Most species infect the intestine, a few species infect the ceca, and *Gastrotaenia* infects the gizzard. Occasionally, cestodes invade abnormal sites including the ureters (Wobeser 1974) and the gizzard muscle (McOrist 1989; Mondal and Baki 1989).

Adult cestodes are white and translucent when alive. They range from 1–2 mm to 1 m long (Rausch 1983), but many are less than 10 cm. Cestode bodies consist of a holdfast (scolex), a short neck, and a body (strobila) made up of repeated units (proglottids) that give it a segmented appearance. A mature strobila consists of three zones: a zone of immature proglottids posterior to the scolex, a zone of sexually mature proglottids with functional reproductive systems, and a postreproductive (gravid) zone consisting of proglottids that contain eggs ready for dispersal from the host.

Three of the 14 orders recognized by Jones et al. (1994) are represented in the cestode fauna of birds. About 70 species belong to the orders Tetrabothriidea and Pseudophyllidea and the remainder belong to the Cyclophyllidea (Schmidt 1986). Most cyclophyllidean species that infect birds are found in the families Hymenolepididae, Dilepididae, and Davaineidae.

Molecular evidence indicates that the Cyclophyllidea is the most highly derived order. The Tetrabothriidea is its closest relative and the Pseudophyllidea occupies a more basal position. The Pseudophyllidea (*sensu* Bray et al. 1994) is polyphyletic with only one family that is found in birds, the Diphyllobothriidae. Recent evidence indicates that the Diphyllobothriidae is independent and ancestral to the Pseudophyllidea (Olsen and Tkach 2005), but formal classification of the cestodes has yet to reflect molecular results. The family Diphyllobothriidae will be treated as though it has ordinal status in this chapter.

These three groups can be distinguished by morphology of the scolex and mature proglottids (Table 14.1). Cyclophyllidean scolices have four muscular suckers. Most species also have a rostellum (a muscular organ within the scolex) that can be projected from its apex. The rostellum is usually armed with hooks and the number, shape, and size of these are of taxonomic importance. Tetrabothriidean scolices have four large leaflike suckers called bothridia and lack a rostellum. Scolices of the diphyllobothriids have one dorsal and one ventral groove (bothria) instead of suckers or bothridia.

Table 14.1. Morphological comparison of scolices and mature proglottids of adult diphyllobothriid, tetrabothriid, and cyclophyllidean cestodes.

Characteristic	Diphyllobothriidae	Tetrabothriidea	Cyclophyllidea
Scolex with:	2 Bothria	4 Bothridia	4 Acetabula
Rostellum	None	None	Present/absent
Genital pore (position)	Ventral midline	Lateral margin	Lateral margin (ventral in Mesocestoididae)
Uterine pore	Present	Present	Absent
Uterine pore (position)	Ventral midline	Dorsal midline	None
Uterus (structure)	Tubular, coiled	Saccular	Saccular
Vitelline gland	Follicular	Compact	Compact
Vitelline gland (position)	Follicles visible throughout proglottid	Preovarian	Usually postovarian

Cestodes have complex reproductive systems. Mature proglottids of these three groups can be distinguished by the type and location of the vitelline (yolk) glands, the structure of the uterus, the position of the genital pore, and by the presence or absence of a uterine pore (Table 14.1).

Larval infections are less common. Larval cestodes are also white and range from several millimeters to several centimeters long. Larval *Mesocestoides* (Cyclophyllidea) occur mainly in the body cavity but can be found in all internal organs of heavily infected birds (Kugi 1983). Larval diphyllobothriids occur in the body cavity (Raethel 1977; Kuntz 1979) and in muscle (Kuntz 1979).

HOST RANGE

Every parasite has limits on the range of species it can infect. An infection is possible only if a series of environmental (contact) and physiological (compatibility) criteria are met (Combes 2001). This is most likely to occur in closely related species or in species that share the same diet and habitat. Most cestodes tend to occur in a single order of birds (Fuhrmann 1932); however, a given cestode may infect multiple host species, genera, or families within an order and may also infect birds of different orders (Table 14.2). Others are restricted to only a few host species. *Fimbriaria fasciolaris*, for example, infects over 60 species (7 families, 30 genera) of waterfowl (Anseriformes) worldwide (McDonald 1969a). By contrast, other species of cestodes infect less diverse orders and are found in fewer species. For example, species of *Schistotaenia* are specific to grebes (Rausch 1983; Stock and Holmes 1987), while species of *Parorchites* infect penguins (Cielecka et al. 1992). In each case, hosts are related phylogenetically, share habitats, and feed on the intermediate hosts to varying degrees.

Exchange of cestode species between related hosts is common, particularly in wetland habitats. Here, the presence of multiple host species, limited foraging space, and similar diets ensure contact between pools of infective larval stages established by each host species (Nerassen and Holmes 1975; Stock and Holmes 1987). When this happens, natural selection will likely favor host switching if parasites can successfully reproduce, eventually selecting for adaptations in the life cycle that enhance continued contact with these new hosts.

Many cestodes have been reported from multiple host orders (see Rausch 1983 and Table 14.2). However, such records by themselves can be misleading. For example, 26 cyclophyllidean species that normally infect orders of birds other than Anseriformes have been reported 37 times in waterfowl. Of these, 1 species has been reported 5 times, 2 species 3 times, 3 species 2 times, and 20 species once in over 100 surveys of wild or domestic waterfowl (McDonald 1969a, b). Fifteen of these species normally infect other aquatic birds: Charadriiformes (10 spp.), Podicipediformes (3 spp.), and Pelecaniformes (2 spp.). Thirteen of these species have been reported once in waterfowl. The normal hosts of these cestodes are aquatic birds that can share habitat, foraging areas, and at least some prey items with ducks. The other 11 species (e.g., species of *Raillietina* and *Amoebotaenia*) normally infect Galliformes (10 spp.) or Passeriformes (1 sp.) and were reported in domestic ducks that were apparently raised in proximity with chickens.

Other examples of natural infections in phylogenetically different hosts exist. Rausch (1983) reported a species of *Schistotaenia* that is normally found in grebes in a crow (*Corvus* sp.). He also suggested that ecological factors rather than physiological factors or phylogenetic relationships were more important determinants of successful infections.

GEOGRAPHIC DISTRIBUTION

Geographic distribution of cestodes can be considered at different spatial scales that are ultimately dependent on the overlap of both avian and intermediate hosts and successful transmission of the parasites. On a global scale, the cestode fauna of birds has been well documented throughout the Holarctic. However, studies in the southern hemisphere have been less intensive (Rausch 1983), and the cestode fauna is less well known. Many of the species listed in Table 14.2 have cosmopolitan or Holarctic distributions. Others, like *Parorchites* in penguins, have more restricted distributions that reflect the distribution of suitable hosts (Cielecka et al. 1992).

At a more local scale, cestode species may be present in some areas and absent in others. This is common in migratory birds where cestode species may be acquired on the breeding grounds and then transported to wintering areas. Some cestode species may persist while others may disappear if parasite life spans are short and local transmission is not possible. This can lead to seasonal declines in cestode diversity in migratory birds particularly on the wintering grounds (Buscher 1965; Hood and Welch 1980; Wallace and Pence 1986).

Alternatively, migrant birds may acquire new species, particularly if they winter in coastal areas. Transmission of cestodes with aquatic life cycles tends to be restricted to either marine or freshwater environments, possibly reflecting osmotic effects on egg or oncosphere survival. Species of *Tetrabothrius*, *Ophryocotyle*, and *Kowalewskiella* are transmitted in marine environments (Stock and Holmes 1987; Bush 1990).

Table 14.2. A partial list of cestode species that have been reported to cause pathology, disease, or mortality in wild birds.

Parasite	Distribution	Host order	Source	Reference
ADULT CESTODES				
ORDER CYCLOPHYLLIDEA				
Family Hymenolepididae				
Gastrotaenia cygni	NA, SA	A	W	Heck (1969)
			W	Willers and Olsen (1969)
Gastrotaenia dogieli	Eu, As	A	E	Egizbaeva and Erbolatov (1975)
			E	Kulukbaeva (1985)
Fimbriaria fasciolaris	C	A*, Ch, Po, Gal, Gr, F, Pe, Pi	C, D?	Basu et al. (1982)
Microsomacanthus collaris		A*, Ca, Ci, Gal, Pa	C, D?	Gitter et al. (1974)
			C, D?	Basu et al. (1982)
Microsomacanthus parvula	Eu, As, NA	A	?	Šlais (1961)
Dicranotaenia coronula	Eu, As, Af, NA	A*, Ch, Gal, Gr	C, D?	Basu et al. (1982)
Sobolevicanthus gracilis	Eu, As, Af, NA	A*, Gal, Co	C, D?	Basu et al. (1982)
Aploparaksis furcigera	Eu, As, NA	A*, Gr, Po	?	Šlais (1961)
Aploparaksis penetrans	Eu, As, NA	Ch	W	Spasskaya (1966, Figure 65)
Cloacotaenia megalops	C	A*, Gal, Gr	W	Wobeser (1974)
Hispaniolepis falcata	Af, As	Gr	W	Jones et al. (1996b)
Family Davaineidae				
Otiditaenia conoideis	Af, Eu, As	Gr	C	Jones et al. (1996a)
Otiditaenia macqueeni	Af, As	Gr	C	Jones et al. (1996a)
Raillietina sp.	NA	Gal	W	Thomas (1985)
Family Dilepididae				
Choanotaenia infundibulum	C	A, Co, F, Gal*, Gr, Pa, St	C, D?	Basu et al. (1982)
			W	McOrist (1989)
Parorchites zederi	Antarctica	Pr	W	Fuhrmann (1921)
Family Gryporhynchidae[†]				
Paradilepis delachauxi	Af, As	Pe	W	Baer (1959)
Paradilepis scolecina	C	Pe	W	Karstad et al. (1982)
Paradilepis sp.	As	Pe	W	Matta and Ahluwalia (1977)
Family Amabiliidae				
Schistotaenia scolopendra	SA	Po	W	Baer (1940)
Schistotaenia srivastavi	NA	Po	W	Rausch (1970)
Schistotaenia tenuicirrus	NA	Po	W, E	Boertje (1974)
Family Paruterinidae				
Ascometra choriotidis	As	Gr	C	Jones et al. (1996a)
Metroliasthes lucida	NA	Gal	W	Ángeles Rebolloso et al. (2006)
Cyclophyllidean sp.	NA via Af	Ph	C	Poynton et al. (2000)
ORDER TETRABOTHRIIDEA				
Tetrabothrius skoogi	As	Pr	W	Nishigai et al. (1981)
Tetrabothrius sp.	Au	Pr	W	Obendorf and McColl (1980)

(*continues*)

Table 14.2. (*Continued*)

Parasite	Distribution	Host order	Source	Reference
Family Diphyllobothriidae				
Schistocephalus solidus	C	A, Ch*, Ci, Co, F, Gal, Gav, Gr, Pa, Pe, Po, Pr	W	Grenquist et al. (1972)
			W	Persson et al. (1974)
			W	Hario et al. (1992)
Ligula intestinalis	C	A, Ci, Ch*, F, Pe*, Po*, Gav*, Pa	W	Bayle (1983)
			W	Betke et al. (2003)
LARVAL CESTODES				
Family Mesocestoididae				
Mesocestoides sp.	C	Mammals	W	Toplu et al. (2006)
			W	Millán et al. (2003)
			W	Kugi (1983)
Family Diphyllobothriidae				
Ligula intestinalis	C	See above	C	Raethel (1977)
Spirometra sp.	C	Mammals	W	Kuntz (1979)

Note: Classification follows Khalil et al. (1994). Distribution refers to the known geographic distribution as presented in McDonald (1969a, b) or Schmidt (1986). Host orders for adult cestodes are based on data in Fuhrmann (1932), McDonald (1969a, b), and Schmidt (1986). Asterisk (*) indicates the major order(s) of host(s) for a specific parasite. Host orders: A, Anseriformes; Ca, Caprimulgiformes; Ch, Charadriiformes; Ci, Ciconiiformes; Co, Columbiformes; F, Falconiformes; Gal, Galliformes; Gav, Gaviiformes; Gr, Gruiformes; Pa, Passeriformes; Pe, Pelecaniformes; Ph, Phoenicopteriformes; Pi, Piciformes; Po, Podicipediformes; Pr, Procellariiformes; Str, Strigiformes;. Asterisks when present indicate the major host order(s) of a particular species. Source of material: W, wild; E, experimental; C, captive; D, domestic; ?, source unknown. Distribution: NA, North America; SA, South America; Eu, Europe; As, Asia; C, Cosmopolitan; Af, Africa.

[†]The family Gryporhynchidae is not included in Khalil et al. (1994).

They infect pelagic and coastal birds but migrant species from freshwater habitats that winter in coastal areas may also become infected. Species of these three genera have been found in birds on breeding areas on the Canadian prairies (Stock and Holmes 1987; Bush 1990), thousands of kilometers from where they were acquired.

EPIZOOTIOLOGY

Life Cycle

Cestode life cycles are indirect and each stage must be eaten by the next host for transmission to occur. One or two intermediate hosts may be required to complete the life cycle. Life cycles are similar for species within each family of cestodes; however, their intermediate hosts may differ. Except for the Tetrabothriidea, scolices of the infective larval stages are identical to those of the adult worm.

Cyclophyllidea

Cyclophyllidean eggs are infective when passed from the host. Each egg consists of a larva (oncosphere) that is armed with three pairs of hooks and surrounded by one or two delicate membranes. In suitable conditions, the eggs of many species can survive for several months at low temperatures and some can survive short periods of freezing (Lee et al. 1992).

With the exception of the Gryporhynchidae and Mesocestoididae, life cycles of most cyclophyllidean families require one intermediate host. In most families, the intermediate hosts are invertebrates. Crustaceans, insect larvae, and annelids serve as intermediate hosts of species that infect aquatic birds. Insects, annelids, and mollusks serve as intermediate hosts of species that infect terrestrial birds. *Cladotaenia* and *Paruterina*, which infect raptors, use rodents as intermediate hosts (Rausch 1983).

Among cyclophyllideans, the oncosphere is released from the egg following ingestion and penetrates the gut

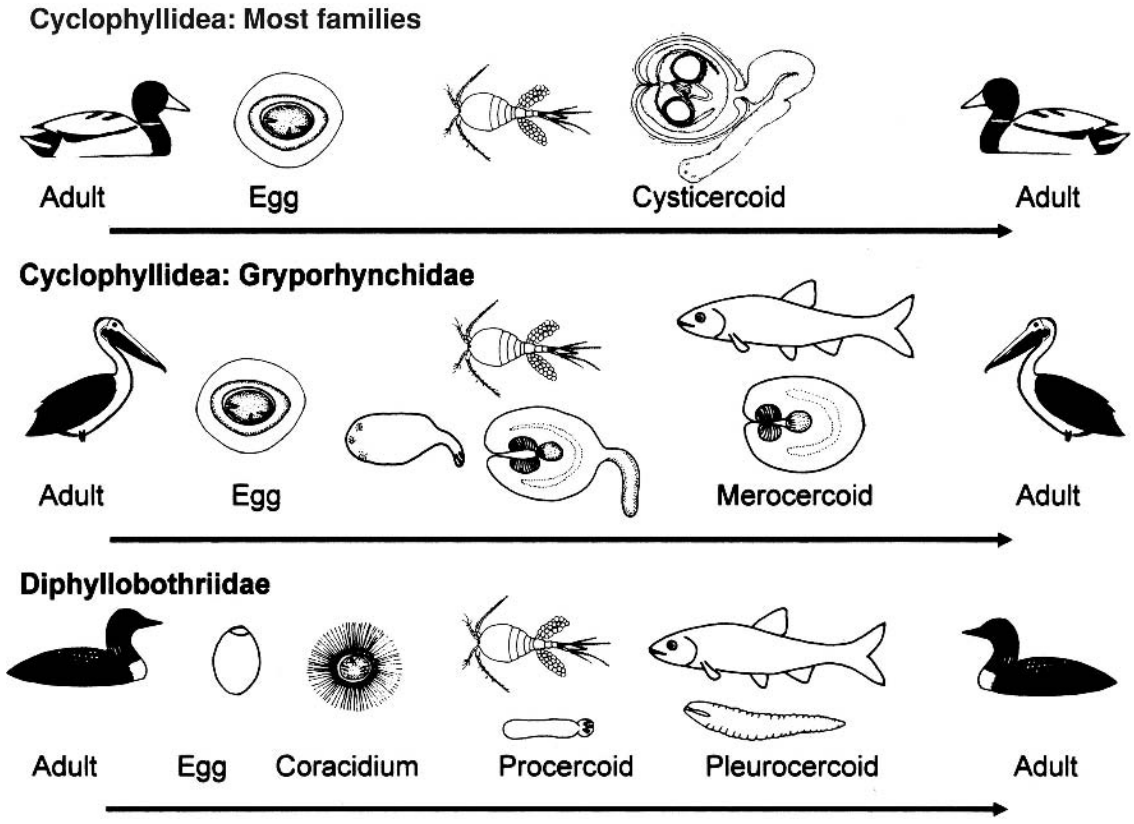


Figure 14.1. Representative life cycles of cyclophyllidean and diphyllobothriid cestodes. Larval stages of the Gryporhynchidae redrawn after Chervy (2002).

of the intermediate host. It localizes in the hemocoel, coelom, or digestive gland where it develops into a cysticercoid larva that is infective to the avian host (Figure 14.1).

Species of the family Gryporhynchidae infect pelicans, cormorants, and other piscivorous birds. Copepods and fresh or brackish water fish are required for transmission (Figure 14.1). The oncosphere develops into a procercoid larva in the hemocoel of the copepod host. When eaten by a fish, the larva migrates to the mesenteries, liver, or gall bladder where it develops into the second larval stage, the merocercoid, which is infective to the avian host (Chervy 2002).

The life cycle of the Mesocestoididae is believed to require two intermediate hosts. The first intermediate host is unknown but is thought to be an arthropod. The stage that develops in what is believed to be the second intermediate host is now considered a merocerocoid (Chervy 2002), but the more familiar term tetrathyridium will be used here. This stage is infective to the definitive host. Second intermediate hosts may be amphibians, reptiles, birds, and mammals. The final hosts are carnivorous mammals or, rarely, birds (Rausch 1994).

Under optimal conditions, developmental times of cyclophyllidean cysticercoids range from 6 days to 4 weeks (McDonald 1969a; Reid 1983). Some hymenolepidid species mature to adults in 4 days (Podesta and Holmes 1970), although 10–14 days appears to be the norm (McDonald 1969a). Dilepidid, davaneiid, and gryporhynchid species may require up to 3 weeks (McDonald 1969a; Reid 1983; Scholz et al. 2004). The adult life spans of species infecting wild birds are unknown.

Tetrabothriidea

Complete life cycles of this family are unknown; however, larval stages occur in marine crustaceans, teleosts, and cephalopods. Unlike other cestodes, the scolex undergoes further development within the final host (Hoberg 1994).

Diphyllobothriidae

Two intermediate hosts are required to complete Diphyllobothriid life cycles (Figure 14.1). The first host is a copepod and the second is a vertebrate, usually a fish. The exception is S*pirometra* whose species use all vertebrates except fish as second intermediate hosts (Bray et al. 1994).

The eggs are thick shelled, operculate, and require a period of development in water before hatching. The egg eventually releases a coracidium larva, which is essentially an oncosphere surrounded by a ciliated covering. The oncosphere penetrates the gut of the copepod host and develops into a procercoid larva in the hemocoel. When eaten by a fish, the procercoid penetrates the host gut, resumes development in the body cavity, visceral organs or musculature, and transforms into the pleurocercoid stage. This stage is infective to the avian host.

Developmental times for diphyllobothriids are 4–12 days for coracidia, 7–15 days for procercoids, and several months for pleurocercoids. Pleurocercoids of *Schistocephalus* require 4–6 months while those of *Ligula* and *Diphyllobothrium* require 10–14 months. Adults mature rapidly in the avian host and survive for 2–12 days (see McDonald 1969a).

CLINICAL SIGNS

Cestode infections in wild birds are normally asymptomatic, but when clinical signs are present, they are nonspecific and similar to those reported in poultry (Reid 1983). Emaciation, weakness, and occasionally diarrhea and hemorrhage may be accompanied by changes in posture or locomotory and feeding behavior.

Interpretation of clinical signs may be confounded by the common occurrence of mixed helminth and protozoan infections in wild birds. Experimental studies have helped to identify signs that are associated with cestode infection. Weakness and inappetence have been documented in ducklings within 6–8 days after experimental infection with *G. dogieli*. These signs became worse with time, leading to anorexia and spasmodic head and limb movements by day 21 postinfection (PI) and death by day 30 PI (Kulukbaeva 1985; Egizbaeva and Kulukbaeva 1985). Similar signs have been reported in domestic ducklings infected with *Microsomacanthus collaris*, including difficulty in walking, an abnormal backward arching of the neck, and unusual huddling behavior (Gitter et al. 1974).

Among naturally infected wild birds, emaciation has been observed in common eiders infected with *Schistocephalus solidus* and *Lateriporus* sp. (Hario et al. 1992), in a Long-tailed Duck (*Clangula hyemalis*) infected with *G. cygni* (Heck 1969), in Short-tailed Shearwaters (*Puffinus tenuirostris*) (Nishigai et al. 1981) and Little Penguins (*Eudyptula minor*) (Obendorf and McColl 1980) infected with *Tetrabothrius* spp., and in an Arctic Loon infected with *Ligula intestinalis* (Bayle 1983). In the latter case, the loon also displayed generalized weakness and diarrhea. A few species of cestodes in ducks and grebes can cause varying degrees of hemorrhage where they attach to the intestinal mucosa that might be detected as bloody feces (Šlais 1961; Heck 1969; Boertje 1974). Unusual changes in feeding behavior have also been reported in Common Eiders infected with *S. solidus*. Eiders normally dive for food in deep water, but heavily infected individuals fed in shallow water by tipping up like dabbling ducks until they died (Hario et al. 1992).

PATHOLOGY OF ADULT CESTODES

Adult cestodes may cause damage to the gizzard lining (*Gastrotaenia*), intestinal blockage, localized damage to the intestinal wall at the site of attachment, or irritation of the intestinal lining. Inflammation is the most common host response to cestode infection and appears to be most intense where prolonged contact occurs between the host and parasite.

Cyclophyllidea

Gizzard Infections: *Gastrotaenia*

Species of *Gastrotaenia* normally live under the softer areas of the gizzard lining in waterfowl but are also found under the grinding plates. The lesions appear as roughened, friable, eroded areas on the lining and are usually discolored. Ecchymoses and necrosis are usually present around the edges of the grinding plates (Heck 1969; Egizbaeva and Erbolatov 1975). The gizzard muscles are weakened, portions of the lining may detach from the underlying tissue, and necrosis of the glandular layer occurs (Willers and Olsen 1969; Kulukbaeva 1985). The area occupied by the cestodes is depressed, inflamed, and may show signs of hemorrhage (Heck 1969; Willers and Olsen 1969).

Embedded worms cause extensive necrosis of the lining that is accompanied by atrophy of the underlying glandular area and altered mucous secretion (Egizbaeva and Erbolatov 1975). Areas surrounding the cestodes and lesions become inflamed and are infiltrated by polymorphonuclear leukocytes, lymphocytes, and eosinophils that may be accompanied by localized hemorrhage (Heck 1969; Willers and Olsen 1969; Egizbaeva and Erbolatov 1975; Kulukbaeva 1985).

Intestinal Infections

Potentially fatal intestinal occlusion has been reported in waterfowl infected with various hymenolepidid

species (Czaplinski 1965; Maksimova 1972; Kulachkova 1973; Gitter et al. 1974; Basu et al. 1982), in Houbara Bustards infected with *Ascometra choriotidis* (Jones et al. 1996a), and in Wild Turkeys (*Meleagris gallopavo*) infected with *Metroliasthes lucida* (Ángeles Rebolloso et al. 2006).

A number of hymenolepidid species and *Choanotaenia infundibulum* produce enteritis in waterfowl (Basu et al. 1982; Schmidt et al. 1987) that may be accompanied by a distention of the intestine and an accumulation of hemorrhagic and/or mucous exudates (Basu et al. 1982). Thomas (1985) provided evidence that intestinal hypertrophy in Willow Ptarmigan infected with *Raillietina* sp. is positively correlated with the number of cestodes present. Various hymenolepidid species from ducks, *S. tenuicirrus* from grebes, and species of *Otiditaenia* from bustards cause inflammation in the intestinal lining (Šlais 1961; Kulachkova 1973; Boertje 1974; Basu et al. 1982; Jones et al. 1996a).

Effects of scolices. Scolices of several cyclophyllidean species penetrate deeply into the intestinal wall. *Parorchites zederi* and *S. tenuicirrus* produce large diverticulae in the intestine of penguins and grebes, respectively, that are visible on the serosal surface of the organ (Fuhrmann 1921; Boertje 1974; Cielecka et al. 1992). A similar condition caused by an unidentified cyclophyllidean has been reported from a Lesser Flamingo (*Phoenicopterus minor*) (Poynton et al. 2000). In each case, the diverticulum contained the scolex and a portion of the strobila. Diverticulae produced by *S. tenuicirrus* are the most complex and consist of four large vesicles averaging 15 mm in diameter (Boertje 1974). Scolices of *Schistotaenia scolopendra*, *Schistotaenia srivastavi*, and three species of *Paradilepis* also penetrate deeply into the intestinal wall of various grebes and cormorants (Baer 1940, 1959; Rausch 1970; Matta and Ahluwalia 1977; Karstad et al. 1982). Species of *Paradilepis*, including *Paradilepis scolecina*, produce nodules (1–2 mm) that are visible on the serosal surface of the intestine (Figure 14.2) (Matta and Ahluwalia 1977; Karstad et al. 1982).

The large scolices of *Schistotaenia* and *Paradilepis* cause extensive damage to the mucosal, submucosal, and muscular layers (Baer 1940; Rausch 1970; Boertje 1974; Matta and Ahluwalia 1977; Karstad et al. 1982) (Figure 14.3). The scolex may be surrounded by a thick fibrous capsule which, in the case of *P. scolecina*, consists of multinucleate giant cells and fibrocytes (Karstad et al. 1982). Leukocyte infiltration, inflammation, and hemorrhage have been reported at the attachment sites of *Schistotaenia* spp. (Rausch 1970; Boertje 1974).



Figure 14.2. Nodules on the intestine of a Great Cormorant (*Phalacrocorax carbo*) infected with *Paradilepis scolecina*. Reproduced from Karstad et al. (1982), with permission of the *Journal of Wildlife Diseases*.

Scolices of *Aploparaksis penetrans* have been reported to produce small nodules in various species of Charadriiformes (Spasskaya 1966). In this case, however, it is the tip of the rostellum rather than the scolex that produces the nodule. Smaller scolices produce less damage. Jones et al. (1996b) reported denudation of mucosal epithelium at the attachment site of *Hispaniolepis falcata* in Houbara Bustards and a hypergenerative response adjacent to it but found little response associated with attachment sites of other species. Scolices of *C. infundibulum* and three species of hymenolepidids produced necrotic foci in domestic ducks (Basu et al. 1982), whereas those of *Aploparaksis furcigera* and *Microsomacanthus parvula* in ducks produced a local inflammation dominated by eosinophils



Figure 14.3. Section through an intestinal nodule infected with *Paradilepis scolecina*. Reproduced from Karstad et al. (1982), with permission of the *Journal of Wildlife Diseases*.

and plasma cells (Šlais 1961). Varying degrees of hemorrhage have been reported in ducks infected with *A. furcigera* and *M. parvula* (Šlais 1961).

Effects of strobilae. The strobila represents virtually all the biomass of a cestode and much of it is in contact with the mucosal surface at any given time. Strobilae do not penetrate the mucosa or cause open lesions, but continuous contact with the mucosa causes irritation that may be exacerbated by the extension and contraction of the worm during normal activity.

The fragmentary information available on host responses to strobilae in wild birds is similar to what has been reported from poultry (Reid 1983; Padhi et al. 1986). Physical changes include desquamation, necrosis, and shortening of villi in ducks infected with *C. infundibulum* and various hymenolepidids (Basu et al. 1982; Kishore and Sinha 1989). Structural changes in Kori Bustards (*Ardeotis kori*) infected with *Otiditaenia conoideis* range from mild atrophy to collapse and fibrosis of the intestinal musosa, and unspecified damage to the mucosa has been described in Houbara Bustards infected with *O. conoideis* and *Hymenolepis falsata* (Jones et al. 1996a).

Inflammation of the intestinal mucosa is common in cestode infections in ducks (Basu et al. 1982; Schmidt et al. 1987) and in bustards (Jones et al. 1996a). In general, intestines of bustards infected with cestodes are inflamed but the degree of inflammation varies among host species. Infiltration of monocytes, lymphocytes, eosinophils, heterophils, and plasma cells occurs to varying degrees. This is accompanied in some cases by the proliferation of connective tissue and enlargement of lymph nodules (Basu et al. 1982; Kishore and Sinha 1989; Jones et al. 1996a). In Red-crested Bustards (*Eupodotis ruficrista*) infected with *Otiditaenia macqueeni*, small inflammatory nodules consisting of plasma cells, lymphoid cells, and clumps of hemosiderin-laden macrophages are present on the mucosa (Jones et al. 1996a).

Infections in abnormal sites. Cestodes rarely invade abnormal sites but when they do, they can produce irritation or atypical lesions (Wobeser 1974; McOrist 1989). Examples include abnormal development of *Cloacotaenia megalops*, a cloacal cestode of waterfowl, in the ureters of ducks (Wobeser 1974), abnormal development of *C. infundibulum*, an intestinal parasite of species of Galliformes and Passeriformes, in the gizzard lining of Barn Owls (*Tyto alba*) (McOrist 1989), and development of immature *F. fasciolaris* in the gizzard muscles of a domestic duck (Mondal and Baki 1989). Lesions in ducks infected with *C. megalops* include enlargement and inflammation of the ureters, flattening and atrophy of the epithelial lining, and a diffuse infiltration of heterophils in the walls of the ureters and in the kidneys (Wobeser 1974). Barn Owls infected with *C. infundibulum* develop conspicuous lesions in the gizzard lining that have attached cestodes and dark, bloody material (McOrist 1989).

Tetrabothriidea

Little is known about host responses to infection with adult tetrabothriid cestodes. Nishigai et al. (1981) reported large numbers of *Tetrabothrius skoogi* in emaciated Short-tailed Shearwaters that died of apparent malnutrition and anemia off the coast of Japan, but gross and microscopic lesions were not reported.

Diphyllobothriidae

Intestinal distention and occlusion have been reported in very intense infections with diphyllobothriid cestodes. As many as 340 adult *S. solidus* were recovered from a duck with a fatal infection (Callot and Desportes 1934). Both intestinal distention and occlusion were present in Common Eiders that died with intense infections of this parasite (Grenquist et al. 1972; Persson et al. 1974). Death of a Marabou Stork (*Leptoptilos crumeniferus*) chick resulted from an intestinal infection of *L. intestinalis* (Betke et al. 2003).

Pathology of Larval Cestodes

Cyclophyllidea

Larval cestodes develop in the body cavity, internal organs, or musculature. Transitory damage occurs during larval migration from the gut and chronic lesions may develop in tissues where larvae become established (Kugi 1983; Roberts and Janovy 2005; Toplu et al. 2006).

Tetrathyridia (*Mesocestoides*) usually occur in the body cavity (Kugi 1983; Millán et al. 2003), but may also infect visceral organs when present in large numbers (Kugi 1983). Generally, there are no visible reactions; however, Toplu et al. (2006) reported nonsuppurative granulomatus pleuritis and peritonitis and a yellow, serous fluid containing tetrathyridia in the abdominal and thoracic cavities of a captive peafowl. Granulomas containing degenerating tetrathyridia were also present on the parietal and pleural peritoneum. These were surrounded by macrophages, lymphocytes, and eosinophils. Additional granulomas containing tetrathyridia were present in the muscles of the proventriculus.

The tetrathyridia of *Mesocestoides* have been reported in livers of Green Pheasants (*Phasianus*

versicolor) (Kugi 1983). Livers were congested and a slight infiltration of lymphocytes was present in the lungs. No changes were evident in hepatic cells despite the presence of parasites in the liver parenchyma. Similarly, no apparent lesions were observed in Red-legged Partridges (*Alectoris rufa*) infected with *Mesocestoides* (Millán et al. 2003).

Diphyllobothriidae

Pleurocercoid larvae have been found in the body cavities of a variety of birds (Raethel 1977; Kuntz 1979; Lafuente et al. 1999). Raethel (1977) described fatalities in captive Pink-backed Pelicans (*Pelecanus rufescens*), a Brown Pelican (*Pelecanus occidentalis*), a Double-crested Cormorant (*Phalacrocorax auritus*), a Guanay Cormorant (*Phalacrocorax bougainvillii*), and a Wood Duck (*Aix sponsa*) infected with migrating pleurocercoids of *L. intestinalis*. Partial penetration of the intestine was seen in some individuals and a pleurocercoid had penetrated the abdominal wall of one bird. A putrescent fibrinous serositis was present and what appeared to be intestinal contents were found in the body cavity. Fibrous deposits were present on the serosa and in most birds fibrous lesions had fused several intestinal loops together. Birds are normally the definitive hosts of *L. intestinalis*, and the presence of *Ligula* pleurocercoids in the body cavity is unusual. Larvae found by Lafuente et al. (1999) were not identified; those reported by Kuntz (1979) were believed to be species of *Spirometra*, which are known to use birds as second intermediate hosts.

No pathology was associated with infections of *Spirometra* pleurocercoids in the body cavity of various birds; however, several pleurocercoids were found embedded in subcutaneous connective tissue of the breast muscles (Kuntz 1979), indicating tissue migration by some of the pleurocercoids in these birds.

DIAGNOSIS

The presence of eggs, gravid proglottids, or cestode fragments in the feces of the host is diagnostic for cestode infection. Although it is not possible to identify cestodes to species in this manner, cyclophyllidean and tetrabothriidean cestodes can be distinguished from diphyllobothriid cestodes on the basis of egg morphology (Figure 14.1) and proglottid structure (Table 14.1). Diphyllobothriid eggs have hard operculate shells (Figure 14.1), but may be difficult to distinguish from eggs produced by trematodes. The gravid proglottids of some families are sufficiently distinct to permit identification to that level.

Identification of cestodes to order, family, and generic levels requires microscopic study of adult specimens and evaluation of the morphology of the scolex and reproductive systems (Table 14.1). Identification to species level requires evaluation of the presence or absence of the rostellar hooks on the scolex and their number, shape, and size. The position, size, and shape of components of the male and female reproductive systems are also important, including the number of testes and their spatial relationships within the mature proglottid. Keys to the generic level are available in Schmidt (1986) and Khalil et al. (1994). Schmidt included species lists for each genus. Unfortunately, few species keys are available. Existing keys, taxonomic revisions, and descriptions of new species can be located through Helminthological Abstracts or similar abstracting services.

IMMUNITY

There is little evidence to suggest that birds develop immunity to cestode infections. Chickens with existing infections of *Raillietina laticanalis* are not immune to reinfections (Ueta and Avancini 1994). No comparable data exist for wild hosts, although parasitological surveys and studies of cestode life histories indicate that repeated infections occur. Juvenile and adult birds are typically infected by the same species, suggesting that individuals infected as juveniles are reinfected as adults. Many cestode species found in waterfowl are transmitted by copepods and ostracods (McDonald 1969a) that can support only one or two larvae because of their small size. Birds with cestode populations in excess of this would have to ingest multiple intermediate hosts, most likely at different times. It is not uncommon to find mature and immature specimens of the same species in a wild bird, which indicates that cestode recruitment is a continuous process, at least at certain times of the year. Finally, aquatic birds in particular are normally infected by multiple species of cestodes, some of which are transmitted by different species of intermediate hosts (Bush and Holmes 1986; Stock and Holmes 1987; Bush 1990). Acquisition of different components of the cestode community likely occurs over a period of time, indicating that prior infections provide little or no immunity to superinfection with the same or different species. Collectively, these observations argue against the presence of an effective immune response to cestode infection in birds.

PUBLIC HEALTH CONCERNS

Adult cestodes found in birds cannot be transmitted directly to humans and do not pose a health threat. However, the larval stages of *Mesocestoides* and *Spirometra* can infect humans when consumed in raw or undercooked meat (Beaver and Jung 1985; Roberts

and Janovy 2005). The few case reports found in a search of the Helmintological Abstracts database from 1990 to the present, however, suggest that human infections with either parasite are relatively rare.

Ingestion of larval *Mesocestoides* can result in the establishment of adult cestodes in the intestine (Beaver and Jung 1985). Ingestion of *Spirometra* pleurocercoids results in their transfer, without further development, to a new host individual. Infections with this type of pleurocercoid are known as sparganosis (Roberts and Janovy 2005). In sparganosis, the parasite may localize in the body cavity among the viscera or it may migrate to organs or muscles. It frequently appears as a lump under the skin that is usually treated surgically. A more serious situation may occur if the larva invades an internal organ and proliferates, in which case it can cause extensive damage (Roberts and Janovy 2005).

DOMESTIC ANIMAL HEALTH CONCERNS

Transmission of cestodes from wild to domestic birds requires a wild reservoir host to provide a source of cestode eggs and intermediate hosts to support development of larval stages of the parasites. The practice of raising ducks and geese on reservoirs and natural wetlands frequented by wild waterfowl can lead to outbreaks of cestode infection, disease, and losses in domestic waterfowl (Gitter et al. 1974). Losses of domestic waterfowl as a result of infection with *G. dogieli* have been documented in Eastern Europe and the former USSR (Egizbaeva and Erbolatov 1975). Similarly, contamination of local ponds by wild anatids can also lead to outbreaks in captive waterfowl in zoological collections (Kotecki 1970). Cestode diversity in captive birds at the Warsaw Zoo was less than that in wild birds, presumably because a suitable range of intermediate hosts was not present.

Cestode larvae develop rapidly and may be infectious to captive or domestic waterfowl in fewer than 2 weeks after a wetland is contaminated by wild birds (McDonald 1969a). Once established, the parasites can be maintained locally by domestic and wild ducks alike. Passerines are often infected with common parasites of poultry such as *C. infundibulum* and *Raillietina echinobothrida* (Reid 1983; Ibrahim 2006) and are a potential source of infection for chickens. In contrast, Zetterman et al. (2005) found two species of cestodes in wild Greater Rheas (*Rhea americana*), but none in captive birds. Other parasites were present in both groups, suggesting that either the intermediate hosts for the cestodes were absent or the area had not been contaminated with eggs.

Diphyllobothriid cestodes found in birds use fish as second intermediate hosts. The pleurocercoids of *Ligula* (Cozma and Friciu 1997; Loot et al. 2001; Heckmann 2005) and *Diphyllobothrium* (Rodger 1991) are pathogenic to fish. Infected birds, attracted to freshwater aquaculture facilities, can easily transmit infections to farmed fish if suitable copepod intermediate hosts are available. Pleurocercoid infections are common in farmed fish (Kititsyna and Nikitenko 1986; Håstein and Lindstad 1991; Cozma and Friciu 1997) and are a common cause of disease and mortality in these intermediate hosts (Kurovskaya 1993; Rhakonen et al. 1996). Pleurocercoids of some *Diphyllobothrium* species may be transmissible to humans (Roberts and Janovy 2005) when consumed in raw or poorly cooked fish and pose a potential health threat.

WILDLIFE POPULATION IMPACTS

The potential influence of parasites on host population dynamics is difficult to assess (Peterson 2004). Adult cestodes are not generally considered pathogenic or a threat to avian populations under normal conditions (Cornwell and Cowan 1963; Wobeser 1981; Harradine 1982; Thomas 1985; Greve 1986; Sasseville et al. 1988; Purvis et al. 1998; Delahay 1999; Haukos and Neaville 2003). Absence of clinical signs makes it difficult to detect infected birds. Sick birds usually die unnoticed and, if they are found, they are usually infected with a variety of parasites, making it impossible to attribute the condition to a specific agent. Reports of mortality in the field need to be interpreted with caution, particularly if mixed infections are involved. For example, cestodes have been associated with emaciation and starvation of large numbers of birds during sudden cold snaps (James and Llewellyn 1967; Jaramillo and Rising 1995), but their role in these deaths remains unresolved.

Parasitism by cestodes may affect reproduction and mate selection, with corresponding impacts on population size. Mortality in breeding Willow Ptarmigan infected with *Hymenolepis microps* increased with intensity of infection, with subsequent reductions in the annual growth rate of the host population (Holmstad et al. 2005). Similarly, female Common Eiders with heavy cestode infections may forgo breeding (Hario et al. 1992) rather than produce smaller clutches that may have to be abandoned later. Eiders reach sexual maturity later than most ducks, produce comparatively small clutches, and do not renest if the first one is lost. In species such as these, reduction either in survival or in the number of nesting females could have a significant impact on population numbers at least at the local level.

Infection with cestodes may also affect plumage quality and sexual ornamentation, with subsequent effects on mate selection. For example, Bar-tailed

Godwits (*Limosa lapponica*) in good body condition may undergo a partial molt during spring migration at staging points in the Wadden Sea, while lighter birds do not (Piersma et al. 2001). Heavy, well-ornamented birds replace some of their contour feathers and display more extensive breeding plumage than do nonmolting birds. Birds undergoing the molt had fewer cestodes and, among females, quality of the breeding plumage was inversely associated with intensity of infection.

TREATMENT AND CONTROL

Wild birds brought in from the field for propagation or relocation programs are usually infected with cestodes. Stress associated with capture or confinement may exacerbate the effects of cestode infections (Jones et al. 1996a). Treatment recommendations vary but in general niclosamide (Yomesin) has been recommended for species of Gruiformes (Carpenter 1986), Anseriformes (except for geese) (Humphries 1986), Falconiformes, and Strigiformes (Ward 1986). Praziquantel is effective in species of Columbiformes (Zwart 1986), starlings and other Sturnidae (Letcher 1986), and in bustards (Jones et al. 1996a). Both niclosamide and flubendazole are effective in controlling infection in captive flocks of bustards (Jones et al. 1996a). Fockema et al. (1985) successfully treated Ostriches infected with *Houttuynia struthionis* with fenbendazole.

There is no practical way to control cestode infections in wild birds. Control measures require a disruption in life cycles by reducing or eliminating contact with potential intermediate hosts. This may be possible on a limited scale for captive flocks but is not feasible on a scale that would affect wild populations.

MANAGEMENT IMPLICATIONS

There are no effective management options available to control cestode infections in wild birds. The vagility of these hosts ensures that the parasites will be spread widely in local habitats and that those of migratory species will be spread over even broader geographic areas. Birds to be transported into new areas either as captives or for release should be treated for cestodes prior to shipment to reduce the possibility of introducing novel species. Similarly, the cestode fauna of local birds should be studied before restoration projects are undertaken to access potential risks to translocated or introduced species (Kocan et al. 1979).

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15
Acanthocephala

Dennis J. Richardson and Brent B. Nickol

INTRODUCTION

Worms of the phylum Acanthocephala (Greek: *akantha*, spine or thorn + *kephale*, head) are known as spiny-headed or thorny-headed worms due to the nature of their holdfast organ, called a proboscis. Acanthocephalans are dioecious pseudocoelomate worms remarkably adapted to a parasitic lifestyle in that there is no mouth or digestive system. Worms absorb nutrients directly through their integument. Adult acanthocephalans vary greatly in size from a few millimeters to over 10 cm long, depending on species, and occur exclusively in the vertebrate small intestine. All acanthocephalans exhibit an indirect life cycle in which the vertebrate definitive host becomes infected by ingesting larvae, known as cystacanths, contained in the hemocoel (body cavity), of an arthropod intermediate host.

Although they are capable of causing extreme pathology and death and may be responsible for epizootic outbreaks under certain circumstances, by and large, acanthocephalans cause little overt pathology in their avian hosts.

SYNONYMS

Acanthocephalosis, Acanthocephaliasis.

HISTORY

Acanthocephalans were first recognized from the intestine of eels by Redi (1684). Since then approximately 1,100 species of acanthocephalans have been described (Golvan 1994), with approximately 400 species being recognized from birds. A concise history of the study of acanthocephalans can be found in Amin (1985).

HOST RANGE AND DISTRIBUTION

Animals of all vertebrate classes serve as definitive hosts for acanthocephalans. Bony fishes are the most parasitized group and reptiles are the least. Birds harbor more species than do mammals, which in turn host more species than do amphibians.

Species of acanthocephalans from avian hosts are mainly represented by a few broadly distributed genera and are harbored by birds of relatively few taxonomic orders. Waterfowl (Anseriformes) are the most heavily parasitized group of birds. Species of the genus *Corynosoma* and *Polymorphus* are the most common forms in waterfowl. Acanthocephalans of the genera *Arhythmorhynchus* and *Plagiorhynchus* are the principle forms in shorebirds (Charadriiformes). Species of *Lueheia*, *Mediorhynchus*, and *Plagiorhynchus* comprise most of the acanthocephalans in perching birds (Passeriformes). Hawks (Falconiformes) and owls (Strigiformes) most frequently are parasitized by species of *Centrorhynchus*, *Sphaerirostris*, and *Oligacanthorhynchus*.

ETIOLOGY

In the most recent complete list, Golvan (1994) considered Acanthocephala to comprise slightly more than 1,100 valid species. The most important character from a taxonomic standpoint is the spiny holdfast structure or proboscis. The retractable and invaginable proboscis is used by the worm to attach to the intestinal wall of its vertebrate definitive host. Representative proboscides of acanthocephalans parasitizing avian hosts are shown in Figure 15.1. Basic acanthocephalan anatomy is shown in Figure 15.2. The review by Miller and Dunagan (1985) should be consulted for a more comprehensive account of functional morphology. Starling (1985) and Taraschewski (2000) reviewed nutrition and metabolism of Acanthocephala.

EPIZOOTIOLOGY

Acanthocephalan species for which life cycles have been confirmed require vertebrates for definitive hosts and arthropods as intermediate hosts. Schmidt (1985) provided a summary of known life cycles. Adult

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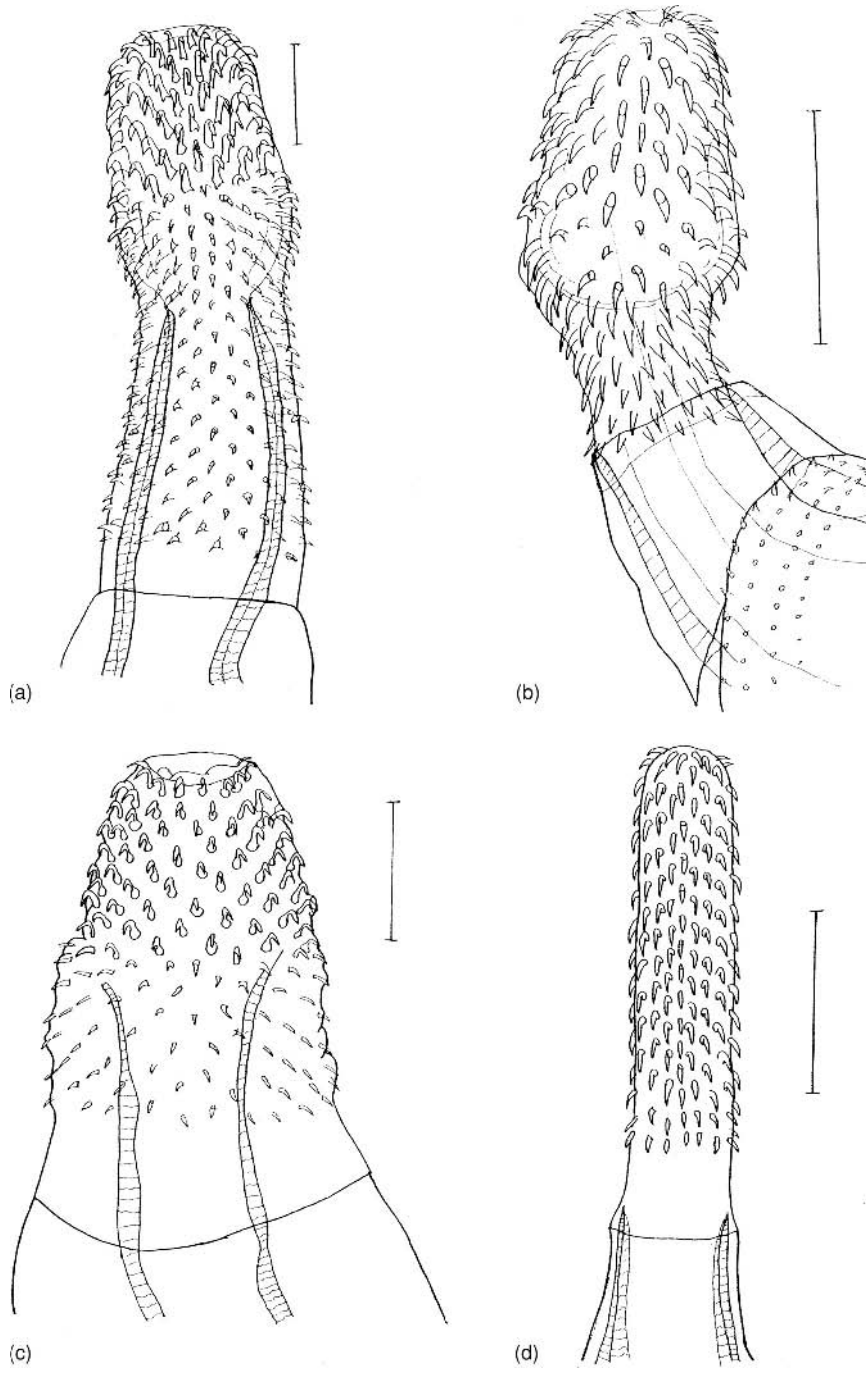


Figure 15.1. Proboscides of some common acanthocephalans of birds. (a) *Centrorhynchus robustus*, an acanthocephalan of owls. Bar = 250 μm. Redrawn from Richardson and Nickol (1995). (b) *Polymorphus cucullatus* from a Hooded Merganser (*Lophodytes cucullatus*). Bar = 500 μm. Redrawn from Van Cleave and Starrett (1940). (c) *Mediorhynchus centurorum* from a Red-bellied Woodpecker (*Melanerpes carolinus*). Bar = 220 μm. Redrawn from Nickol (1969). (d)) *Plagiorhynchus cylindraceus* from an American Robin (*Turdus migratorius*). Bar = 1 mm. Redrawn from Schmidt and Olsen (1964).

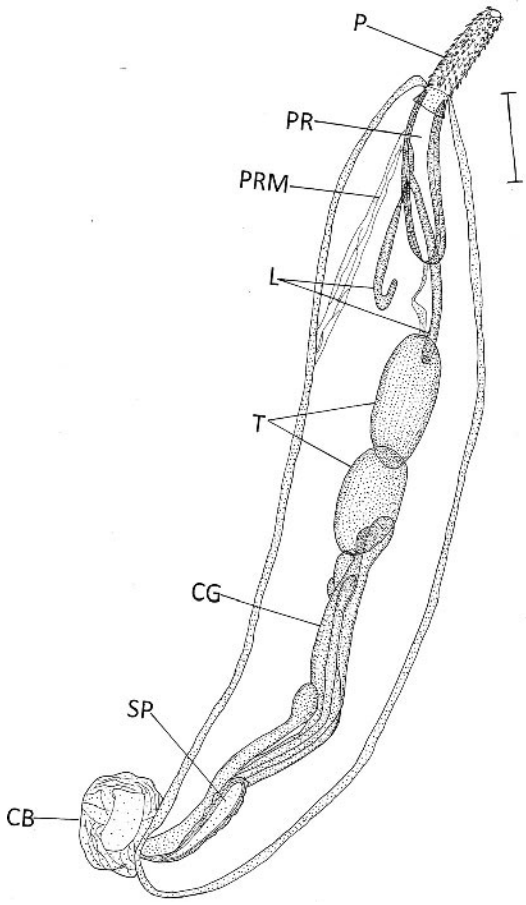


Figure 15.2. Male *Plagiorhynchus cylindraceus* from an American robin (*Turdus migratorius*). P, proboscis; PR, proboscis receptacle; L, lemnisci; T, testes; CG, cement glands; SP, Saefftigen's pouch; CB, copulatory bursa. Bar = 1 mm.

female worms release eggs that are passed in the feces of the definitive host. Only eggs exist outside of a host, free in the environment, and transmission from one definitive host to another requires that appropriate invertebrate intermediate hosts ingest eggs. A typical acanthocephalan life cycle is shown in Figure 15.3.

Intermediate hosts are known for only about 7% of the species that parasitize birds. Those with terrestrial life cycles usually have insects, frequently species of Coleoptera or Orthoptera, or terrestrial isopods for intermediate hosts. Decapods and microcrustaceans, usually species of Amphipoda or Isopoda, are intermediate hosts for those with aquatic life cycles.

A larval stage, the acanthor, emerges from the egg upon its ingestion by an arthropod. After penetration of the wall of the alimentary canal, the acanthor undergoes development within the body cavity of its intermediate host, ultimately achieving the final ontogenetic stage, the cystacanth, which is infective to potential definitive hosts. "Cystacanth" has achieved general usage as a name for the stage infective to a final host regardless of whether it is found in the arthropod intermediate host or in a vertebrate paratenic host (Van Cleave 1953).

No species of Acanthocephala has been demonstrated to require more than the arthropod intermediate host in order to develop infectivity to vertebrates. However, in the life cycle of some species, another vertebrate host occurs between the arthropod intermediate and vertebrate definitive host. In such hosts, cystacanths penetrate the intestinal wall and localize in mesenteries or visceral organs, but do not attain sexual maturity. Although such intercalated hosts may be required to complete transfer of acanthocephalans from intermediate hosts at the trophic level which potential definitive hosts feed, there is no evidence that they are essential for achievement of infectivity to the final host. The term "paratenic host" has attained wide usage for such animals in which ontogeny does not proceed (Baer 1951; Beaver 1969). An example of a life cycle of an acanthocephalan utilizing a paratenic host is shown in Figure 15.4.

Many, if not most, birds acquire acanthocephalans from an intermediate rather than a paratenic host. This is the route of transmission for a large number of species of *Corynosoma* and *Polymorphus* found in waterfowl and for species of *Plagiorhynchus* that occur in charadriiform and passerine birds. Species of *Mediorhynchus* are also transmitted to passerine birds in this manner.

Piscivorous birds frequently acquire acanthocephalans from fishes in their diet although other poikilothermic vertebrates are paratenic hosts for some species. *Southwellina hispida* has a broad geographical distribution in the Black-crowned Night Heron (*Nycticorax nycticorax*) and it occurs in mesenteries of fish, frogs, and snakes (Van Cleave 1925; Yamaguti 1935, 1939).

Amphibians and reptiles also serve as paratenic hosts for some acanthocephalan species that mature in flesh-eating birds. Species of *Centrorhynchus* and the related *Sphaerirostris* are well known as cystacanths in frogs, lizards, and snakes. Adults occur in raptors and other kinds of carnivorous birds. Golvan (1956) and Schmidt and Kuntz (1969) listed many of the definitive and paratenic hosts for species of these genera. Likewise several species of *Oligacanthorhynchus* occur as

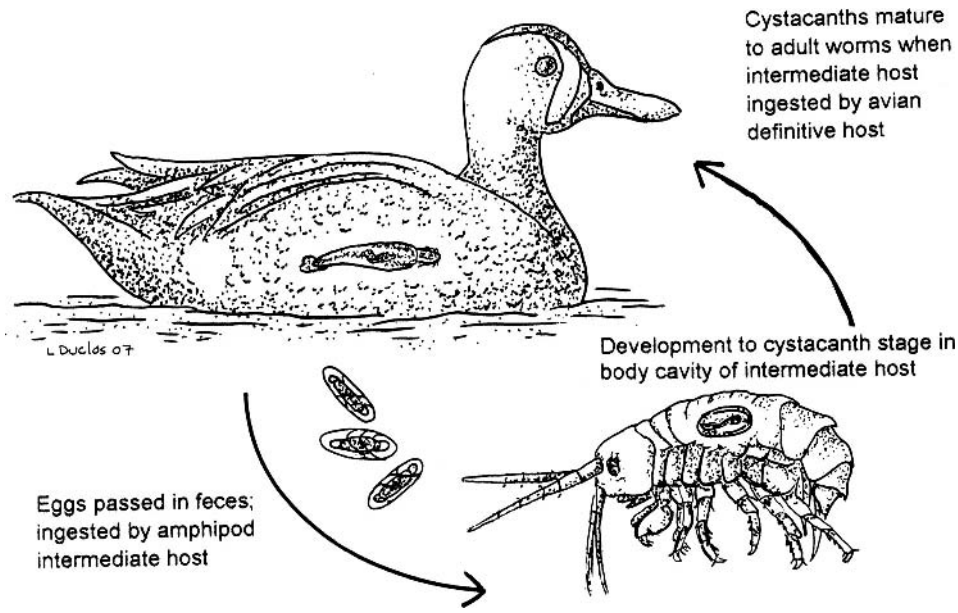


Figure 15.3. Life cycle of *Corynosoma constrictum*, a common acanthocephalan of waterfowl, particularly ducks, throughout North America and parts of South America. *Corynosoma constrictum* uses amphipods (*Hyallela azteca*) as intermediate hosts.

adults in birds of prey and the literature abounds with worldwide reports of oligacanthorhynchid cystacanths in the viscera of reptiles, usually snakes.

Acanthocephalans are found infrequently in extraintestinal sites in birds, but they seem to be important paratenic hosts only for some species of *Oncicola*. *Oncicola canis* and *Oncicola oncicola*, parasites of canine and feline definitive hosts in the Americas, have been found in the outer surface of the esophagus and crop of the Northern Bobwhite (*Colinus virginianus*), and subcutaneously in the musculature of domestic chickens (Cram 1931; Zeledon and Arroyo 1960). Australian and Asian species of *Oncicola* also use birds for paratenic hosts (Schmidt 1983).

CLINICAL SIGNS

Little is understood about clinical signs in birds infected with acanthocephalans. There are many reports of paralyzed and moribund birds with acanthocephalans (e.g., Jones 1928; Webster 1943; Holloway 1966; McOrist and Scott 1989). Many of these cases have involved American Robins (*Turdus migratorius*) infected with *Plagiorhynchus cylindraceus* (Figure 15.5). Birds with high-intensity infections are frequently emaciated and stunted (Hynes and Nicholas 1963).

PATHOGENESIS AND PATHOLOGY

It has long perplexed helminthologists that, in some cases, acanthocephalan infections of low intensity seem to have serious adverse effects on an infected animal. In other instances, clinical signs are absent in conspecific animals with infections of high intensity of the same species (Soulsby 1958). On the basis of their extensive study of *Polymorphus minutus* in domestic ducks, Hynes and Nicholas (1963) suggested that under normal circumstances infections build slowly and that density-dependent establishment might limit infections to subclinical levels. However, infections of high intensity with clinical consequences might result if an uninfected bird were suddenly exposed to a large number of acanthocephalans.

In contrast to mortality attributed to intense acanthocephalan infections, it is far more common to find equally intense infections in birds that show no sign of disease (Hynes and Nicholas 1963; Schmidt 1972; Moore and Bell 1983). There are very few studies that assist with assessing the importance of infections of low intensity or infections, even if intense, in which clinical effects appear to be lacking. The extent of pathogenesis is likely influenced by the nutritional status of the host (Holmes 1987) and by environmental stress (Grenquist 1970). Connors and

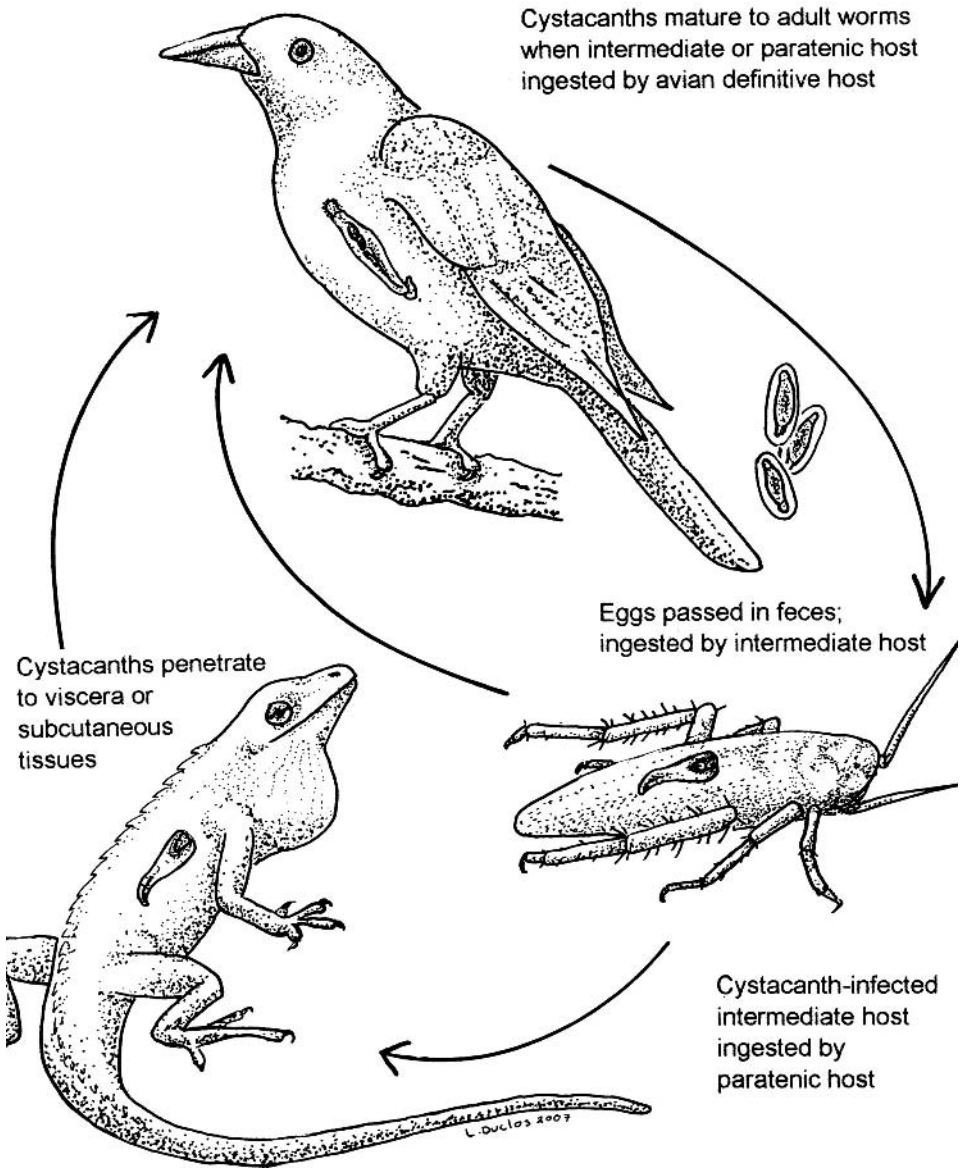


Figure 15.4. Paratenic transmission of *Lueheia inscripta*. Reptiles become infected by ingesting cystacanths contained within the body cavity of cockroaches. Within the reptilian paratenic host the cystacanths localize in the mesenteries or visceral organs but do not obtain sexual maturity. Passerine birds may become infected when they ingest cystacanths contained within paratenic hosts.

Nickol (1991) demonstrated that *P. cylindraceus* has a significant detrimental effect on the flow of food energy through infected European Starlings (*Sturnus vulgaris*). Both male and female starlings show reductions in standard metabolic rates as a result of infection, indicating that their basal metabolism and thermal regulatory abilities are altered. Infected male birds have an increased consumption and excretion of energy, and they average lower daily body weights than do uninfected males when they are temperature-stressed (Connors and Nickol 1991). There seems little question that subclinical infections can become serious in times of nutritional or environmental stress.



Figure 15.5. *Plagiorhynchus cylindraceus* in the intestine of an American Robin (*Turdus migratorius*) found frozen near Owensboro, Kentucky. The robin harbored 69 acanthocephalans. Courtesy of D. F. Oetinger.

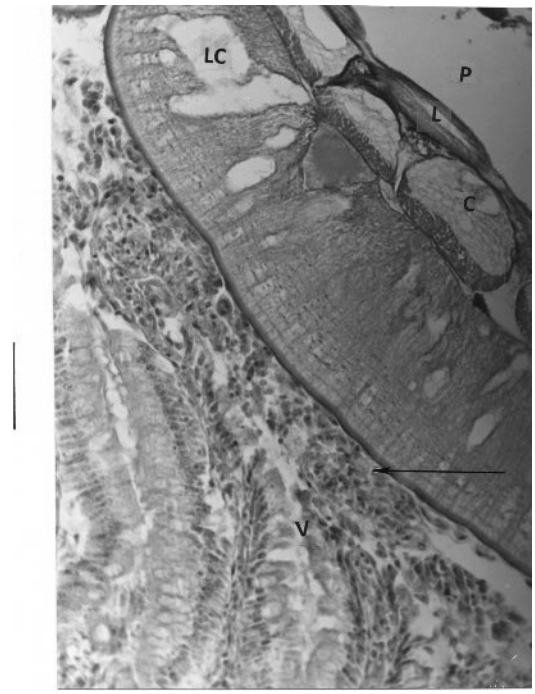


Figure 15.6. *Mediorhynchus centurorum* in the intestine of a Red-bellied Woodpecker (*Melanerpes carolinus*). The trunk of the worm has eroded villi, allowing blood vessels to leak. The resulting pus (arrow) contains blood cells, macrophages, polymorphonuclear cells, clotted fibrin, and damaged columnar epithelial cells. C, circular muscle of worm body wall; L, longitudinal muscle of worm body wall; LC, lacunar canal of worm tegument; P, pseudocoelom of worm; V, abraded villus of bird mucosa. Bar = 50 μm.

The significance of acanthocephalans in dying or dead animals (Figure 15.5) is difficult to interpret because the link is always circumstantial and because intensities are often no greater in affected animals than in conspecific individuals showing no adverse effect (Hynes and Nicholas 1963; Schmidt 1972; Moore and Bell 1983); thus, acanthocephalan infection at any intensity should be considered to have pathogenic potential.

Attachment of the acanthocephalan proboscis sometimes causes formation of fibrinous nodules on the serosal surface of the intestine. In Red-bellied Woodpeckers (*Melanerpes carolinus*), nodules that form around the proboscis, neck, and foretrunk of *Mediorhynchus centurorum* are frequently at least 4 mm long (Nickol 1969). Adult worms of most acanthocephalan species have not been reported to induce nodules in their hosts. Others, such as *P. minutus* in domestic ducks, may or may not induce nodule formation (Nicholas and Hynes 1958). Still others, such as *Profilicollis botulus*, seem always to cause nodules to form (Bishop and Threlfall 1974; Bourgeois and Threlfall 1982).

Upon necropsy, acanthocephalans occasionally are found protruding from the intestine into the coelom, having perforated the intestinal wall. In Western Bluebirds (*Sialia mexicana*) infected with *P. cylindraceus* and in Common Eiders (*Somateria mollissima*) and Mute Swans (*Cyngus olor*) infected with *P. minutus*, peritonitis resulting from perforation has been linked to mortality (Clark et al. 1958; Sanford 1978; Thompson-Cowley et al. 1979). Although perforation through acanthocephalan-induced nodules sometimes occurs (Bishop and Threlfall 1974), perforation apparently is independent of nodule formation (Thompson-Cowley et al. 1979).

Histological damage caused by acanthocephalans has been studied more extensively in fish and mammals (Bullock 1963; Chaicharn and Bullock 1967; Szalai and Dick 1987; Richardson and Barnawell 1995) than in birds, with a few notable exceptions (e.g., Nicholas and Hynes 1958; Petrochenko 1958; Schmidt 1963; Sanford 1978; Moore and Bell 1983; Taraschewski and Hofmann 1991). Along the trunks of *Mediorhynchus gallinarum* and *M. centurorum*, villi and basal glands of domestic fowl and woodpeckers, respectively, are compressed and eroded (Figure 15.6) in much the same manner as has been described for mammals (Nath and Pande 1963; Nelson and Nickol 1986; Richardson and

Barnawell 1995) and fish (De Buron and Nickol 1994) infected with acanthocephalans.

At sites of attachment of the proboscis, frequent microscopic changes for birds are acute, multifocal, necrotizing transmural inflammation and necrosis and ulcerative enteritis resulting from penetration of the intestinal wall by the proboscis (Bolette 1987). Chronic inflammation at the site where the proboscis attaches may cause fibrinous adhesions that bind viscera, reduce mobility of the gut, and cause emaciation (Bishop and Threlfall 1974).

DIAGNOSIS

Acanthocephalan infections are detected in live birds by observation of eggs in feces of infected animals. Acanthocephalan eggs are recognized by their characteristic membranes that enclose a spined acanthor (Figure 15.7a). Generic identification is possible from the eggs (Figure 15.7). Specific identification may be made by a specialist in many instances. Because acanthocephalan eggs do not float readily in standard floatation mixtures, sedimentation techniques are preferred. The ethyl acetate/formalin sedimentation procedure, developed by Ritchie (1948) and modified by Markell et al. (1999), works well.

Diagnosis can also be made by identifying worms obtained at necropsy. Lack of properly prepared specimens is one of the primary reasons for the paucity of information concerning acanthocephalans from wild birds. The bird should be examined as soon after death as possible and carcasses should not be frozen. The intestine should be removed and carefully dissected longitudinally. Proboscides of firmly attached acanthocephalans should be removed from the intestinal wall using insect pins or fine needles. Care must be taken not to cut or puncture the worm. For identification, the proboscis must be evaginated and the armature preserved intact. After removal from the intestine, all acanthocephalans should be held in tap water for at least 24 h to promote full evagination of the proboscis. If the proboscis fails to evaginate in tap water, worms may be placed in distilled water for an additional 24 h. After the period in water, worms may be fixed in a solution of 85% ethanol, formalin, and glacial acetic acid mixed at a ratio of 85:10:5 (Van Cleave 1953). After 24 h in glacial acetic acid, specimens may be stored in 70% ethanol. Richardson (2006) provided detailed information on preparing specimens for microscopic examination, and Richardson (2005) discussed how to properly assess proboscis morphometrics.

The most recent comprehensive keys for identification are those by Yamaguti (1963). McDonald (1988) provided a key helpful for identification of forms found in waterfowl. There are, however, few comprehensive taxonomic keys available for acanthocephalans found in birds. Most are limited to specific acanthocephalan groups or to individual taxa of birds. The original systematic literature is the most reliable means of specific identification.

PUBLIC HEALTH CONCERNS

None of the five acanthocephalan species reported from humans occur in birds (Counselman et al. 1989; Richardson 2003). It is doubtful that wild birds serve as reservoirs for human acanthocephalan infections.

DOMESTIC ANIMAL HEALTH CONCERNS

Although acanthocephalans rarely constitute an economic consideration in agricultural operations, three species that occur naturally in wild birds can infect domestic poultry. *Plagiorhynchus cylindraceus* (=*Plagiorhynchus formosus*), a cosmopolitan species found in passerine birds, is of occasional concern as a potential parasite of domestic chickens in the US. (Jones 1928; Holloway 1966). This species, common in American Robins and European Starlings, can infect many kinds of birds, including domestic fowl. *M. gallinarum* occurs in wild and domestic galliform birds throughout Asia and Africa (Schmidt and Kuntz 1977). Significant consequences, however, have not been reported for either of these acanthocephalan species.

Polymorphus minutus and the similar *Polymorphus magnus* have been implicated as important parasites of domestic ducks in Europe (Antipin 1956; Petrochenko 1958; Hynes and Nicholas 1963). These polymorphids are frequent parasites of anatid birds and can be transmitted from amphipod intermediate hosts to domestic fowl that have access to water frequented by wild waterfowl.

Mortality in birds at the National Aviary in Pittsburgh, Pennsylvania, was attributed to *Mediorhynchus orientalis* that apparently was present in an imported bird. The eggs of the parasite-infected species of cockroaches and cystacanths developed for transmission to other birds (Bolette 1990, 2000). Acanthocephalans are relatively specific for intermediate hosts. Consequently, importation of exotic species seldom results in continuation of the life cycle through transmission to other animals in the collection. Occurrences such as that in the Pittsburgh aviary apparently are exceptional, but they attest to the fact that domestic and captive animals are at potential risk from acanthocephalans present in wildlife.

WILDLIFE POPULATION IMPACTS

Dead or dying birds are often discovered with large numbers of acanthocephalans (Figure 15.5).

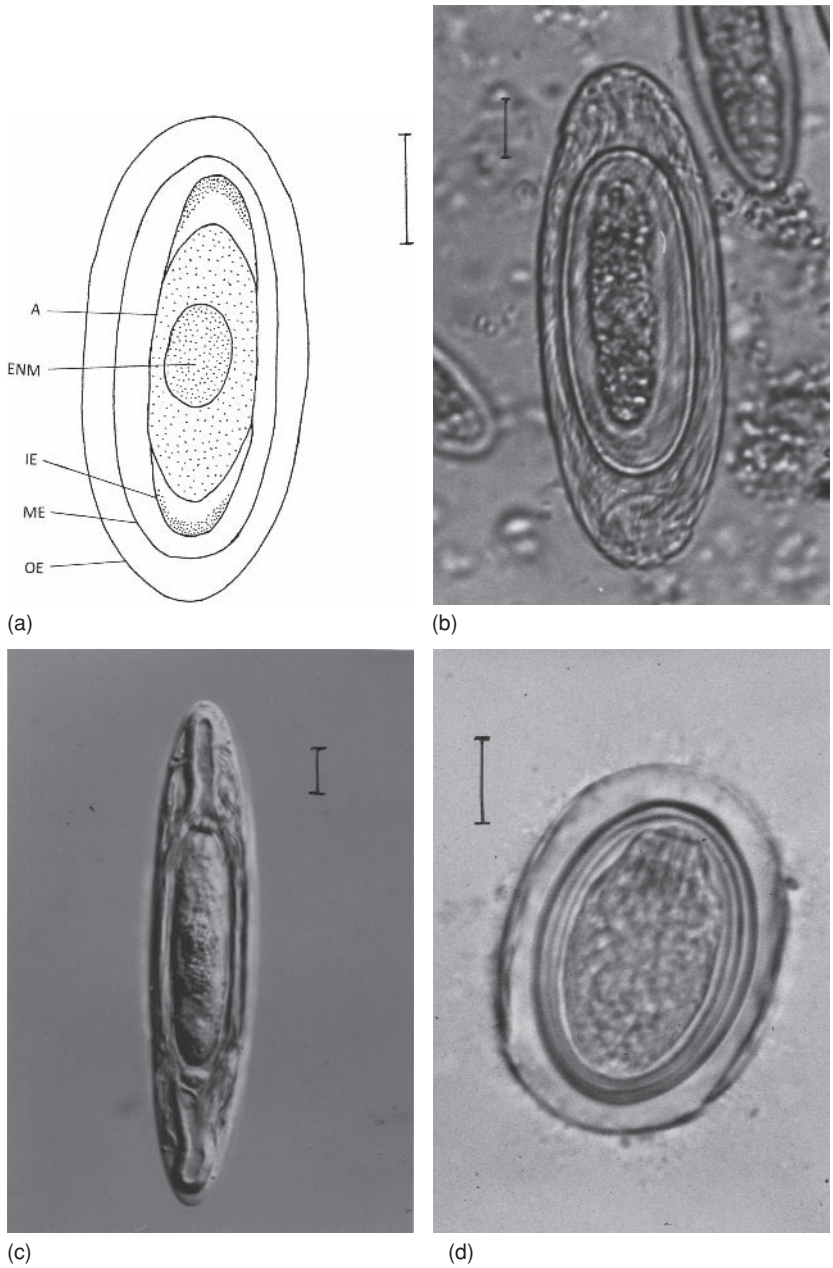


Figure 15.7. Eggs of some common acanthocephalans of birds. Scale bars = 10 μm. (a) Line drawing of egg of *Centrorhynchus microcephalus* from the intestine of a Groove-billed Ani (*Crotophaga sulcirostris*). Note the series of envelopes or membranes that surround the spined acanthor. Terminology follows Awachie (1966). A, acanthor; ENM, embryonic nuclear mass; IE, inner envelope of egg; ME, middle envelope of egg; OE, outer envelope of egg. (b) Egg of *Profilicollis botulus* that was removed from the body cavity of a gravid worm taken from a Common Eider (*Somateria mollissima*). Courtesy of D. W. T. Crompton. (c) Egg of *Polymorphus minutus* removed from the body cavity of a gravid worm taken from a domestic duck. Photograph courtesy of D. W. T. Crompton. (d) Egg of *Mediorhynchus grandis* removed from the body cavity of a gravid worm taken from a Western Meadowlark (*Sturnella neglecta*).

Thompson-Cowley et al. (1979) considered large numbers of *P. cylindraceus* to be a contributory cause of death among Western Bluebirds. Perry (1942) found up to 1,842 specimens of *Profilicollis altmani* in dead or dying Surf Scoters (*Melanitta perspicillata*) and White-winged Scoters (*Melanitta fusca deglandi*). Up to 3,500 specimens of *P. minutus* occurred in Common Eiders found dead during an epizootic in Finland (Itämies et al. 1980).

Acanthocephalan-induced epizootics with extensive mortality occur infrequently. Among birds, populations of Common Eiders seem especially vulnerable to epizootics that are usually caused by *P. botulus* and occasionally by *P. minutus*. Liat and Pike (1980) reviewed reports of many of these occurrences throughout the Northern Hemisphere.

Populations of swans also experience occasional decimation due to acanthocephalan infections. Large numbers of the acanthocephalan species *P. minutus* and *Filicollis anatis* were considered contributors to increased mortality in a flock of Mute Swans in central Scotland (Pennycott 1998). An earlier epizootic caused by *P. magnus* and *Polymorphus mathevossianae* led to loss of 40% of the cygnets inhabiting a lake in the Kurgal'dzhin Nature Reserve in Russia (Maksimova 1972). The Scottish and Russian epizootics followed drastic reductions in water levels that probably concentrated intermediate hosts and let to unusually intense infections.

Acanthocephalans that normally occur in birds may pose a threat to other kinds of wildlife when feeding activities cause exposure to large numbers of cystacanths. Mortality in California sea otters (*Enhydra lutris*) due to acanthocepalan-induced peritonitis has been reported in Monterey, California (Thomas and Cole 1996; Mayer et al. 2003). Otters most likely become infected by ingesting the infective cystacanths of *P. altmani*, normally a parasite of shore birds, in a sand crab (*Emerita analoga*) intermediate host (Nickol et al. 2002). Presence of gravid females in the body cavity of otters observed by Richardson (unpublished data) suggests that otters may also become infected through postcyclic transmission (ingestion of adult worms harbored in the intestine of a prey animal) while feeding on infected birds. Predation of seabirds by sea otters is rare but appears to have increased in recent years (Riedman and Estes 1988). Significant climatic events, such as *El Nino* and global warming, might lead to changes in feeding behavior, alter the normal cycles of transmission between parasites and hosts, and lead to greater frequency of aberrant pathogenic infections.

In at least one instance, acanthocephalans of birds may have commercial impact on an intermediate host population. *Southwellina dimorpha*, an acanthocephalan of the White Ibis (*Eudocimus albus*) and Whooping Crane (*Grus americana*) uses the commercially important cultured red crawfish (*Procambrus clarki*) as an intermediate host. Although infrequently reported, *S. dimorpha* may occur in high enough prevalence in crawfish in the southeastern US to have a commercial impact (Lantz 1974; Richardson and Font, 2006).

TREATMENT AND CONTROL

Although many courses of chemotherapy have been credited with efficacy in individual instances (Petrochenko 1958; Hynes and Nicholas 1963; Goldsmid et al. 1974; Counselman et al. 1989; Richardson 2003), no satisfactory treatment for acanthocephalan infection is known. Prevention depends on keeping birds away from environments that harbor infected intermediate hosts (Nicholas and Hynes 1958, Sanford 1978) although such control for wild birds seems impractical.

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16
Eustrongylidosis

Marilyn G. Spalding and Donald J. Forrester

INTRODUCTION

Eustrongylidosis is a disease of piscivorous birds caused by infection with a large dioctophymoid nematode of the genus *Eustrongylides*. Although a large number of species have been described, only three are considered valid at present: *Eustrongylides tubifex*, *Eustrongylides excisus*, and *Eustrongylides ignotus* (Measures 1988a). Only *E. ignotus* seems to cause significant disease when infecting herons and egrets. When infected fish containing larval stages of *E. ignotus* are consumed by herons, egrets, and long-legged wading birds, the parasites perforate the stomach wall and cause severe tubular fibrinous to fibrous peritonitis. Young birds are particularly sensitive and often die from hemorrhage or secondary bacterial infection. Older birds may survive infections of low intensity, but subsequently develop chronic peritonitis.

Eustrongylidosis is the most commonly documented cause of epizootic mortality in nestling wading birds in some areas. Up to 80% of nestlings in some locations in Florida have been documented to have died directly or from complications related to infection with this parasite (Spalding et al. 1993). Eutrophication of foraging sites has been associated with increased prevalence of eustrongylidosis in both nestlings and adult birds (Spalding et al. 1993).

Morbidity and mortality is less common with infections of *E. tubifex*, although this species can cause a nodular mass in the proventriculus of mergansers. Morbidity and mortality have not been reported in cormorants infected with *E. excisus.*

SYNONYMS

Verminous peritonitis, eustrongylidiasis.

HISTORY

Epizootics of eustrongylidosis were first reported in the US in the 1970s. Large numbers of herons and egrets died at a colony in Delaware in 1976 (Wiese et al. 1977) followed by additional epizootics in Louisiana and Florida (Roffe 1988; Spalding et al. 1993). Individual bird mortalities, however, involving Great Blue Herons (*Ardea herodius*) and Black-crowned Night-Herons (*Nycticorax nycticorax*) were noted before that in the Washington, DC, area (Chapin 1926; Cram 1933).

These parasites were first known in fish and were subsequently shown to be the same as those infecting piscivorous birds (Leuckart 1868). Interest in these parasites increased when outbreaks in Romania in 1927 decreased the value of commercial fish collected from freshwater lakes. Ciurea (1938) was able to demonstrate through experimental infections that larvae from these fish could infect cormorants. This led to early management suggestions that the parasite could be controlled by removing piscivorous birds and draining and refilling lakes.

More recent work has been done to resolve the taxonomy (Measures 1988a), the life cycle (Coyner et al. 2003a), and to uncover relationships between nutrient pollution and epizootic outbreaks of disease (Spalding et al. 1993; Coyner et al. 2002).

HOST RANGE AND DISTRIBUTION

The distribution of *Eustrongylides* spp. in both fish and birds is worldwide and includes both temperate and tropical climates (Figure 16.1). Remarkably, reports of its occurrence are spotty in spite of the large size and bright red color of the parasites. Reports of larval worms from fish intermediate hosts are much more common than those from avian definitive hosts where adult, sexually reproducing worms are found, undoubtedly because of the economic importance of the fish as food for humans. Species-specific distribution information is limited by confusion over identification of the adult parasites in the definitive avian host, identification of larvae in intermediate or paratenic hosts, and complicated by occurrence of multiple species in the same definitive host (Jagerskiold 1909b, Karmanova

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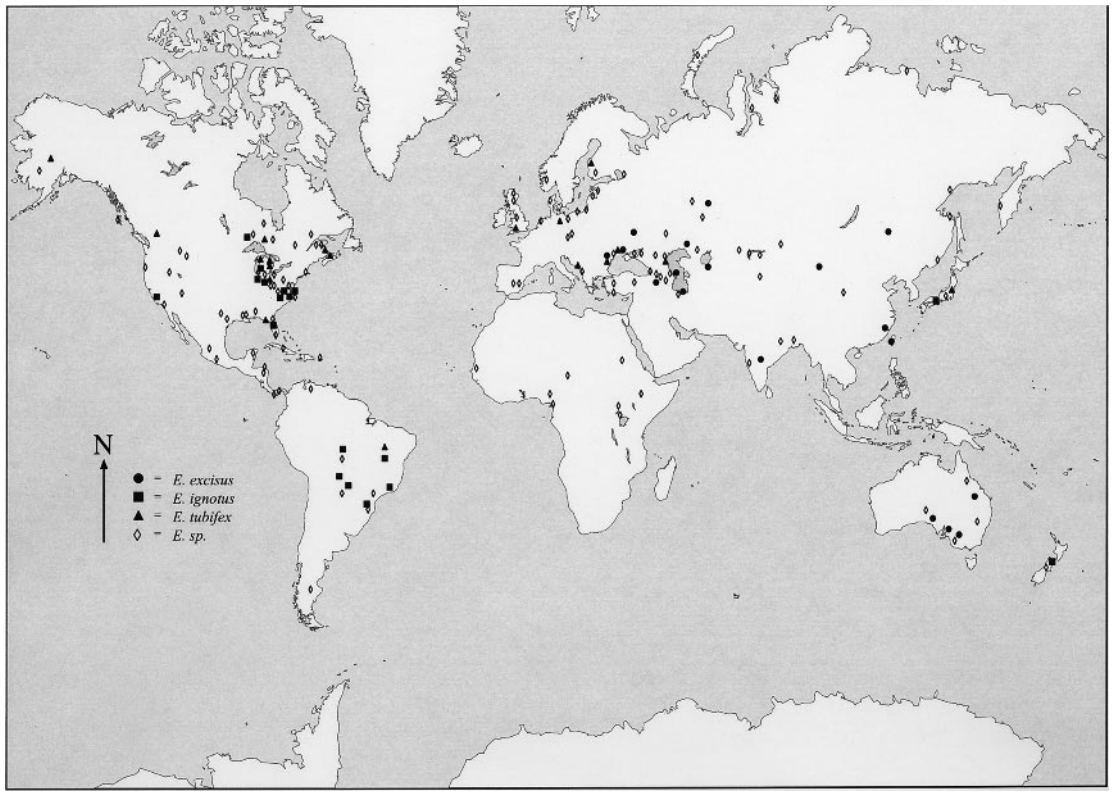


Figure 16.1. Map illustrating the distribution of *Eustrongylides* spp. in fish and birds throughout the world. When known, the species is indicated. Square denotes *E. ignotus*; triangle, *E. tubifex*; circle, *E. excisus*; diamond, *Eustrongylides* sp.

1986). *Eustrongylides ignotus and E. tubifex* are found most frequently in the Americas and Europe, whereas *E. excisus* is most common in Asia.

The geographic locations of infected birds are listed in Table 16.1 and include such diverse groups as herons, egrets, spoonbills, penguins, cormorants, coots, eagles, ducks, geese, gulls, and even some passeriform birds. The definitive hosts of *E. ignotus* appear to be limited to species of Ciconiiformes of the family Ardeidae (Spalding and Forrester 1993), even though parasites and lesions are found in other Ciconiiformes and Pelecaniformes. For example, although infections are found in White Ibis (*Eudocimus albus*) and Roseate Spoonbills (*Platalea ajaja*) (family Threskiornithidae), only larval stages have been reported.

The range of *E. ignotus* includes the Americas, from Ontario, Canada, to Brazil and from the East Coast of the US to California. There is a single verified record from a Little Pied Cormorant (*Phalacrocorax melanoleucos brevirostris*) in New Zealand (Measures 1988a). Great Blue Herons in Ontario, Canada, were infected with both *E. tubifex* and *E. ignotus* (Measures 1988c); therefore, species of Ardeidae appear to be susceptible to infections with more than one species of *Eustrongylides*. The failure to find *E. tubifex* in piscivorous birds in Florida indicates that there may be regional differences in the distribution of the two species (Spalding et al. 1993). It is possible that locations where transmission occurs are more geographically limited than suggested by definitive host records because of migratory behavior of these hosts. Infected fish are commonly reported throughout Central and South America, but remarkably little is known about distribution and prevalence of infections in avian hosts there. *Eustrongylides ignotus* was identified in all avian cases in Central and South America except for a single report of *E. tubifex* in a Cocoi Heron (*Ardea cocoi*) from Brazil (Measures 1988a).

Important fish intermediate hosts of *E. ignotus* in North America are livebearers (Poeciliidae), especially the eastern mosquitofish (*Gambusia holbrooki*), sunfish (Centrarchidae), and killifish (*Fundulus heteroclitus*). By contrast, piranhas (*Serrasalmus nattereri*) are important intermediate hosts in South America

Table 16.1. Avian hosts of *Eustrongylides* spp.

Host order	Host species	Locality	Species	Type	Number infected/ Number of examinations	Reference
Sphenisciformes	Jackass Penguin (*Spheniscus demersus*)	Pennsylvania, USA	*E*. sp.	C	NG	Winsor (1948)
Gaviiformes	Red-throated Loon (*Gavia stellata*)	Europe	*E. tubifex*	N	NG	Measures (1988a)
		Europe	*E*. sp.	N	NG	Jagerskiold (1909b)
		Russia	*E. tubifex*	N	2/NG	Kontrimavichus and Bakhmet'eva (1960)*
		Ukraine	*E. tubifex*	N	NG	Smogorzhevskaya (1954, 1959,1962)*
	Arctic Loon (*Gavia arctica*)	Europe	*E. tubifex*	N	NG	Measures (1988a)
		Finland	*E. tubifex*	N	NG	Karmanova (1986)
		Russia	*E. tubifex*	N	1/NG	Kontrimavichus and Bakhmet'eva (1960)*
		Ukraine	*E. tubifex*	N	NG	Smogorzhevskaya (1954, 1959, 1962)*
	Common Loon (*Gavia immer*)	Florida, USA	*E. tubifex*	N	3/45	Forrester and Spalding (2003)
		Nova Scotia, Canada	*E. tubifex*	N	1/NG	USNPC† # 87045
		Ontario, Canada	*E. tubifex*	N	1/25	Measures (1988a)
Charadriiformes	Ruff (*Philomachus pugnax*)	Finland	*E. tubifex*	N	NG	Fagerholm (1979)
	Mew Gull (*Larus canus*)	Russia	*E*. sp.	N	NG	Skrjabin (1920)*
	Black-headed Gull (*Larus ridibundus*)	Russia	*E*. sp.	N	NG	Shigin (1961)*
	Common Murre (*Uria aalge*)	Russia	*E. ignotus*	N	1/1	Morishita (1930)
	Murre (*Uria* sp.) species not given	Europe	*E*. sp.	N	NG	Jagerskiold (1909b)
Podicipediformes	Little Grebe (*Tachybaptus ruficollis*)	England	*E. tubifex*	N	NG	Measures (1988a)
		Europe	*E*. sp.	N	NG	Jagerskiold (1909b)
		Yugoslavia	*E. tubifex*	N	3/18	Brglez (1980)
		Japan	*E. tubifex*	N	1/1	Murata et al. (1997)
		Japan	*E*. sp.	N	NG	Yamaguti (1935)*
	Great Crested Grebe (*Podiceps cristatus*)	Yugoslavia	*E. tubifex*	N	1/12	Brglez (1980)
		Europe	*E*. sp.	N	NG	Jagerskiold (1909b)
		Russia	*E*. sp.	N	NG	Shigin (1957)*

(*continues*)

Table 16.1. (*Continued*)

Host order	Host species	Locality	Species	Type	Number infected/ Number of examinations	Reference
		Ukraine	*E*. sp.	N	NG	Smogorzhevskaya (1954, 1962, 1964)*
	Horned Grebe (*Podiceps auritus*)	Russia	*E*. sp.	N	NG	Kosupko (1963)*
		Ukraine	*E. excisus*	N	NG	Smogorzhevskaya (1954, 1962, 1964)*
		Romania	*E. excisus*	N	NG	Ciurea (1938)*
		Russia	*E. excisus*	N	NG	Karmanova (1986)
		Russia	*E*. sp.	N	NG	Nikol'skaya (1939)*
Pelecaniformes	Double-crested Cormorant (*Phalacrocorax auritus*)	Ontario, Canada	*E. tubifex*	E	8/10	Measures (1988a)
	Little Pied Cormorant (*Phalacrocorax melanoleucos*)	New Zealand	*E. ignotus*	N	NG	Measures (1988a)
		Australia	*E. excisus*	N	NG	Johnston and Mawson (1941)
	Pygmy Cormorant (*Phalacrocorax pygmaeus*)	Europe	*E*. sp.	N	NG	Jagerskiold (1909b)
		Romania	*E. excisus*	N	NG	Ciurea (1938)*
	Long-tailed Cormorant (*Phalacrocorax africanus*)	Uganda	*E*. sp.	N&E	4	Paperna (1974)
	Great Cormorant (*Phalacrocorax carbo*)	Europe	*E. excisus*	N	NG	Jagerskiold (1909b)
		Australia	*E. excisus*	N	NG	Johnston and Mawson (1941)
		East Arabia	*E. excisus*	N	NG	Measures (1988a)
		Europe	*E*. sp.	N	NG	Jagerskiold (1909b)
		New Zealand	*E. ignotus*	N	—	Dickinson (1951)*
		Ukraine	*E. tubifex*	N	NG	Smogorzhevskaya (1954, 1959,1962)*
		Ukraine	*E. excisus*	N	NG	Smogorzhevskaya (1954, 1962, 1964)*
	Black-faced Cormorant (*Phalacrocorax fuscescens*)	Australia	*E. excisus*	N	NG	Johnston and Mawson (1941)
	Cormorant (*Phalacrocorax* sp.) species not given	South America	*E. ignotus*	N	NG	Caballero (1982)

	Anhinga (*Anhinga anhinga*)	Florida, USA	*E. ignotus*	N	1/1	Forrester and Spalding (2003)
		Florida, USA	*E. ignotus*	N	NG	Measures (1988a)
		Brazil	*E. ignotus*	N	NG	Rego and Vicente (1988)
	Darter (*Anhinga melanogaster*)	Australia	*E. excisus*	N	—	Johnston and Mawson (1941)
		Sudan	*E*. sp.	N	NG	Jagerskiold (1909a)
	Pink-backed Pelican (*Pelecanus rufescens*)	Sudan	*E*. sp.	N	NG	Jagerskiold (1909a)
	American White Pelican (*Pelecanus erythrorhynchos*)	Florida, USA	*E*. sp.	N	1/6	Forrester and Spalding (2003)
	Brown Pelican (*Pelecanus occidentalis*)	Florida, USA	*E*. sp.	N	1/4	Forrester and Spalding (2003)
		Puerto Rico	*E*. sp.	N	2/23	Dyer et al. (2002)
Ciconiiformes	Gray Heron (*Ardea cinerea*)	Yugoslavia	*E*. sp.	N	NG	Brglez (1981)
	Great Blue Heron (*Ardea herodias*)	Delaware, USA	*E. ignotus*	N	5/5	Ziegler et al. (2000)
		Florida, USA	*E. ignotus*	N	45/225	Spalding et al. (1993)
		Indiana, USA	*E. ignotus*	N	2/25	Winterfield and Kazacos (1977)
		Maryland, USA	*E. ignotus*	N	1/NG	Locke (1961)
		Maryland, USA	*E*. sp.	N	NG	Measures (1988a)
		New Jersey, USA	*E*. sp.	N	NG	Measures (1988a)
		New Jersey, USA	*E. ignotus*	N	1/NG	Bowdish (1948)
		New York, USA	*E. ignotus*	N	NG	Measures (1988a)
		Ohio, USA	*E*. sp.	N	8/23	Cooper et al. (1978a)
		Ontario, Canada	*E. tubifex*	N	3/68	Measures (1988c)
		Ontario, Canada	*E. tubifex*	E	1/1	Measures (1988c)
		Virginia, USA	*E. ignotus*	N	NG	Measures (1988a)
		Virginia, USA	*E*. sp.	N	NG	Evans (1990)
		Washington, DC, USA	*E. ignotus*	N	1/NG	Chapin (1926)
		Washington, DC, USA	*E*. sp.	N	NG	Measures (1988a)
		Washington, DC, USA	*E. ignotus*	E. sp.	NG	Measures (1988a)

(*continues*)

Table 16.1. (*Continued*)

Host order	Host species	Locality	Species	Type	Number infected/ Number of examinations	Reference
	Blue Heron (species not given)	Wisconsin, USA	*E. ignotus*	N	—	Windingstad and Swineford (1981)
	Cocoi Heron (*Ardea cocoi*)	Brazil	*E. ignotus*	N	NG	Rego and Vicente (1988); Measures (1988a)
		Brazil	*E. ignotus*	C	7/9	Pinto et al. (2004)
	Goliath Heron (*Ardea goliath*)	Sudan	*E*. sp.	—	—	Jagerskiold (1909a); Measures (1988a)
	Great Egret (*Ardea alba*)	Florida, USA	*E. ignotus*	N	94/427	Spalding et al. (1993)
		Louisiana, USA	*E*. sp.	N	5/5	Roffe (1988)
		Maryland, USA	*E. ignotus*	N	2/NG	Locke (1961)
		Delaware, USA	*E. ignotus*	N	2/NG	Wiese et al. (1977)
		Texas, USA	*E*. sp.	N	8/10	Franson and Custer (1994)
		Ohio, USA	*E*. sp.	N	4/36	Cooper et al. (1978b)
		Russia	*E*. sp.	N	NG	Dubinin and Dubinina (1940)
		Brazil	*E. ignotus*	N	NG	Rego and Vicente (1988)
		Brazil	*E. ignotus*	C	30/42	Pinto et al. (2004)
	Tricolored Heron (*Egretta tricolor*)	Florida, USA	*E. ignotus*	N	49/472	Spalding et al. (1993)
		Florida, USA	*E. ignotus*	E	3/3	Spalding et al. (1994)
		Delaware, USA	*E. ignotus*	N	2/4	Wiese et al. (1977)
	Little Blue Heron (*Egretta caerulea*)	California, USA	*E. ignotus*	C	NG	Measures (1988a)
		Delaware, USA	*E. ignotus*	N	1/7	Wiese et al. (1977)
		Florida, USA	*E. ignotus*	N	25/360	Spalding et al. (1993)
	Snowy Egret (*Egretta thula*)	Florida, USA	*E. ignotus*	N	82/439	Spalding et al. (1993)
		Delaware, USA	*E. ignotus*	N	50/53	Wiese et al. (1977)
		Texas, USA	*E*. sp.	N	9/9	Franson and Custer (1994)
		California, USA	*E*. sp.	N	3/15	Franson and Custer (1994)
		Rhode Island, USA	*E*. sp.	N	1/10	Franson and Custer (1994)
	Little Egret (*Egretta garzetta*)	China	*E*. sp.	N	NG	Wu and Liu (1943)*
	Chinese Pond-heron (*Ardeola bacchus*)	China	*E. excisus*	N	NG	Hoeppli et al. (1929)*
	Cattle Egret (*Bubulcus ibis*)	Taiwan	*E. excisus*	N	1/NG	Sugimoto (1933)*
	Green Heron (*Butorides virescens*)	Florida, USA	*E. ignotus*	N	1/11	Spalding et al. (1993)

Black-crowned Night-Heron (*Nycticorax nycticorax*)	Brazil	*E. ignotus*	N	NG	Rego and Vicente (1988); Schaffer et al. (1990)
	Brazil	*E. ignotus*	C	15/35	Pinto et al. (2004)
	California, USA	*E*. sp.	N	1/15	Franson and Custer (1994)
	China	*E. excisus*	N	NG	Hoeppli et al. (1929)*
	Delaware, USA	*E. ignotus*	N	2/4	Wiese et al. (1977)
	Florida, USA	*E. ignotus*	N	1/4	Spalding et al. (1993)
	Maryland, USA	*E. ignotus*	N	NG	Measures (1988a)
	New Jersey, USA	*E. ignotus*	N	1/NG	Bowdish (1948)
	Ohio, USA	*E*. sp.	N	23/36	Cooper et al. (1978a)
	Rhode Island, USA	*E*. sp.	N	1/10	Franson and Custer (1994)
	Russia	*E*. sp.	N	NG	Dubinina (1937)
	Taiwan	*E. excisus*	N	NG	Sugimoto (1933)*
	Texas, USA	*E*. sp.	N	1/8	Franson and Custer (1994)
	Washington, DC, USA	*E. ignotus*	N	1/NG	Cram (1933)
Yellow-crowned Night-Heron (*Nyctanassa violacea*)	Florida, USA	*E. ignotus*	N	1/6	Spalding et al. (1993)
	Brazil	*E. ignotus*	N	NG	Rego and Vicente (1988) and Schaffer et al. (1990)
Rufescent Tiger-Heron (*Tigrisoma lineatum*)	Brazil	*E. ignotus*	N	NG	Rego and Vicente (1988)
Pinnated Bittern (*Botaurus pinnatus*)	Brazil	*E. ignotus*	N	NG	Rego and Vicente (1988)
American Bittern (*Botaurus lentiginosus*)	Ohio, USA	*E*. sp.	N	1/NG	Cooper et al. (1978a)
Wood Stork (*Mycteria americana)*	Florida, USA	*E. ignotus* (l)	N	3/15	Forrester and Spalding (2003)
	Brazil	*E. ignotus*	C	3/10	Pinto et al. (2004)
Maguari Stork (*Ciconia maguari*)	Brazil	*E. ignotus*	N	NG	Vicente et al. (1995)
Marabou Stork (*Leptoptilos crumeniferus*)	Sudan	*E*. sp.	N	NG	Jagerskiold (1909a)
	Uganda	*E*. sp.	N	1/6	Moriearty et al. (1972)

(*continues*)

Table 16.1. (*Continued*)

Host order	Host species	Locality	Species	Type	Number infected/ Number of examinations	Reference
	Black-headed Ibis (*Threskiornis melanocephalus*)	India	*Eustrongylides excisus*	N	NG	Ali (1971)
	Bare-faced Ibis (*Phimosus infuscatus*)	Brazil	*Eustrongylides ignotus*	N	NG	Rego and Vicente (1988) and Schaffer et al (1990)
	White Ibis (*Eudocimus albus*)	Florida, USA	*Eustrongylides ignotus* (l)	N	5/171	Spalding et al. (1993)
	Glossy Ibis (*Plegadis falcinellus*)	Yugoslavia	*Eustrongylides* sp.	N	1/2	Brglez (1980)
		Russia	*Eustrongylides* sp.	N	NG	Dubinin and Dubinina (1940)
		Ukraine	*Eustrongylides* sp.	N	NG	Smogorzhevskaya (1954, 1962, 1964)*
	Eurasian Spoonbill (*Platalea leucorodia*)	Azerbaidzhan	*Eustrongylides* sp.	N	NG	Feizullaev (1962, 1963)*
		Kazakhstan	*Eustrongylides* sp.	N	NG	Skryabin (1916, 1923)*
		Russia	*Eustrongylides* sp.	N	NG	Dubinin and Dubinina (1940)*
	African Spoonbill (*Platalea alba*)	Ethiopia	*Eustrongylides* sp.	N	NG	Graber (1975)
	Roseate Spoonbill (*Platalea ajaja*)	Florida, USA	*Eustrongylides ignotus*	N	1/136	Sepulveda et al. (1994)
		Florida, USA	*Eustrongylides ignotus*	N	1/129	Spalding and Forrester (1993)
Anseriformes	Greylag Goose (*Anser anser*)	Denmark	*Eustrongylides* sp.	N	NG	Madsen (1952)
	Mallard (*Anas platyrhynchos*)	Ohio, USA	*Eustrongylides tubifex*	E	NG	Fastzkie and Crites (1977)
		Ohio, USA	*Eustrongylides tubifex*	E	5/74	Cooper et al. (1978a)
		Georgia (former USSR)	*Eustrongylides* sp.	N	NG	Ryzhikov (1950)*
	Domestic White Pekin (*Anas platyrhynchos*)	Ontario, Canada	*Eustrongylides tubifex*	E	4/11	Measures (1988c)
		Denmark	*Eustrongylides* sp.	N	NG	Madsen (1952)
		Japan	*Eustrongylides* sp.	N	NG	Sugimoto (1932)*
		Taiwan	*Eustrongylides excisus*	NG	NG	Sugimoto (1931)*
	Northern Pintail (*Anas acuta*)	Georgia (former USSR)	*Eustrongylides* sp.	N	NG	Ryzhikov (1950)*
	Garganey (*Anas querquedula*)	Georgia (former USSR)	*Eustrongylides* sp.	N	NG	Ryzhikov (1950)*
		USSR	*Eustrongylides* sp.	N	NG	Lyubimov (1926)*
	Northern Shoveler (*Anas clypeata*)	The Netherlands	*Eustrongylides* sp.	N	NG	Measures (1988a)
	Tufted Duck (*Aythya fuligula*)	Russia	*Eustrongylides* sp.	N	NG	Ryzhikov (1963)*
	White-winged Scoter (*Melanitta fusca*)	Alaska, USA	*Eustrongylides tubifex*	E	NG	Measures (1988a)

	Long-tailed Duck (*Clangula hyemalis*)	Europe	*Eustrongylides* sp.	N	NG	Jagerskiold (1909b)
	Common Goldeneye (*Bucephala clangula*)	Ukraine	*Eustrongylides tubifex*	N	NG	Smogorzhevskaya (1964)*
	Smew (*Mergellus albellus*)	Germany	*Eustrongylides tubifex*	N	NG	Measures (1988a)
		Europe	*Eustrongylides* sp.	N	NG	Jagerskiold (1909b)
	Hooded Merganser (*Lophodytes cucullatus*)	Ontario, Canada	*Eustrongylides tubifex*	E	7/10	Measures (1988c)
	Red-breasted Merganser (*Mergus serrator*)	Florida, USA	*Eustrongylides* sp.	N	3/10	Forrester and Spalding (2003)
		Virginia, USA	*Eustrongylides* sp.	N	36/36	Locke et al. (1964)
		Ohio, USA	*Eustrongylides* sp.	N	6/6	Cooper et al. (1978a)
		Ontario, Canada	*Eustrongylides tubifex*	N	2/50	Measures (1988c)
		Ontario, Canada	*Eustrongylides tubifex*	E	2/2	Measures (1988c)
	Common Merganser (*Mergus (merganser)*	Germany	*Eustrongylides tubifex*	E	NG	Measures (1988c)
		Europe	*Eustrongylides* sp.	N	NG	Jagerskiold (1909b)
		Ontario, Canada	*Eustrongylides tubifex*	N	22/106	Measures (1988c)
		Ontario, Canada	*Eustrongylides tubifex*	E	27/34	Measures (1988c)
		New Brunswick, Canada	*Eustrongylides tubifex*	N	5/435	Measures (1988c)
	Merganser (species not given)	British Columbia, Canada	*Eustrongylides tubifex*	N	NG	Shillinger (1936)
Falconiformes	Bald Eagle (*Haliaeetus leucocephalus*)	Alaska, USA	*Eustrongylides tubifex*	N	NG	Tuggle and Schmeling (1982)
	Greater Spotted Eagle (*Aquila clanga*)	Russia	*Eustrongylides* sp.	N	1/2	Oshmarin (1963)*
	Lesser Spotted Eagle (*Aquila pomarina*)	Russia	*Eustrongylides excisus*	N	1/NG	Ciurea (1938)*
Gruiformes	Eurasian Coot (*Fulica atra*)	Spain	*Eustrongylides* sp.	N	3/3	Acosta et al. (1988)
Passeriformes	Eurasian Nutcracker (*Nucifraga caryocatactes*)	NG	*Eustrongylides* sp.	N	NG	Rudolphi (1819)* (see Measures (1988a))

l, identification based upon larval nematode; N, native or wild captured; C, captive; E, experimental infection; NG, no prevalence data given; —, reference not seen or not in translated abstract.

*Information taken from Karmanova (1986), original paper not seen.

†United States National Parasite Collection in Beltsville, MD, USA.

(Wiese et al. 1977; Hirshfield et al. 1983; Weisberg et al. 1986; Rego and Vicente 1988; Coyner et al. 2002).

E. excisus has the widest geographic distribution and is a common parasite of cormorants and species of Ciconiiformes in Asia, although infections have been recorded in a few other species of Pelecaniformes from Australia and Europe. It has not yet been reported from the Americas. The most important fish intermediate hosts are gobies (*Neogobius kessleri* and *Neogobius melanostomus*) and the Caspian roach (*Rutilus rutilus caspicus*) (Karmanova 1986). Other secondary reservoir hosts include predatory fish, especially perch (*Perca fluviatilis*), and frogs and snakes.

Eustrongylides tubifex is found most commonly in mergansers and cormorants, and occasionally in species of Ciconiiformes in North America, but also causes lesions in loons and grebes in Europe and Asia. There are only two reports of mortality due to *E. tubifex*, and these involved mergansers in British Columbia and Virginia (Shillinger 1936; Locke et al. 1964). Records for *E. tubifex* in North America south of Ohio are infrequent. *Eustrongylides ignotus* was the only species found in a survey of species of Ciconiiformes in Florida (Spalding et al. 1993). However, a few Common Loons (*Gavia immer*) were found infected with *E. tubifex* in Florida (Forrester and Spalding 2003), but it is likely that they acquired the infections in northern North America where they breed. Other than a single record of an infected Cocoi Heron in Brazil (Measures 1988a), most other records in South and Central America are for *E. ignotus* (Rego and Vicente 1988). A similar pattern is evident in Europe and Asia and most records of *E. tubifex* are from northern latitudes. Pumpkinseed (*Lepomis gibbosus*) and yellow perch (*Perca flavescens*) serve as important intermediate hosts in North America (Measures 1988b).

There is some evidence that raptors may act as definitive hosts of *Eustrongylides*, but such reports are not very common. Tuggle and Schmeling (1982) reported *Eustrongylides* spp. in a Bald Eagle (*Haliaeetus leucocephalus*) from Alaska, and these were later identified as gravid females of *E. tubifex* (J. R. Lichtenfels, personal communication). Lesions from one of two Greater Spotted Eagles (*Aquila clanga*) from Russia are suggestive of a mature eustrongylid infection (Oshmarin 1963). Unfortunately, the species and stage of these nematodes were not given.

ETIOLOGY

Eustrongylides spp. are large, red, grossly visible nematodes in the order Enoplida, superfamily Dioctophymatoidea, and family Dioctophymidae. They are distinguished from other members of the superfamily by the lack of a posterior sucker. Adults measure about 4–15 cm or greater in length and 1–4 mm in diameter (Measures 1988a). The taxonomy of the genus *Eustrongylides* was reviewed by Karmanova (1986) and revised by Rautela and Malhotra (1984), Fastzkie and Crites (1977), and recently by Measures (1988a) who recognized three species: *Eustrongylides tubifex* (synonym = *E. perpapillatus*), *E. excisus* (synonyms = *E. formosensis*, *E. indicus*, *E. phalacrocoracis*, *E. plotinus*, *E. tricolor*, and *E. excisus amoyensis*), and *E. ignotus*. An additional description of *E. ignotus* was given by Rego and Vicente (1988). Distinguishing features include characteristics of the labial papillae around the mouth and the perimeter of the caudal sucker of the male and are illustrated by Measures (1988a) (Figures 16.2 and 16.3). A number of other poorly defined species were listed by Measures (1988a) as species *inquirendae.* These include *E. africanus*, *E. mergorum*, *E. papillosus*, *E. rubrum*, *E. sinicus*, *E. spinispiculum*, and those described from larvae in fish—*E. acrochordi*, *E. gadopsis*, and *E. wenrichi*.

EPIZOOTIOLOGY

Our understanding of the epizootiology of *Eustrongylides*, although initiated in the early 1900s, still remains very incomplete despite the significant harm these worms cause in birds and fish. The relatively recent discovery of an association between nutrient pollution and increases in the prevalence of these parasites appears to be fundamental to understanding the epizootiology of these organisms. Factors that must come together for an epizootic to occur include eutrophic wetlands that support high densities of fish, attraction of infected birds to feed on the fish, and nutrient-polluted sites that provide habitat for the appropriate oligochaete intermediate hosts. Thus, the ultimate initiator of this system is nutrient input. These characteristics are important for transmission of *E. ignotus* in Florida and have been suggested for the other two species of *Eustrongylides* (Karmanova 1986; Measures 1988b; and Coyner et al. 2002).

Life History

Transmission to the definitive host is by ingestion of infected fish or, in some cases, by ingestion of a paratenic host. The life history of all three species of *Eustrongylides* is believed to involve piscivorous avian definitive hosts infected with fourth-stage (L4) larvae and mature, sexually reproducing adults, oligochaete first intermediate hosts infected with first- (L1), second- (L2), and third-stage (L3) larvae, and fish second intermediate hosts infected with L3 and L4 larvae (Karmanova 1986; Anderson 2000). Coyner et al. (2003a), however, were able to infect fish directly with larvated eggs of *E. ignotus* without passage through

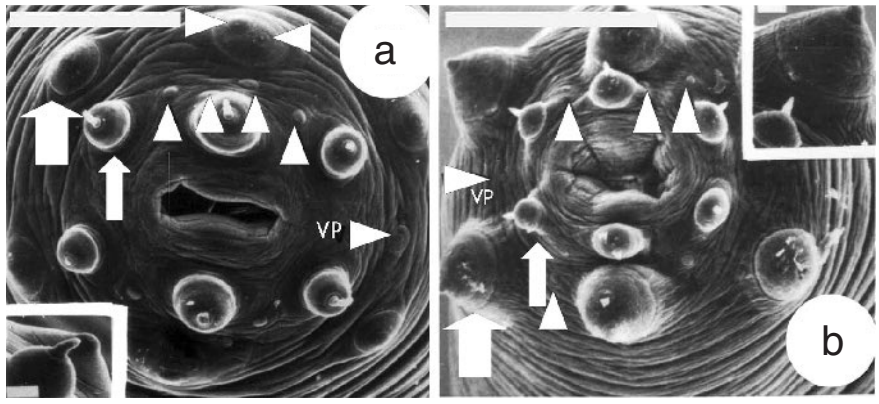


Figure 16.2. Diagnostic features of the cranial end of *Eustrongylides* spp. (a) *Eustrongylides ignotus* and (b) *Eustrongylides tubifex.* Note the relatively larger inner circle papillae (narrow arrow) of *E. ignotus*. *E. tubifex* is the only species in which the inner labial papillae are smaller than the outer (wide arrow). Some lateral field papillae (small arrowheads) and ventral papilla (vp) are also visible. Scale bar = 100 μm (insets = 10 μm). From Measures (1988a), with permission of the *Canadian Journal of Zoology*.

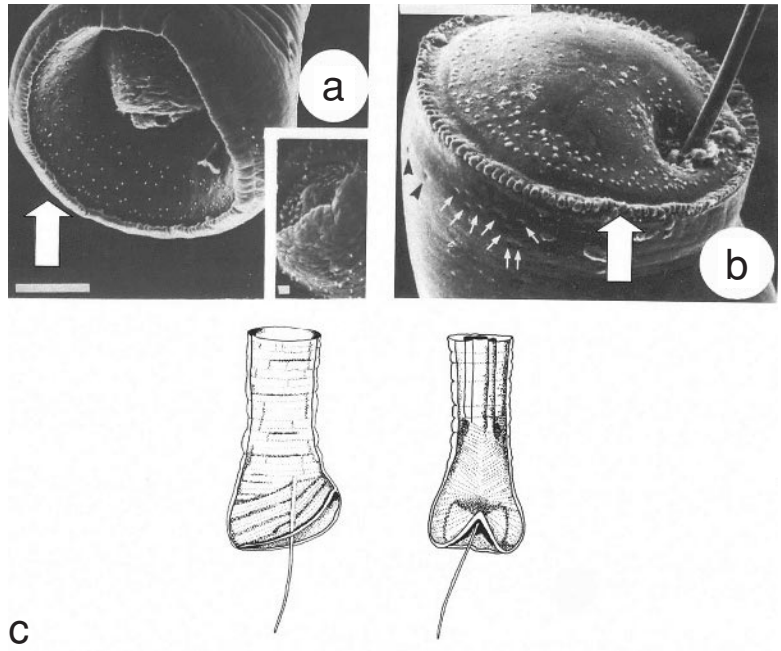


Figure 16.3. Diagnostic features of the caudal end of male *Eustrongylides* spp. (a) *Eustrongylides ignotus*, (b) *Eustrongylides tubifex*, and (c) *Eustrongylides excisus*. Note the projections from the ruffled edge (large arrow) on the rim of *E. tubifex*, and the wide cuticular hem on the outer perimeter of the sucker (wide arrow) of *E. ignotus*. Small white arrows and black arrowheads indicate groups of caudal papillae. The dorsal wall of the posterior part of the rectum of *E. ignotus* bears spines and protrudes from anus (inset). Note the ventral cleft in the drawing of *E. excisus*. Scale bar = 100 μm (insets = 10 μm). From Measures (1988a), with permission of the *Canadian Journal of Zoology*.

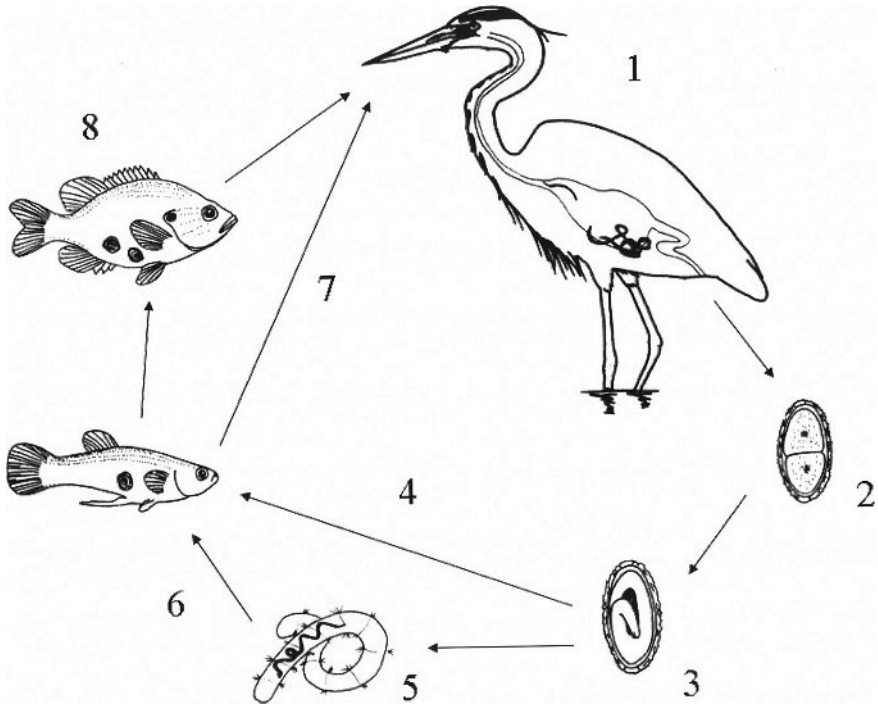


Figure 16.4. Life cycle of *Eustrongylides* sp. (1) Definitive host, a piscivorous bird, consumes infected fish. The encysted fourth-stage larva (L4) burrows through the stomach wall and matures to an adult. Adults mate and eggs develop in the uterus of the female. (2) Eggs are produced and pass out in feces. The zygote cleaves. (3) The egg matures to the infective stage containing a first-stage larva (L1). (4) A fish intermediate host consumes an egg containing an L1. The L1 encysts and matures to an infective L4, or (5) an egg containing an L1 is consumed by an oligochaete intermediate host and matures to an infective third-stage larva (L3). (6) A fish intermediate host consumes an oligochaete intermediate host containing an L3. The larvae encyst in the fish and develop to an L4. (7) A definitive host consumes a fish containing an encysted L4. (8) A predatory fish or other paratenic or accidental host consumes a fish intermediate host and the L4 encysts. The short cycle can be completed in 115 days, the long cycle takes >330 days.

oligochaetes (Figure 16.4). This mode of transmission shortens the life cycle by 78–156 days, but its importance in natural cycles or in other species is unknown. It could be significant in situations where rapid infection of the fish intermediate host is critical or where overwintering of larvae is not necessary. With the exception of *E. excisus*, the survival time of adult worms in the avian host is not known. Developmental times of the parasite in the environment and in oligochaete, fish, and avian hosts have been determined by experimental studies (Table 16.2).

For *E. tubifex*, the first appearance of L3 larvae in oligochaetes takes longer than for *E. ignotus*. Multiple infections, with an average intensity of 5.5 larvae in each oligochaete worm (*Limnodrilus hoffmeisteri*), have been documented experimentally for *E. tubifex*. Like *E. ignotus*, the development time is temperature dependent. The length of time for development in a fish to the L4 stage is not known for *E. tubifex* and *E. excisus*. Adult parasites die and nodular lesions resolved within 30 days (Measures 1988c).

Under experimental conditions, it takes 5–5.5 months for the larva *E. excisus* to develop to infective L3 larvae in oligochaetes, but Karmanova (1986) suspected that natural transmission might occur faster based upon evidence of reinfection of avian hosts in the Volga Delta. The natural life cycle from egg to death of adult worms is reported to be 8–9 months in Great Cormorants (*Phalacrocorax carbo*) (Karmanova 1986). Overwintering of larval *E. excisus* is thought to occur in fish since cormorants arrive in the Volga Delta in the spring with no evidence of infection. Young nematodes

Table 16.2. The range of development times in days for the life history stages of *Eustrongylides ignotus, Eustrongylides tubifex,* and *Eustrongylides excisus* as determined through experimental studies.

	Eustrongylides ignotus without oligochaete*		*Eustrongylides ignotus* with oligochaete		*Eustrongylides tubifex*†		*Eustrongylides excisus*‡	
Stage	Min	Max	Min	Max	Min	Max	Min	Max
Egg in feces to infective L1 in egg	17	28	17	28	23	26	22	—
L2 to infective L3 in oligochaete	0	0	35	77	70	109	150	165
L3 (or L2) to L4 in fish	84	105	127	184	—	—	—	—
L4 to egg in bird feces	20	23	20	23	10	17	12	14
Days to complete life cycle	121	156	199	312	—	—	—	—

L1, first-stage larvae; L2, second-stage larvae; L3, third-stage larvae; L4, fourth-stage larvae.

*From Coyner et al (2003a), the first column omits the oligochaete phase, the second column includes it. Third-stage larvae (L3) can survive up to 284 days in an oligochaete.

†From Measures (1988c), infection of fish with oligochaetes containing third-stage larvae (L3) was not successful. Larvae could be maintained in captive fish for 18 months (Crites 1982).

‡From Karmanova (1986), development of second- to third-stage larvae (L2–L3) is expected to occur faster in a natural system.

can be found in cormorants at the end of May to early June, and sexually mature nematodes are found by the end of June. Immature nematodes were recovered from avian hosts throughout the summer, indicating that reinfection was occurring. Cormorants appeared to be free of infection when they migrated for the winter (Karmanova 1986).

The range of oligochaete intermediate and/or paratenic hosts is also wide. Identification is complicated both by the difficulty in finding naturally infected oligochaetes and also by the necessity to pass larval nematodes through fish intermediate and avian definitive hosts in order to identify these stages to species. Oligochaetes infected naturally with larval nematodes have not been documented with certainty, but one was suspected to be parasitized by *E. tubifex* (Lichtenfels and Stroup 1985). Evidence for inclusion of oligochaetes in the life cycle is based almost entirely on experimental work. In Florida, L. *hoffmeisteri* seems to be the most important, if not the only oligochaete intermediate host for *E. ignotus* (Spalding et al. 1993; Coyner et al. 2002, 2003a). Both *L. hoffmeisteri* and *Tubifex tubifex* can be experimentally infected with *E. tubifex* (Measures 1988d). Based on experimental studies, the oligochaete intermediate hosts of *E. excisus* are *Limnodrilus* sp., *T. tubifex*, and *Lumbriculus variegatus* (Karmanova 1986).

There is evidence that larval *Eustrongylides* (L4, and possibly L3) can be transmitted from fish to fish and to amphibians and reptiles by predation. This has been demonstrated in the laboratory for *E. ignotus* (Coyner et al. 2003a), *E. tubifex* (Measures 1988b), and is presumed to occur in the field for *E. excisus* (Karmanova 1986). This route of transmission allows the parasite to accumulate in a paratenic or reservoir host and may account for high prevalence and intensity of infection in larger predatory fish (Karmanova 1986; Measures 1988b; Coyner et al. 2002). In an experimental setting, infected fish are more likely to be captured and are more quickly captured by predatory fish than uninfected fish (Coyner et al. 2001). Larval *Eustrongylides* have been collected from various mammals, reptiles, and amphibians (Table 16.3), but these are likely aberrant hosts. In some cases, these may be small enough to be consumed by the definitive host birds and thereby act as paratenic hosts.

There is a single report of sturgeon (*Acipenser gueldenstaedtii*) with encysted mature *E. excisus* that contained eggs, indicating that it may be possible for fish also to act as definitive hosts (Mikailov et al. 1992). There have been no other reports of adult *Eustrongylides* spp. in fish, but they could be overlooked since histologic examination of encysted nematodes is not routinely done.

Environmental Limitations

Eutrophication and nutrient input are significant environmental factors affecting transmission of

Table 16.3. Paratenic and accidental hosts of *Eustrongylides* spp.

Host	Locality	Species*	Type	Number infected/ Number of examinations	References
Mammals					
River otter (*Lutra canadensis*)	Maryland, USA	*E*. sp.	N	1/NG	Abram and Lichtenfels (1974)
	Oregon, USA	*E*. sp.	N	12/29	Hoberg et al. (1997)
Caspian seal (*Phoca caspica*)	Russia	*E. excisus* (l)	N	6/15	Shchupakov (1936)*
Muskrat (*Ondatra zibethica*)	Ontario, Canada	*E*. sp.	N	1/1	Gibson and McKiel (1972)
Raccoon Dog (*Nyctereutes procyonoides*)	Japan	*E*. sp.	N	NG	Morishita (1923)
"Rat"	Maryland, USA	*E*. sp.	E	NG	von Brand and Cullinan (1943)
New Zealand white rabbit (*Oryctolagus cuniculus*)	Maryland, USA	*E*. sp.	E	6/6	Shirazian et al. (1984)
	Brazil	*E. ignotus*	E	7/10	Barros et al. (2004)
Human (*Homo sapiens*)	Maryland, USA	*E*. sp.	N	2/NG	Gunby (1982); Guerin et al. (1982)
	New Jersey, USA	*E. ignotus*	N	1/NG	Eberhard et al. (1989)
	New York, USA	*E*. sp.	N	1/NG	Arias (1989)
Reptiles					
Brown water snake (*Nerodia taxispilota*)	Florida, USA	*E*. sp.	N	1/5	Ferenc et al. (1986)
Northern water snake (*Nerodia sipedon*)	Virginia, USA	*E*. sp.	N	18/18	Bursey (1986)
	Pennsylvania, USA	*E*. sp.	C	NG	Winsor (1948)
Indigo snake (*Drymarchon corais*)	Florida, USA	*E*. sp.	N	1/12	Foster et al. (2000)
Garter snake (*Thamnophis sirtalis*)	Maryland, USA	*E*. sp.	E	20/25	Lichtenfels and Lavies (1976)
	Pennsylvania, USA	*E*. sp.	C	NG	Winsor (1948)
Grass snake (*Natrix tessellata*)	Russia	*E. excisus*	N	NG	Karmanova (1986)
Blue Racer (*Coluber constrictor*)	Pennsylvania, USA	*E*. sp.	C	NG	Winsor (1948)
Whip snake (*Masticophis flagellum*)	Pennsylvania, USA	*E*. sp.	C	NG	Winsor (1948)
Pine snake (*Pituophis melanoleucus*)	Pennsylvania, USA	*E*. sp.	C	NG	Winsor (1948)
	Pennsylvania, USA	*E*. sp.	C	NG	Winsor (1948)
Fer-de-lance (*Bothrops atrox*)	Pennsylvania, USA	*E*. sp.	C	NG	Winsor (1948)
Copperhead (*Agkistrodon contortrix*)	Pennsylvania, USA	*E*. sp.	C	NG	Winsor (1948)
Javan wart snake (*Acrochordus javanicus*)	Australia	*E*. sp.	N	1/8	Jones (1978)
Jararaca (*Bothrops jararaca*)	Brazil	*E. ignotus*	N	NG	Rego and Vicente (1988)
Green anaconda (*Eunectes murinus*)	Brazil	*E. ignotus*	N	NG	Rego and Vicente (1988)
Coral snake (*Micrurus* sp.)	Brazil	*E. ignotus*	N	NG	Rego and Vicente (1988)

Cazadora brown-lined snake (*Dryadophis bifossatus*)	Brazil	*E. ignotus*	N	NG	Rego and Vicente (1988)
Mussurana (*Clelia clelia*)	Brazil	*E. ignotus*	N	NG	Rego and Vicente (1988)
Graceful brown snake (*Rhadinea merremii*)	Brazil	*E. ignotus*	N	NG	Rego and Vicente (1988)
Snapping turtle (*Chelydra serpentina*)	Ohio, USA	*E. tubifex*	E	NG	Cooper et al. (1978b)
	Texas, USA	*E*. sp.	N	NG	Caballero (1982)
Blandings turtle (*Emys blandingi*)	Ohio, USA	*E. tubifex*	E	NG	Cooper et al. (1978b)
Spiny softshell turtle (*Apalone spinifer*)	Ohio, USA	*E. tubifex*	E	NG	Cooper et al. (1978b)
Painted turtle (*Chrysemys picta*)	Maryland, USA	*E*. sp.	E	3/3	von Brand (1944)
American alligator (*Alligator mississippiensis*)	Maryland, USA	*E*. sp.	E	1/1	von Brand (1944)
Paraguayan caiman (*Caiman yacare*)	Paraguay	*E*. sp.	N	3/115	Goldberg et al. (1991)
Spectacled caiman (Caiman *crocodilus*)	Brazil	*E. ignotus*	N	NG	Rego and Vicente (1988)
Amphibians					
Pig frog (*Rana grylio*)	Florida, USA	*E. ignotus* (l)	N	3/11	Spalding and Forrester (1991)
Northern leopard frog (*Rana pipiens*)	Ohio, USA	*E. tubifex*	E	NG	Cooper et al. (1978b)
	Maryland, USA	*E*. sp.	E	7/7	von Brand (1944)
Bullfrog (*Rana catesbeiana*)	Maryland, USA	*E*. sp.	N	NG	von Brand (1944)
	Louisiana, USA	*E*. sp.	E	20/25	Modzelewski and Culley (1974)
	Nevada, USA	*E*. sp.	N	–	Babero and Golling (1973)
	Cuba	*E*. sp.	N	–	Coy-Otero and Martinez (1987)
Lake frog (*Rana ridibunda*)	Russia	*E. excisus*	N	NG	Karmanova (1986)
	Turkey	*E*. sp.	N	3/258	Dusen and Oz (2006)
Bigfoot leopard frog (*Rana megapoda*)	Mexico	*E*. sp.	N	10/80	Lezama and Sarabia (2002)
Smokey jungle frog (*Leptodactylus pentadactylus*)	Brazil	*E. ignotus*	N	NG	Rego and Vicente (1988)
African clawed frog (*Xenopus laevis*)	California, USA	*E*. sp.	N	1/230	Kuperman et al. (2004)
Mudpuppy (*Necturus maculosus*)	Maryland, USA	*E*. sp.	E	1/1	von Brand (1944)
Three-toed amphiuma (*Amphiuma tridactylum)*	Maryland, USA	*E*. sp.	E	1/1	von Brand (1944)
	Louisiana, USA	*E*. sp.	N	NG	Panesar and Beaver (1979)

l, identification based upon larval nematode; N, native or wild captured; C, captive; E, experimental infection; NG, no prevalence data given; –, reference not found or not in translated abstract.

*Information taken from Karmanova (1986), original paper not seen.

Eustrongylides. Infected fish are never found in undisturbed, naturally oligotrophic habitats in Florida, even under the naturally eutrophic conditions that seasonally occur under wading bird colonies (Spalding et al. 1993; Coyner et al. 2002). The physical and chemical conditions associated with sites containing infected fish are dramatically different from more natural, usually oligotrophic sites. Sites containing infected fish generally have a long-term history of nutrient input such as sewage outflow or storm water runoff. The changes are characteristic of eutrophic water bodies and include physical alterations of the substrate, higher densities of fish and oligochaetes, decreased dissolved oxygen, increased total nitrogen, total phosphorus and chlorophyll a in surface water, higher soil oxygen demand and total phosphorus in sediment, larger grain size, and higher percentage of emergent vegetation and grasses (Coyner et al. 2003b).

Eggs of *Eustrongylides* spp. are tolerant of a wide range of temperatures (Karmanova 1986), but both development and development time may be affected by temperature, salinity, and desiccation. Development of the eggs of *E. tubifex* ceases at 0–15°C in the laboratory, but resume at higher temperatures (Measures 1988d). The eggs of *E. ignotus* are not tolerant of hypersaline (>33 ppt) water or desiccation, but develop normally in fresh or estuarine water (20 ppt) (Coyner et al. 2004).

The range of fish intermediate hosts of *Eustrongylides* is very wide (Karmanova 1986; Measures 1988b; Coyner et al. 2002). Freshwater fish are much more commonly infected than marine fish, and there is some evidence that transmission of *E. ignotus* may not occur in saltwater in Florida (Spalding et al. 1993). Almost no infections are found in wading birds nesting in marine environments (Spalding et al. 1993). The few exceptions are colonies located close to freshwater foraging sites. This may be due to the distribution of the oligochaete intermediate hosts that are not able to reproduce in marine systems. Marine fish may become infected when visiting estuarine or freshwater sites, or when preying on infected fresh or estuarine fish that move into marine systems, leading to the low levels of infections that were found. There is some evidence that *E. excisus* may be more tolerant of marine environments, based on recovery of *E. excisus* from sturgeon in the southern portion of the Caspian Sea where higher salinities are more typical of ocean environments (Sattari and Mokhayer 2005).

Prevalence and Intensity

Prevalence in species of Ciconiiformes can be quite high and range from 11 to 100%, depending on species (Cooper et al. 1978a) (Table 16.1). In a large sample of birds that were collected by gunshot in Ohio, all Red-breasted Mergansers (*Mergus serrator*) were infected. The species of eustrongylid was not identified, but Mallards (*Anas platyrhynchos*) experimentally fed with yellow perch from the area became infected with *E. tubifex*.

Franson and Custer (1994) collected nestling Snowy Egrets (*Egretta thula*), Black-crowned Night-Herons, and Great Egrets (*Ardea alba*) in Texas, Rhode Island, and California. Overall, 31% of broods were infected with the highest prevalence in Texas (35%) where 100% of Snowy Egret, 80% of Great Egret, and 12% of Black-crowned Night-Heron broods were infected. A conservative estimate of 400 dead young birds for the year from eustrongylidosis was made for a colony of 900 pairs of Great Egrets in Louisiana (Roffe 1988).

For *E. ignotus*, an overall prevalence of 14% was reported for all species of Ciconiiformes in Florida, with prevalences ranging from 0% in Cattle Egrets (*Bubulcus ibis*), a species that is not piscivorous, to 33% in Great Blue Herons (Spalding et al. 1993). It was estimated that 80% of nestlings died of eustrongylidosis in one colony. Prevalence was higher in juveniles and adults when compared with nestlings; however, infection was more likely to cause mortality in younger birds. In Great Egret, nestlings prevalences varied among years between 5 and 28% at a colony in central Florida (Spalding et al. 1994). Prevalence increased in fledgling Great White Herons (*Ardea herodias occidentalis*) when they dispersed from marine to freshwater sites in southern Florida (Spalding et al. 1993).

Intensity of *E. excisus* in Great Cormorants and Pygmy Cormorants (*Phalacrocorax pygmaeus*) ranged from 3 to 62 worms per individual in a study to discover the life cycle of the parasite that was impacting commercial fisheries in Romania (Ciurea 1938). In Russia, 50% of young cormorants and 40% of adults were infected (Dubinin 1949). Prevalence of infection was similarly high in an earlier study, with 67% of adults and 40% of young infected (Nikol'skaya 1939).

Increases in prevalence of *Eustrongylides* in infected fish have been reported by several researchers, suggesting that this parasite has become more common in recent years. Prevalence of *E. tubifex* in channel catfish (*Ictalurus punctatus*) from Lake Erie increased from 0% in 1939 (Bangham and Hunter 1939) to 15% by the 1970s (Cooper et al. 1978a). Similar increases in prevalence from 3 to 42% between 1930 and 1978 have been observed in fish collected at the same sites in Florida (Frederick et al. 1996).

CLINICAL SIGNS

Clinical signs in species of Ardeidae of all ages include ataxia, lethargy, depression, emaciation, and pale mucous membranes (Spalding and Forrester 1993;

Ziegler et al. 2000). Little information is available about changes in clinical chemistry or hematology, although anemia and eosinophilia have been reported (Ziegler et al. 2000). In experimental studies, nestling Tricolored Herons (*Egretta tricolor*) that were infected at a few days of age consumed less food, regurgitated more frequently, and had lower bill and mass growth rates than uninfected control chicks (Spalding et al. 1994). Regurgitation or attempts to regurgitate may be associated with pain from parasite perforation (Measures 1988c; Spalding and Forrester 1993). Reports of clinical signs in other avian families are limited. Red-breasted Mergansers had severe nervous spasms of the head and neck and ocular twitching just before death (Locke et al. 1964).

PATHOLOGY

Gross Pathology

Lesions in species of Ardeidae infected with *E. ignotus* have been described by several authors (Locke 1961; Roffe 1988; Schaffer et al. 1990; Spalding and Forrester 1993; Ziegler et al. 2000; Pinto et al. 2004). Larvae that perforate the ventricular portion of the stomach generally cause minor local hemorrhage. In very small chicks, there can be significant hemorrhage associated with the perforation. Larvae that have recently perforated the stomach can be seen either free in the coelom or just below the serosa. In subacute infections, parasites are surrounded by a thin, friable tube composed of fibrin and cellular debris of host origin and bacteria. At this stage, the parasites can easily be removed intact. The tubules in more chronic infections (Figure 16.5) are larger, prominent, firm, and are composed of granulomatous fibrosis. The tubules of adult nematodes, especially large females, are frequently twisted and contain 180° turns, making removal of intact adult females almost impossible. Fibrous tubules that adhere to the ventral abdominal wall may be visible or palpable on the exterior of the abdomen. Tubules communicate with the lumen of the stomach and rarely the small intestine through a raised portal with either the head or the tail of the worm protruding. Adhesions and penetrating tubules may also involve intestine, liver, airsacs, gall bladder, proventriculus, cloaca, abdominal wall, pancreas, and pericardium in decreasing frequency. The tubules remain when parasites die and gradually resolve to irregularly shaped, firm, often black nodules on the serosal surface of the stomach. In very severe chronic cases, the entire abdominal contents may be woven together with an inseparable mat of fibrous tubules. Bacteria cultured from the abdominal cavity and various organs can include a wide range of species typically found in the intestinal tract of fish-eating birds (Wiese et al. 1977; Roffe 1988; Spalding and Forrester 1993; Ziegler et al. 2000).

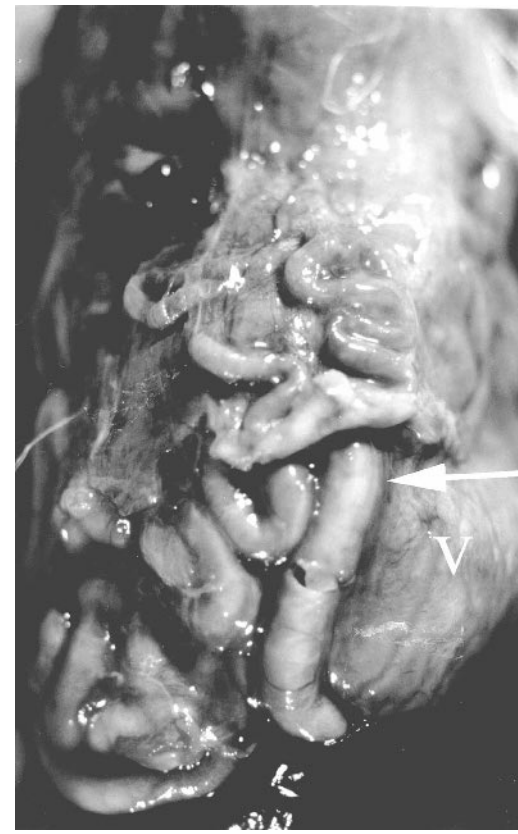


Figure 16.5. Chronic eustrongylidosis in a Snowy Egret (*Egretta thula*). Note tubules (arrow) on the surface of the ventriculus (V). The smaller tubules are from a more recent infection. From Spalding and Forrester (1993), with permission of the *Journal of Wildlife Diseases*.

Lesion appearance may be more dependent on host species than on parasite species. For example, a much more generalized and severe peritonitis occurs in nestling White Ibises (family Threskiornithidae) than in species of nestling Ardeidae (Spalding and Forrester 1993). Lesions similar to those caused by *E. ignotus* occur in Great Blue Herons and Common Loons infected with *E. tubifex*, but appear as nodular masses in the proventriculus of mergansers (Measures 1988c). Nothing is known about whether hosts respond immunologically to the parasite, the bacteria or gastric fluids, or to the tissue damage associated with the perforation.

Measures (1988c) described the nodular or cyst-like lesions found in the proventriculus of mergansers

infected with *E. tubifex* to be 5 mm to 3 cm in diameter and projecting into the peritoneal cavity (Figure 16.6). The nematodes communicated with the lumen of the proventriculus. It is interesting to note that von Brand and Cullinan (1943) were not able to infect ducks and chicks *per os* and speculated that the parasites were killed in the grinding gizzard (ventriculus). That may explain why lesions in mergansers (with grinding gizzards) develop in the proventriculus and esophagus, whereas *E. ignotus* perforates the nongrinding, poorly muscled ventricular stomach in species of Ciconiiformes. It does not explain, however, why cystlike lesions occur in species of Pelecaniformes as they do with *E. excisus* in cormorants.

Lesions caused by *E. excisus* in the proventriculus of cormorants are similar to those produced by *E. tubifex* (Ciurea 1938). Nematodes are coiled in a capsule that projects from the serosal surface like a pea with the head and tail ends protruding from the mucosal surface into the lumen. During the course of infection, the middle portion of the nematode quickly becomes covered with a connective tissue capsule. Dubinin (1949) also described nematodes in the proventriculus, between the glandular and muscular layers with the anterior and posterior ends in the lumen.

More severe and fatal lesions were described by Locke et al. (1964) in Red-breasted Mergansers during a die-off in Virginia. Most had peritoneal hematomas with nematodes burrowing into and causing destruction of the liver, especially the left lobe, the proventriculus, and airsacs. Involvement of the lung, heart, and kidney was less severe.

Microscopic Pathology

Acute lesions in species of Ardeidae infected with *E. ignotus* are generally associated with larval stages and located in the tunica muscularis or below the serosa of the stomach (Figure 16.7) (Locke 1961; Roffe 1988; Spalding and Forrester 1993). Occasionally in very early infections, or in severely debilitated birds, no inflammation is present. More commonly and in more subacute lesions, the parasite is surrounded by fibrin with associated erythrocytes, granulocytes, and monocytes. Subserosal hematomas are frequent (Spalding and Forrester 1993). The parasites are easily distinguished from other nematode species and have prominent ventral cords, coelomyarian musculature, and a pseudocoelomic membrane (Figure 16.7).

Chronic lesions are observed more frequently and are characterized by a parasite within the lumen of a tubule surrounded by cellular debris, free bacteria, and eustrongylid eggs. Often the cuticle remains, surrounding the nematode. In some cases, degenerate pieces of nematode are present. The tubule itself is composed of a prominent layer of fibrous connective tissue lined by multinuclear giant cells on the luminal side. Fibrin can often be seen on the peritoneal surface.

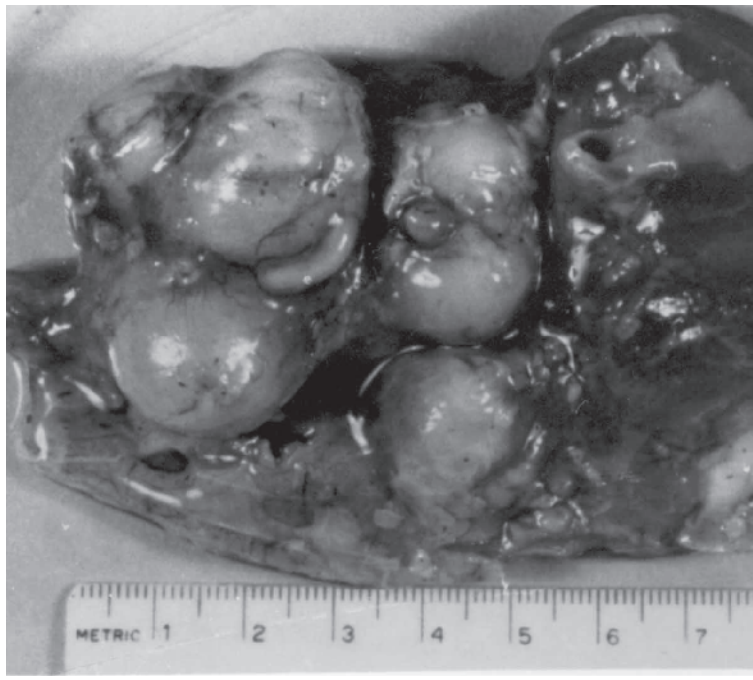
Chronology of experimental *E. tubifex* infections in Common Mergansers (*Mergus merganser*) has been described in detail by Measures (1988c). Fibrinous tubes on the serosal surface of the proventriculus are evident at 2 days postinfection. By 6 days postinfection, the tubules are surrounded by a fibrous connective capsule that contains inflammatory cells, bacteria, and erythrocytes. As these encapsulated lesions expand, they compress the proventricular glands. By 30 days postinfection, only resolving granulomas are present.

We found no microscopic descriptions for infections with *E. excisus.*

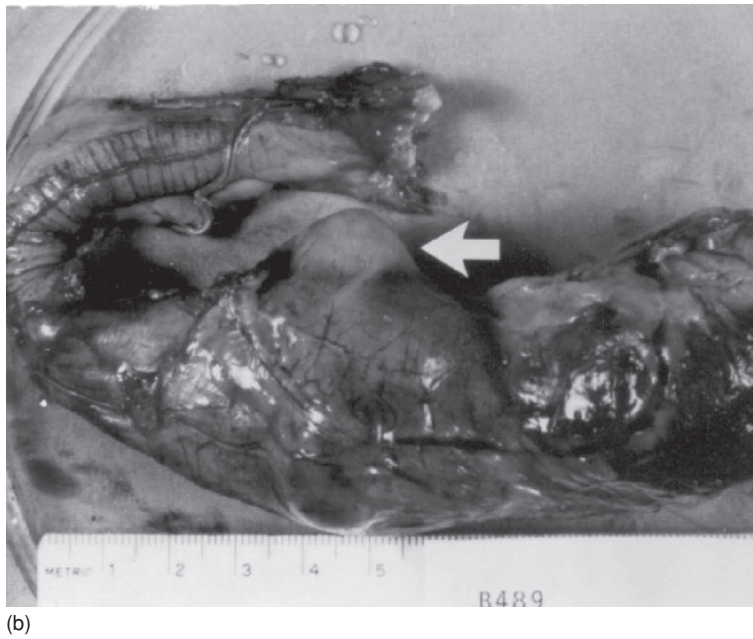
DIAGNOSIS

Diagnosis in species of live Ciconiiformes is possible. Palpation of the abdomen can be quite reliable in species of Ardeidae if the lesions are subacute to chronic (Spalding 1990). The tortuous tracts can also be seen sometimes on radiographs and by laproscopy or laparotomy. Necropsy remains the most common method of diagnosis. Once observed, the tubular serpininous tracts over the surface of the stomach and intestines and occasionally coursing through organs are pathognomonic. When the parasite dies, the tracts degenerate into black multifocal nodules adherent to the serosa, indicating a chronic resolved infection. The only possible confusion is with occasional perforating ascaroid nematodes or foreign object perforation, which never cause the characteristic serpiginous tracts of eustrongylidosis. These tracts have only been described thus far in birds of the order Ciconiiformes. Patent infections can be demonstrated by direct microscopic examination of feces or by fecal flotation techniques. This method frequently results in false negatives (Spalding 1990). When present, the eggs are highly crenulated, making the diagnosis relatively easy (Figure 16.8).

Identification of the nematodes to species can be made by light microscopy by an experienced specialist, but is best made by examination of adult nematodes by scanning electron microscopy. Female specimens of *E. ignotus* are distinguished from those of *E. tubifex* by the larger size of the six inner circle labial papillae, but are difficult to differentiate from female specimens of *E. excisus* (Figure 16.2). Male specimens of *E. ignotus* have a caudal sucker that lacks a cuticular cleft, while a cuticular cleft is present in the caudal sucker of male specimens of *E. excisus*. Male specimens of *E. tubifex* have projections on the inner perimeter of the cuticular hem, while male *E. ignotus* do not (Figure 16.3).



(a)



(b)

Figure 16.6. Gross lesions caused by *Eustrongylides tubifex* in a Common Merganser (*Mergus merganser*). (a) Note the large tumors on the serosal surface of the proventriculus. (b) The nodules protrude from the serosal surface. From Measures (1988c), with permission of the *Canadian Journal of Zoology*.

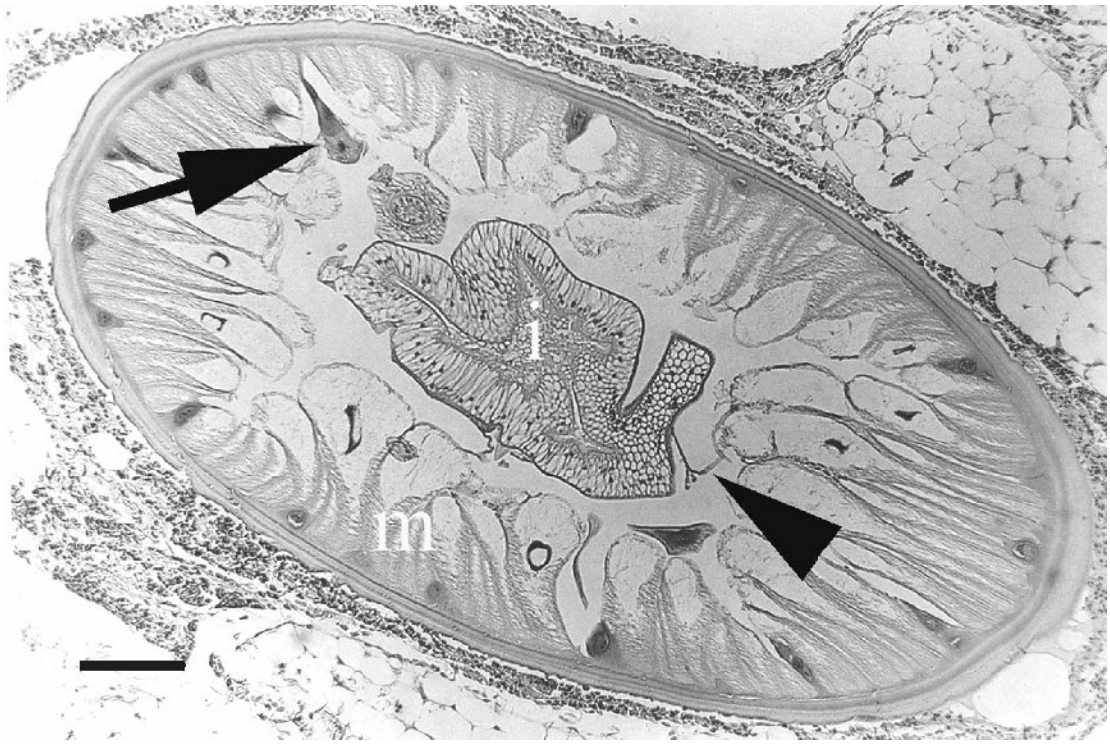


Figure 16.7. Cross section of a fourth-stage larva in adipose tissue below the serosa of the ventriculus of a Great Blue Heron (*Ardea herodias occidentalis*). Note the large ventral chord (arrow), the coelomyarian musculature (m), pseudocoelomic membrane (arrowhead), and intestine (i). Bar = 100 μm. From Spalding and Forrester (1993), with permission of the *Journal of Wildlife Diseases*.

Additional electron micrographs of nematodes can be found in Measures (1988a) and Coyner et al. (2003a).

Diagnosis in fish to the level of species is difficult. Larval stages of *E. ignotus* and *E. tubifex* are illustrated by Coyner et al. (2003a) and Measures (1988b), respectively. Larvae in fish can be distinguished from *Dioctophyma renale* by size, cephalic and reproductive morphology, and the tendency for *E. ignotus* to be associated with the mesentery rather than the musculature (Lichtenfels and Madden 1980; Measures and Anderson 1985; Measures 1988b). Small fish that are infected with larval *Eustrongylides* have swollen abdomens that affect appearance when swimming. In advanced infections, the coiled L4 larvae can be seen through the abdominal wall (Figure 16.9).

PUBLIC HEALTH AND DOMESTIC ANIMAL CONCERNS

Humans can become infected and die from ingesting poorly cooked or raw infected fish, but only a few reports exist (Table 16.3). Generally humans do not eat raw fish, or if they do, the parasites are easy to see if the flesh is carefully examined. Most reported cases are the result of fisherman eating baitfish that are usually collected from locations with high fish density and risk of infection with larval worms is high. In humans, the perforation of the intestinal tract is very painful. One patient underwent surgery for appendicitis, whereupon a larval eustrongylid was discovered in the peritoneal cavity (Gunby 1982; Arias 1989).

There are no reports of infections in domestic animals, although consumption of raw fish by cats and dogs may lead to infection.

WILDLIFE POPULATION IMPACTS

Eustrongylidosis is probably the most common cause of disease-related mortality in nestling wading birds from North America, but is frequently underreported. Similar mortality likely goes unnoticed throughout the range of *E. ignotus* and other species. Although

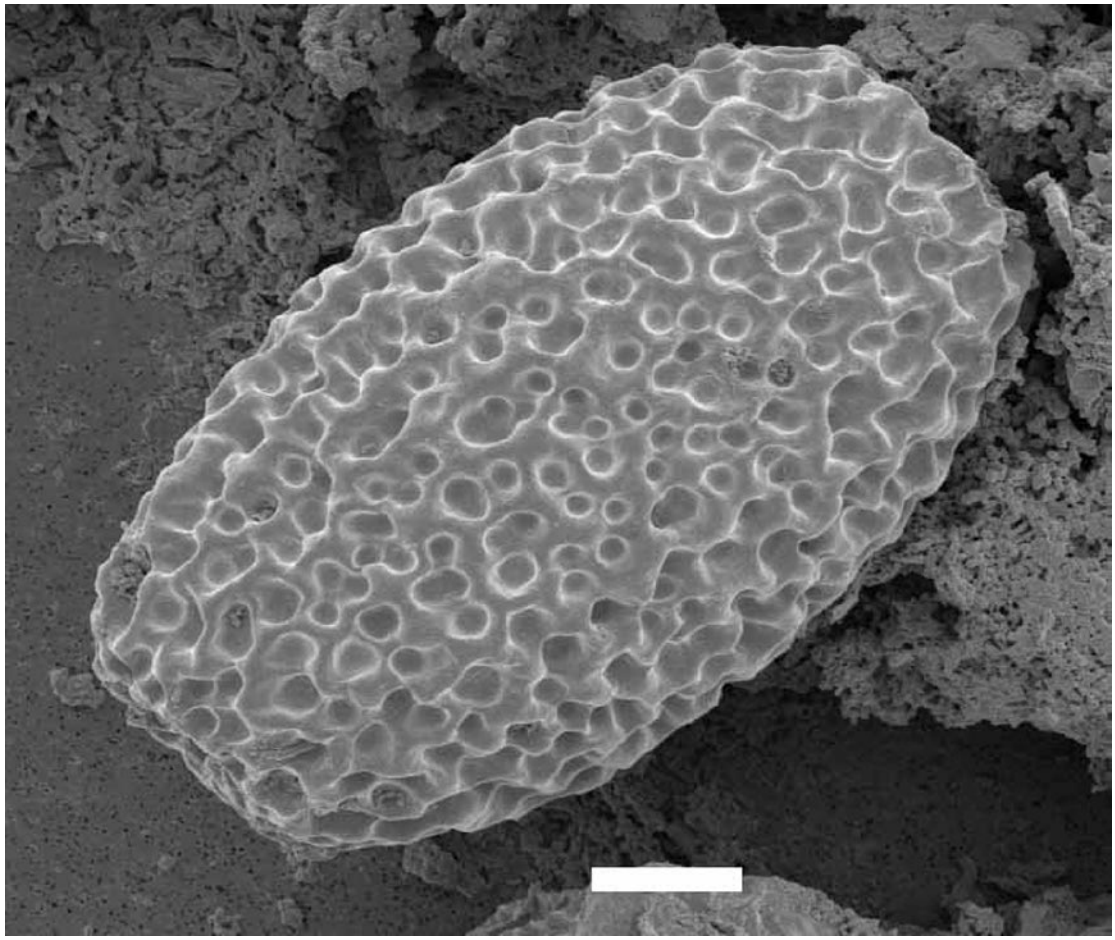


Figure 16.8. Electron micrograph of the egg of *Eustrongylides ignotus*. Note the depressions. These depressions can also be seen at the light microscope magnification. From Coyner et al. (2003a), with permission of the *Journal of Parasitology*.

the prevalence of *E. ignotus* infections in species of Ardeidae is high, morbidity, mortality, and effects on recruitment of young birds into the breeding population are less well studied. Most mortality occurs within colonies that are visited rarely. Presence of dead nestlings is often not considered alarming because wading birds routinely lay and hatch more nestlings than will fledge. As a result, this group of parasites has gone relatively unnoticed as a cause of significant and increasing wildlife mortality at the population level.

Eustrongylides tubifex causes rare sporadic mortality in mergansers. It is interesting that despite the availability of prevalence data and intensive life history work on these parasites, there is no indication that either *E. tubifex* or *E. excisus* have significant effects on the health of their avian hosts. However, like *E. ignotus* in species of Ardeidae, such information may go unreported until an interest is taken in the health of the avian species involved.

TREATMENT AND CONTROL

Treatment of infected birds with anthelmintics has not been tried in any systematic fashion and is unlikely to be practical. Mortality of large numbers of nematodes within the host might do more harm than good.

MANAGEMENT IMPLICATIONS

The presence of nutrient pollution in waters used by wading birds increases risk for eustrongylidosis and



Figure 16.9. Eastern mosquitofish (*Gambusia holbrooki*) with a single eustrongylid larva (top) which has been dissected from the coelomic space and removed from its capsule (bottom). From Spalding et al. (1993), with permission of the *Journal of Wildlife Diseases.*

ultimately nest failure. Nutrients increase fish density and thus indirectly increase the attraction of wading birds to a potentially hazardous site. They also enhance conditions for completion of the parasite life cycle.

Reduction or elimination of nutrient release into water bodies that can be used by wading birds may be the most practical large-scale management option. Wading birds are attracted to such sites because increased productivity results in higher fish densities (Frederick and McGehee 1994). Coyner et al. (2003b) documented the decline of infected fishes in an urban watershed that coincided with the decline of nutrient pollution, suggesting that this may be an effective solution. Another approach can be to exclude or discourage birds from using sites with nutrient-rich polluted water by preventing access or by ensuring that wastewater releases occur in locations unlikely to be visited by wading birds.

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17
Trichostrongylus

Daniel M. Tompkins

INTRODUCTION

Members of the genus *Trichostrongylus* are small, fine, reddish worms that occur primarily in the ceca, but also the small intestine of birds, as well as the small intestine of rodents, lagomorphs, and ruminants. Two species are currently described from birds. Infection in wild birds has been across Europe, Asia, North America, Africa, and Australasia, reported primarily in Anseriformes (waterfowl) and Galliformes (fowl), but also in Gruiformes (cranes) and Otidiformes (bustards). Species of *Trichostrongylus* are monoxenous, requiring only a single host to complete the life cycle. The free-living, infective third-stage larvae tend to crawl onto vegetation where they are ingested.

As with many other gastrointestinal parasites, intense infections of *Trichostrongylus* worms can cause morbidity, with signs that include loss of appetite, plumage dullness, and emaciation (Wehr 1971). However, intense infections such as these tend to occur naturally only in Red Grouse (*Lagopus lagopus scotica*, referred to as Willow Ptarmigan in *The Clements Checklist of Birds of the World*) infected with *Trichostrongylus tenuis* on mainland UK shooting estates. Here, prevalence of infection is almost 100% (Hudson 1992), geometric average worm burdens have been estimated at 2,471 per bird (Webster 2005), and parasite impacts on both individuals and populations have been characterized (Hudson 1986a; Shaw and Moss 1990; Dobson and Hudson 1992; Hudson et al. 1992a, 1998; Delahay and Moss 1996). Infection of *T. tenuis* in Red Grouse was also the first system in which regulation of host population size by parasites was experimentally demonstrated in the field (Hudson et al. 1998; Tompkins and Begon 1999).

SYNONYMS

Cecal strongyle, caecal threadworm, grouse disease, hairworms, trichostrongylosis, trichostrongyliasis.

HISTORY

Trichostrongylus tenuis (Mehlis, *in* Creplin 1846) Raillet and Henry, 1909, was the first of the avian *Trichostrongylus* species to be described, originally as *Strongylus tenuis* from the European Ring-necked Pheasant *(Phasianus colchicus)*. Other synonyms for *Trichostrongylus tenuis* included *Strongylus pergracilis* Cobbold, 1873; *Strongylus serratus* Linstow, 1876; *Trichostrongylus pergracilis* (Cobbold 1873) Raillet and Henry, 1909. *Trichostrongylus tenuis* was subsequently recognized as the trichostrongylid inhabiting the ceca of many Old-World birds, although it was first described in Red Grouse as *T. pergracilis* (Cobbold 1873) Raillet and Henry, 1909. Cram (1925) applied the latter name to the cecal worm from the Northern Bobwhite (*Colinus virginianus*) in the US. She later recognized two species: *Trichostrongylus tenuis* from 10 bird species (including waterfowl) and *T. pergracilis* from the Northern Bobwhite and Red Grouse (Cram 1927). After further study of material from many different hosts, including the type host (Ring-necked Pheasant) and from Red Grouse, the type host of *T. pergracilis*, Cram and Wehr (1934) synonymized both taxa as *T. tenuis*. Recently, however, the trichostrongylid infecting Northern Bobwhite has been recognized as a distinct North American species, *Trichostrongylus cramae*, with the name *T. tenuis* reserved for Old-World hosts (Durette-Desset et al. 1993). This designation has been independently supported by experimental challenges of Northern Bobwhites, in which infective *Trichostrongylus* larvae of Northern Bobwhite origin readily established while those of Red Grouse origin failed to do so (Freehling and Moore 1993). In light of this, the status of species of *Trichostrongylus* infecting other wild birds in North America needs reassessment (Anderson 1996), a process that has already started.

Trichostrongylus tenuis has been associated with losses in managed populations of Red Grouse in England and Scotland for more than 100 years (Cobbold 1873; Shipley 1909; Lovat 1911). The development and transmission of *T. tenuis* in Red Grouse was the

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subject of an in-depth investigation by several authors in the early 1900s. In their collated findings (Leslie and Shipley 1912), the authors relate the prevalence, intensity, and pathogenicity of *T. tenuis* to "grouse disease" and fluctuations in grouse numbers on moors in the UK. Most of the basic findings reported by these authors have been confirmed by more recent investigations, culminating in the classic experimental demonstration of the role that *T. tenuis* plays in regulating the population size of Red Grouse in the wild (Hudson et al. 1998).

ETIOLOGY

The genus *Trichostrongylus* belongs to the superfamily Trichostrongyloidea, the largest superfamily in the group known as the "bursate nematodes" (order Strongylida). Avian species of *Trichostrongylus* are readily observed under the microscope as small, fine reddish worms, after extraction (usually through sieves) from the cecal and intestinal contents (see Doster and Goater 1997 for extraction methods). Adult male worms are approximately 4–8 mm long and adult females are 6–10 mm, with width gradually increasing from approximately 10 μm at the anterior end to approximately 50 μm in front of the caudal bursa (Figure 17.1). The size and tapered morphology of these worms, in addition to characteristic cuticular striations (see below), makes them unlikely to be confused with other helminths found in the ceca and small intestine of birds.

Two characters can be easily and reliably used to distinguish adult *T. tenuis* from *T. cramae* (Durette-Desset et al. 1993). First, transverse striations on *T. tenuis* start from behind the excretory pore of both sexes and cover three- to four-fifths of the body surface. While male specimens of *T. cramae* also have pronounced transverse striations, they are present in females only on 35–55 μm of the cephalic region in front of the secretory pore (Figure 17.1a). Second, the ratio of the distance between bursal papillae 3 and 4 to the distance between papillae 2 and 3 for *T. tenuis* ranges from 1:2.2 to 1:3.9. In *T. cramae*, the ratio ranges from 1:1.3 to 1:1.7 (Figure 17.2b). The two species also differ in the configuration of the caudal bursa dorsal array (Durette-Desset et al. 1993).

EPIZOOTIOLOGY

Being monoxenous (requiring only a single host), the life cycle of avian species of *Trichostrongylus* is relatively simple, consisting of egg, four larval stages, and adult (Figure 17.2). Unlike other parasites in the superfamily Trichostrongyloidea, paratenic "transport" hosts are unknown for species of *Trichostrongylus*. For *T. tenuis*, eggs measuring approximately 75 × 46 μm are excreted in the fecal droppings of infected hosts at the morula (64 cells) stage of development. Eggs are not resistant to desiccation or extreme cold (−15°C), with little development occurring at temperatures below freezing point (Shaw et al. 1989; Connan and Wise 1994). In moist feces at suitable temperatures (>5°C), eggs hatch in approximately 36–48 h. Emerging first-stage larvae measure approximately 360 μm in length. Larvae molt into the second stage after approximately 36–48 h, and then into the third (infective) stage after 8–16 days, depending on temperature. Like the eggs, the larvae are susceptible to desiccation and extreme cold. Continuous temperatures of −15°C are lethal to infective larvae within 12 days (Shaw et al. 1989; Connan and Wise 1994). However, they are capable of withstanding winter temperatures on grouse moors in Yorkshire, England, where temperatures fall as low as −10°C), and remain infective for up to 3 weeks (Connan and Wise 1994). However, little to no transmission to new hosts occurs during the winter months (Shaw 1988). Similar environmental limits on infection are seen with infections of *T. tenuis* in wild Red-legged Partridges (*Alectoris rufa*) in Spain, where parasite abundance is inversely correlated to latitude and directly correlated to yearly mean temperature (Calvete 2003).

Host infection by species of *Trichostrongylus* is via ingestion of third-stage larvae that are still sheathed by the cuticle of the second stage; skin penetration is unknown in the genus. For *T. tenuis* on UK moors, third-stage larvae have been experimentally demonstrated to crawl to the growing tips of moist heather plants (*Calluna vulgaris*), the staple diet of adult Red Grouse. Here, they increase their probability of infecting a suitable host by accumulating in drops of water at the most likely point of ingestion by an adult grouse. Movement to heather tips is governed by negative geotaxis, positive phototaxis, and may also be influenced by responses to chemical cues given off by heather (McGladdery 1984; Saunders et al. 2001). The resultant pattern is a highly aggregated distribution of infective larvae among heather plants in the field (Saunders et al. 1999, 2000). Larvae are more available on plants during daylight hours in experimental diurnal periods and on plants collected from the field in the afternoon rather than the morning, indicating that larvae migrate back down plants at night (Saunders et al. 2000). This behavior most likely evolved to avoid desiccation. Indeed, time-series analyses suggest that some of the year-to-year variation in parasite egg numbers that are passed by Red Grouse is explained by rainfall in the previous summer, likely caused by greater transmission during wetter summers (Moss et al. 1993). Furthermore, there is strong evidence that the interactions between *T. tenuis* and climate act to synchronize the dynamics of Red Grouse

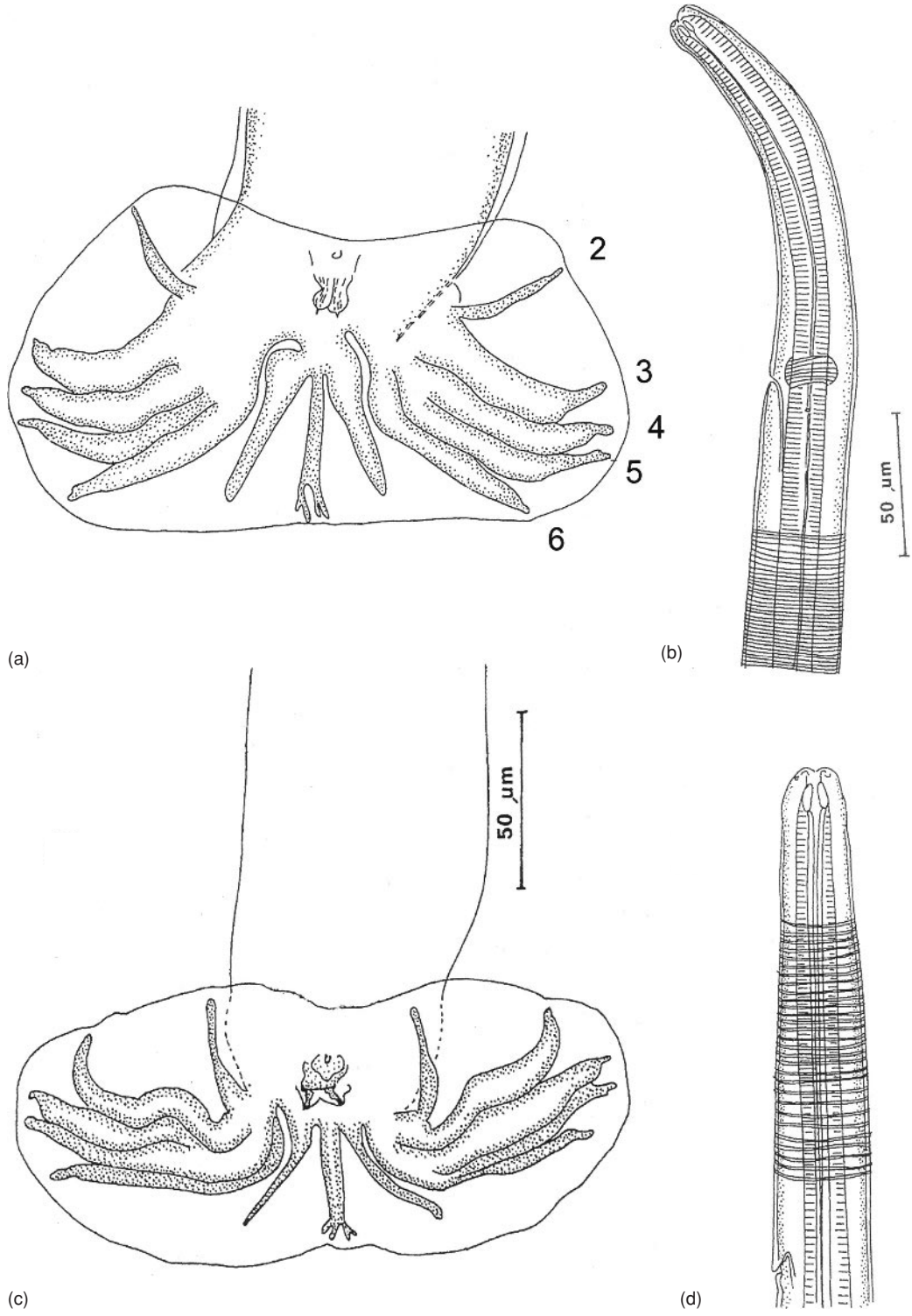


Figure 17.1. (a) *Trichostrongylus tenuis* caudal bursa, ventral view. (b) *T. tenuis* female, anterior end, lateral view. (c) *Trichostrongylus cramae* caudal bursa, ventral view. (d) *T. cramae* female, anterior end, lateral view. Numbers on part (a) denote ray papillae. Modified from Durette-Desset et al. (1993), with permission of *Annales de Parasitologie*.

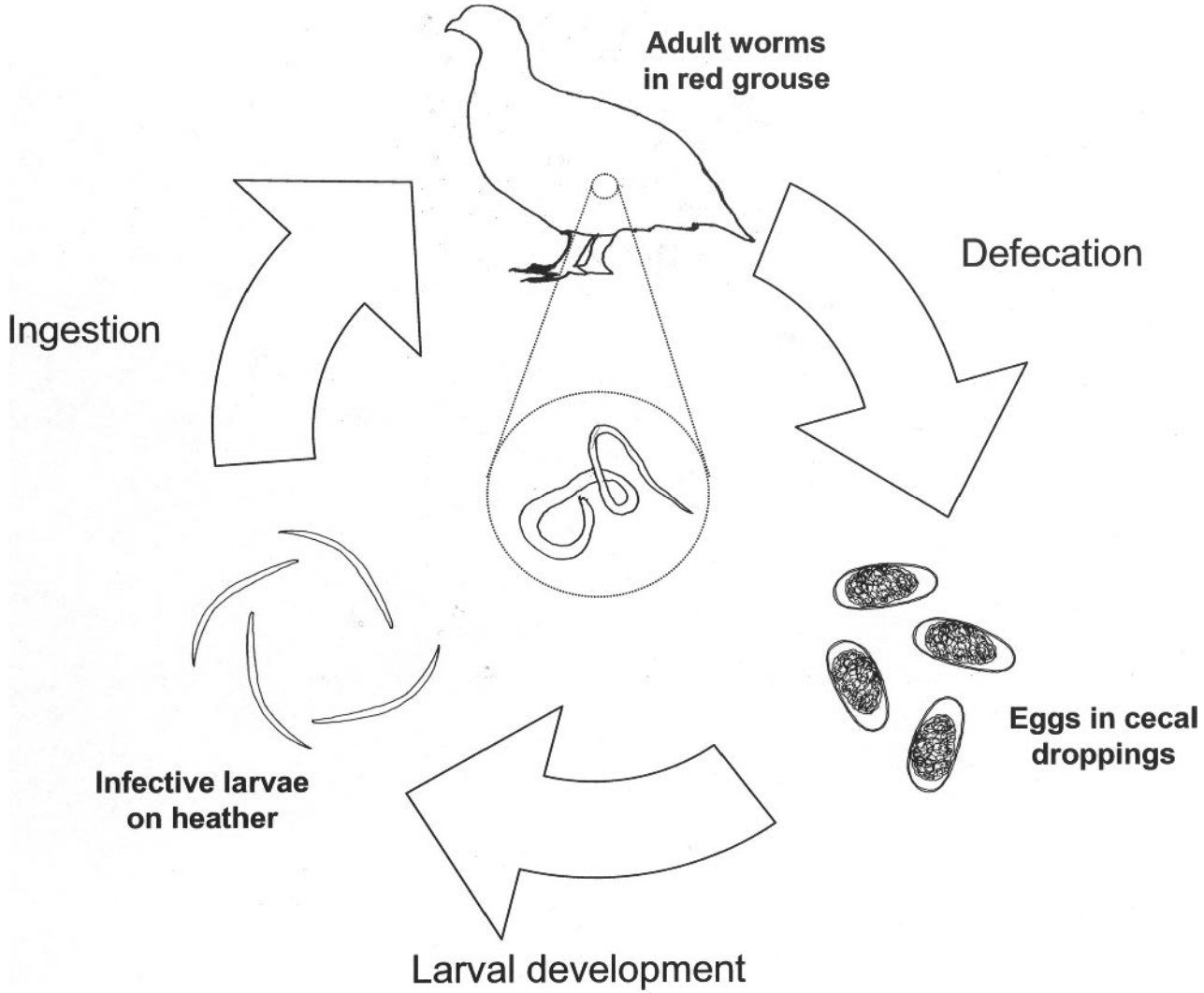


Figure 17.2. Life cycle of *Trichostrongylus tenuis* infecting Red Grouse (*Lagopus lagopus scotica*). Parasites are not drawn to scale.

populations in northern England (Cattadori et al. 2005).

Exsheathment, the process by which a third-stage larva escapes from the cuticle of the second stage, occurs within a few days of ingestion. Unsheathed third-stage larvae invade the small intestine and cecal mucosa (Shaw 1988). At this point, as with other species of *Trichostrongylus*, the larvae may either develop directly into fourth-stage larvae and then return to the gut lumen where they mature into adults within a few days, or undergo a period of "arrest" at the third stage within the mucosa. In his analysis of the larval parasite population present within individual grouse in different seasons, Shaw (1988) provides strong evidence for this "arrestment" by *T. tenuis* in the UK. In late summer, most larvae are in the fourth stage but some third-stage larvae are sheathed, indicating that transmission is still taking place. In winter, ensheathed larvae are absent but exsheathed larvae are present, suggesting that few new larvae are being ingested during this period and that the exsheathed third-stage larvae are arrested. In spring, the proportion of third-stage larvae decreases significantly in the parasite population and sheathed larvae are absent, indicating the development of overwintering third-stage larvae to fourth and adult stages. This synchronized development results in a "spring rise" in worm egg production that is evident in grouse feces (Shaw 1988; Shaw et al. 1989). A second peak of recruitment into the adult worm population occurs during late summer that reflects increased numbers of larvae on heather in the preceding weeks. These increases are associated with optimal conditions for transmission where mean temperatures are in the range of 10–15°C and mean monthly rainfall is in the range of 20–50 mm.

When development is direct for *T. tenuis* in Red Grouse, the prepatent period between ingestion of infective stages and the appearance of parasite eggs in the feces is approximately 7–8 days. Regardless of whether development is direct or arrested, however, the resulting distribution of parasites among hosts retains the characteristic aggregation of macroparasite infections (Shaw and Dobson 1996). Few host individuals have many parasites, while most host individuals have few parasites (Wilson 1983; Hudson et al. 1992a). Infected host individuals can carry worm intensities of up to 24,000 worms (Hudson 1986b).

Adult worms favor the proximal region of the cecal mucosa, threading themselves into the mucosal tissue with anterior and posterior ends protruding into the cecal lumen (Watson et al. 1987, 1988). Eggs are produced at a rate of approximately 5×10^5 eggs per worm per year (Shaw and Moss 1989a; Hudson et al. 1992a). Adult worms can survive for more than 2 years, although parasite egg output decreases with age of the worm population, especially in winter (Shaw and Moss 1989a). There is no evidence of density-dependent suppression of parasite establishment or egg production at high worm intensities (Hudson and Dobson 1997; Seivwright et al. 2004).

There are notable differences between the biology of *T. tenuis* in Red Grouse and the biology of *T. cramae* in Northern Bobwhites (Freehling and Moore 1993). While *T. tenuis* produces a chronic infection in Red Grouse that increases throughout the life of the bird (Potts et al. 1984), *T. cramae* in Northern Bobwhites in northern Florida is a seasonally occurring parasite. Moore et al. (1986) demonstrated a winter peak in acquisition of larvae that was followed by a peak in adult worms, with a decrease in prevalence and intensity occurring during warmer months. Infections of *T. tenuis* in other host species also appear to be more similar to those of *T. cramae*, with the high burdens and chronic infections observed in Red Grouse likely being an exception rather than the rule (Holmstad et al. 2004; Millan et al. 2004; Fedynich et al. 2005; Schei et al. 2005; Webster et al. 2007). This may very well be a product of the unnaturally high densities of managed Red Grouse populations in the UK.

CLINICAL SIGNS

Hosts carrying high intensities of parasites show evidence of appetite loss, malnutrition (including dullness of the plumage), diarrhea, and emaciation ("grouse disease"). Among Red Grouse, signs peak during spring, coinciding with the "spring rise" in worm egg production, and in the autumn in young hatch-year birds.

PATHOLOGY

Being threaded into the cecal mucosa, adult worms cause trauma, atrophy, and flattening of the epithelial cells. This likely interferes with the normal digestion of heather and other plant material (Watson et al. 1987, 1988). As is common for other cecal nematodes, heavy infections may cause hemorrhagic typhlitis, and in chronic cases, the contents of the ceca may contain a yellowish white material of cheesy consistency.

Since the remarkably large ceca of Red Grouse play a key role in food digestion and nutrient recovery in this species (Hudson 1986b), the disruption in normal cecal function by high-intensity infections with *T. tenuis* leads to a loss of body condition, increased mortality (Hudson 1986b; Hudson et al. 1992a), and decreased fecundity with reductions in both clutch size and brood size (Hudson 1986a; Shaw and Moss 1990; Hudson et al. 1992a; Delahay and Moss 1996). Negative associations between the abundance of *T. tenuis* and host body mass, body condition, and breeding survival have also been reported for Willow Ptarmigan in Norway

(Holmstad et al. 2005). For *T. cramae*, no impacts related to disease have been noted on either Northern Bobwhites or Attwater's Prairie Chicken (*Tympanuchus cupido attwateri*), a subspecies of the Greater Prairie-Chicken (*Tympanuchus cupido pinnatus*) (Purvis et al. 1998), although *T. cramae* has been recorded at a mean intensity of 1,019 worms per bird in the latter species (Peterson et al. 1998). The pathological effects of these infections should be investigated further in this species.

Both redness of the comb of male birds (a secondary sexual character of Red Grouse that functions in both intra- and intersexual selection; Redpath et al. 2006a) and the concentration of plasma carotenoids (the molecules responsible for the redness; Mougeot et al. 2007a) negatively correlate with abundance of *T. tenuis* (Mougeot et al. 2005a, 2007b). This link has been experimentally confirmed, through observations of plasma carotenoid concentration and comb redness both increasing following reduction of infections with *T. tenuis* (Martinez-Padilla et al. 2007) and decreasing following challenge with *T. tenuis* (Mougeot et al. 2007b).

DIAGNOSIS

The recovery of parasite individuals as fragments in feces or, preferably, intact parasites from postmortem examinations is required for reliable diagnosis. Infective stages (eggs or larvae in feces) are also highly suggestive of infection. However, diagnosis based on infective stages should be confirmed by a postmortem examination to verify the presence of adult *Trichostrongylus* in the cecae and to establish the extent of infection and damage. Fecal egg counts can be used as a reliable estimate of relative parasite intensity among individuals at the same time of year (Shaw and Moss 1989a; Seivwright et al. 2004), but should not be considered reliable enough to assess impacts on populations.

Presence of parasites alone is not indicative of disease in infections with *Trichostrongylus*. The impacts of disease are only likely to be common when intensity of infection is in the thousands of worms per individual, as has been observed in the field for *T. tenuis* in Red Grouse and *T. cramae* in Attwater's Prairie Chicken.

IMMUNITY

Watson et al. (1988) demonstrated that young domestic chickens acquire resistance to *T. tenuis* on repeated exposure to the parasite, such that artificially induced infections are rejected and worms are actively expelled. These findings are supported by observations of higher parasite prevalence and intensity in growers as opposed to adult free-range chickens (Magwisha et al. 2002). While the potential immune responses of wild birds to infection by *T. cramae* need further investigation, studies with Red Grouse have found no evidence of such "acquired" immunity to infection. Here, the number of worms present in the cecae increases throughout the life of the bird (Wilson 1983). A similar increase with age has also been observed in infections of *T. tenuis* in Red-legged Partridges in Spain (Millan et al. 2004). In a study on transmission dynamics and host–parasite interactions, Hudson and Dobson (1997) used age-intensity data to show that the rate of uptake of *T. tenuis* by Red Grouse increases during the first 6 months of a bird's life, reflecting an increase in feeding rate with age, with no sign of self-cure. Furthermore, reinfection rates of adults that were treated to reduce parasite intensities were not significantly different from infection rates of naïve immature grouse. This continued rise of infection, observed over 18 months, demonstrates a lack of any strong host-mediated response against the parasite. However, Red Grouse do vary in their innate susceptibility to infection with *T. tenuis*, with wide and repeatable variation in individual resistance (Shaw and Moss 1989b). The intensities of *T. tenuis* and other parasite species positively covary in the Willow Ptarmigan, suggesting that repeatable variation in innate susceptibility to gastrointestinal nematode infection also occurs in this species (Holmstad and Skorping 1998).

Susceptibility to infection by *T. tenuis* also varies with season in Red Grouse, with rates of parasite establishment in cocks being higher in autumn than in summer. Shaw and Moss (1989b) hypothesized that this is likely linked with behavior, since cocks show higher levels of territoriality in the autumn. Increased corticosteroid levels from stress associated with such behavior could lower resistance to parasites. Recent research has partly supported this hypothesis, indicating that elevated testosterone (not corticosteroid) levels in male Red Grouse increases susceptibility to infection by *T. tenuis* (Mougeot et al. 2005b, 2006; Seivwright et al. 2005). Furthermore, the opposing effects of testosterone and *T. tenuis* on carotenoid availability in Red Grouse, whereby infection by *T. tenuis* decreases availability (as noted above) while testosterone enhances comb redness but tends to deplete plasma carotenoids (Mougeot et al. 2007b), likely makes the red comb an "honest" signal of male health for mate choice by females (Mougeot et al. 2004).

DOMESTIC ANIMAL HEALTH CONCERNS

Avian species of *Trichostrongylus* tend to occur only at low prevalence in domestic poultry, geese, and ducks, with infected individuals tending not to exhibit signs of disease (Svoboda 1992; Romaniuk and Lipinski 1999; Poulsen et al. 2000; Magwisha et al. 2002; Irunga et al. 2004). Cross-infection from wild birds can occur,

although prevalence is generally low without the need for preventative management.

WILDLIFE POPULATION IMPACTS

The genus *Trichostrongylus* has been detected in many wild populations of Galliformes and Anseriformes, although, as mentioned above, high intensities of infection have only been observed for *T. tenuis* infecting Red Grouse and *T. cramae* infecting Attwater's Prairie Chicken (Hudson 1986b; Watson and Shaw 1991; Holstadt et al. 1994; Peterson et al. 1998; Purvis et al. 1998; Millan et al. 2004). To date, impacts of such high-intensity infections have only been demonstrated in Red Grouse—disease impacts of *T. cramae* on Attwater's Prairie Chicken require further investigation (Peterson 2004).

For Red Grouse with infections of *T. tenuis*, the direct effects of infection on mortality and fecundity interact with other factors to impact individual hosts. Heavily infected birds also experience increased predation, most likely due to increases in emission of scent (Hudson et al. 1992b; Dobson and Hudson 1995). In addition, birds with intense infections are less able to maintain territories due to the energetic consequences of parasitism by *T. tenuis* (Delahay et al. 1995) and its interaction with testosterone (Mougeot et al. 2006). Indeed, when parasites are removed, aggressive territorial behavior increases (Fox and Hudson 2001; Mougeot et al. 2005c). Finally, parasite infection also influences mating success of male Red Grouse via the interactions with carotenoids discussed above.

Impacts of parasites at the scale of host populations have been both extensively modeled and experimentally demonstrated for *T. tenuis* in Red Grouse on heather moors in northern England. Populations of red grouse have characteristic cyclical dynamics, with cycle periods between 4 and 8 years. Involvement of *T. tenuis* in these cyclic dynamics has long been suspected, because of the association between population crashes and high parasite intensities recorded from individuals (Leslie and Shipley 1912). Dobson and Hudson (1992) demonstrated in an experimentally parameterized modeling framework that the interaction with *T. tenuis* was a potential cause of these cycles. They then proved that this was indeed the case by using anthelmintic application to drastically reduce the extent of population crashes at replicated sites (Hudson et al. 1998). This work was the first experimental demonstration of parasite regulation of host numbers in any wildlife population, but did not prove that the population dynamics observed were a function of *T. tenuis* parasitism alone (Tompkins and Begon 1999). Indeed, the weight of evidence from more recent research supports the hypothesis that it is an interaction between parasitism, aggressiveness, and territoriality (as observed at the individual scale) that drives the observed dynamics, with differences in the interaction driving observed differences in cyclic behavior of populations between different parts of the UK (Mougeot et al. 2005d; Redpath et al. 2006a, b).

TREATMENT AND CONTROL

Adult and developing worms can be eliminated via the oral application of standard anthelmintics, such as fenbendazole or levamisole hydrochloride (Samour 2000), although anthelmintic treatment is not highly effective against arrested larvae in the gut mucosa. Anthelmintic resistance has not yet been reported for species of *Trichostrongylus* of birds (Webster et al. 2007).

MANAGEMENT IMPLICATIONS

Trichostrongylus parasites are currently only of real concern to managed populations of Red Grouse in northern England and Scotland, where *T. tenuis* plays a role in cyclical population crashes. Although these populations rapidly recover following crashes, the game industry is interested in maintaining high numbers of birds at all times.

Wild populations containing infected individuals may also act as reservoirs, increasing risks of infection on sympatric populations of more susceptible host species. It has been hypothesized that such a relationship may occur in North America between Northern Bobwhite and the endangered Attwater's Prairie Chicken (Peterson 2004), although further research is required to investigate this idea.

Large-scale management of *T. tenuis* in Red Grouse, through the indirect application of anthelmintics via "medicated grit," has been experimentally proven at the estate scale. This treatment reduces parasite burdens in grouse hens and increases breeding success by improving chick survival (Newborn and Foster 2002). The increase in grouse numbers arising from the use of medicated grit is a viable method for enhancing hunting for Red Grouse, since the cost of treatment is low relative to the return from an increased harvest. Similar large-scale application of anthelmintics may also be applicable to the management of endangered species that are affected by infections with *Trichostrongylus*, particularly since anthelmintic resistance is not known for this genus of parasites in birds.

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18
Dispharynx, *Echinuria*, and *Streptocara*

Ramon A. Carreno

INTRODUCTION

Nematodes of the genera *Dispharynx*, *Echinuria*, and *Streptocara* are parasitic in the proventriculus and gizzard of many avian taxa. All three are spirurid nematodes belonging to the superfamily Acuarioidea, the acuarioid nematodes. The acuarioids are characterized by their large pseudolabia and modified cuticular structures at the anterior end known as cordons (Figure 18.1).

Twenty-eight acuarioid genera were listed by Chabaud (1975) and most are parasites of birds. Of the three genera that are discussed in this chapter, several species exist that are significant pathogens. *Dispharynx nasuta* is pathogenic mostly in passerine birds and Galliformes, while *Streptocara* species and *Echinuria uncinata* are pathogens primarily of waterfowl.

HOST RANGE AND DISTRIBUTION

Streptocara spp. and *Echinuria* spp. are parasitic primarily in waterfowl while *D. nasuta* is most frequently reported from the Galliformes, Columbiformes, and Passeriformes (Table 18.1). *Dispharynx nasuta* is the most widespread of the three and reports of *Echinuria* spp. and *Streptocara* spp. from Africa and South America are rare.

Dispharynx nasuta has been reported from Africa (Algeria, Congo, Egypt, Morocco, South Africa, Tunisia, Zimbabwe), Asia (China, Georgia, India, Kazakhstan, Pakistan, Russia, Turkestan, Uzbekistan), Australia, Central America (Costa Rica, Guatemala), Cuba, Europe (Austria, Denmark, France, Italy, Spain), Guam, North America (Canada, US), Puerto Rico, and South America (Argentina, Brazil, Ecuador, Venezuela). *Echinuria uncinata* has been reported from Africa (Algeria), Asia (Afghanistan, Azerbaijan, India, Japan, Russia), Australia, Europe (Czech Republic, France, Germany, Italy, Poland, Spain, UK), New Zealand, North America (Canada, US), and South America (Brazil). *Streptocara crassicauda* has been reported from Asia (Azerbaijan, China, Kazakhstan, Russia, Uzbekistan), Australia, Europe (Austria, Bulgaria, Denmark, France, Italy, the Netherlands, Norway, Spain, UK), and North America (Canada, US).

There is evidence that some acuarioid nematodes such as those parasitizing shorebirds have distinct distributions that correspond to migratory flyways (Anderson et al. 1996). However, pathogenic species of *Echinuria*, *Streptocara*, and *Dispharynx* are cosmopolitan in their distribution, probably because of their spread through domestic and migratory birds and because of the abundance of possible intermediate hosts that can support development of the parasites.

DISPHARYNX

SYNONYMS

Gizzard worms, proventricular worms, acuariosis, dispharagosis, dispharyngosis, dispharynxiasis, grouse disease.

HISTORY

The first description of *D. nasuta* was from the proventriculus of a House Sparrow (*Passer domesticus*) by Rudolphi in Austria (Goble and Kutz 1945). Both this and similar species were placed in various genera and subgenera until the genus *Dispharynx* was eventually erected by Skrjabin (1916). Piana (1897) first reported the intermediate hosts, terrestrial isopods (sow bugs or pillbugs), and the life cycle of *D. nasuta* has been further studied throughout the twentieth century (Cram 1931; Birova et al. 1974a, b; Nagy et al. 1977). The pathogenicity of *D. nasuta* (often recorded as *Dispharynx spiralis*) has been recognized at least since the early twentieth century. Early descriptions of dispharynxiasis were reported in wild game birds such

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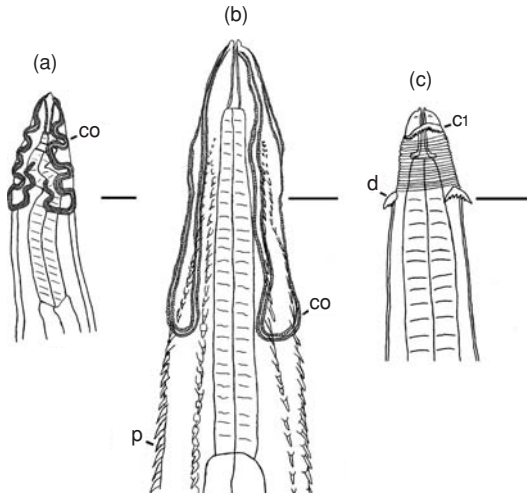


Figure 18.1. (a) *Dispharynx nasuta*, male, anterior, from a Rock Pigeon (*Columba livia*). Note the recurring cordons (co) and absence of cuticular spines. Scale bar = 50 μm. (b) *Echinuria uncinata*, male, anterior, from a Mute Swan (*Cygnus olor*). Note the cordons (co) and cuticular spines (sp). Scale bar = 100 μm. (c) *Streptocara crassicauda*, female, anterior from a King Eider (*Somateria spectabilis*). Note the collarette (cl), prominent deirids (d), and somewhat inflated cuticle between the collarette and the deirids. Scale bar = 25 μm.

as grouse, and in recent years, outbreaks of the disease have also occurred in private collections, outdoor poultry farms, and in zoos.

ETIOLOGY

Taxonomic Synonyms: Dispharynx nasuta (Rudolphi 1819) Stiles and Hassall, 1920; *Spiroptera nasuta* Rudolphi, 1819; *Dispharagus spiralis* Molin, 1858; *Acuaria spiralis* (Molin 1858) Railliet, Henry, and Sisoff, 1912; *Dispharagus nasutus* (Rud. 1819) Dujardin, 1844; *Filaria nasuta* (Rud. 1819) Schneider, 1866; *Dispharagus tentaculatus* Colucci, 1893; *Dispharagus spiralis columbae* Bridré, 1910; *Acuaria nasuta* (Rud. 1819) Railliet, Henry, and Sisoff, 1912; *Cheilospirura nasuta* (Rud. 1819) Ransom, 1916; *Dispharynx stonae* Harwood, 1933.

The species of *Dispharynx* comprise a genus of acuarioid nematodes that includes *D. nasuta*, the spiral stomach worm. About 15 species of *Dispharynx* have been described, and *D. nasuta* is considered to be the most pathogenic of these.

Adults of *D. nasuta* have prominent cordons that, at their distal ends, curve back toward the anterior of the worm in a recurrent pattern (Figure 18.1a). The posterior ends of the males are often spirally coiled. Males are about 5 mm long and females are 5–9 mm in length. The phylogenetic relationships of the Acuarioidea have not been assessed using both morphological and molecular data. Consequently, the sister groups of *Dispharynx* species and their precise phylogenetic position within the spirurids are unknown.

EPIZOOTIOLOGY

The life cycle of *D. nasuta* requires development of larvae in intermediate hosts, usually in various terrestrial isopods commonly known as "pillbugs." *Porcellio scaber, Armadillidium vulgare*, and *Oniscus* species (Anderson 2000) as well as *Venezillo evergladensis* and *Oscelloscia floridana* (Rickard 1985) are suitable intermediate hosts. Eggs of *D. nasuta* from Ruffed Grouse (*Bonasa umbellus*) undergo development to third-stage larvae in 26 days when fed to *P. scaber* and *A. vulgare* (Cram 1931) and 16 days when fed to *Porcellionides pruinosus* from Cuba (Birova et al. 1974a). Temperature is a likely factor that influences development of the larvae. Infected isopods behave differently from uninfected isopods and are observed at greater frequencies on light-colored surfaces (Moore and Lasswell 1986). This altered behavior may make infected isopods more susceptible to predation by definitive avian hosts.

Following ingestion of an infected isopod, full development to adults occurs in 27 days in Ruffed Grouse, Northern Bobwhite (*Colinus virginianus*), and Rock Pigeons (*Columba livia*) according to Cram (1931), and 21 days in domestic chickens (*Gallus gallus*) (Birova et al. 1974b). Ingested infective larvae spend a short period of time (up to 15 days) in the lumen of the proventriculus before penetrating into the mucosa (Nagy et al. 1977). It is at the latter stage when pathogenic effects are induced. In experimental infections of domestic turkeys with eggs of *D. nasuta* originating from Boat-tailed Grackles (*Quiscalus major*), Blue Jays (*Cyanocitta cristata*), Northern Cardinals (*Cardinalis cardinalis*), American Crows (*Corvus brachyrhynchos*), and Wild Turkeys (*Meleagris gallopavo*), eggs were not detected in the feces of infected turkeys until 42 days postinfection (PI). An experimentally infected grackle became patent at 32 days PI (Rickard 1985).

CLINICAL SIGNS

Young birds with high intensities of *D. nasuta* exhibit droopiness, inactivity, and weight loss (Cram 1928; Hwang et al. 1961; Nagy et al. 1977). They undergo

Table 18.1. Host distribution of *Dispharynx nasuta* (DN), *Echinuria uncinata* (EU), *Streptocara crassicauda* (SC), and *Streptocara incognita* (SI).

Host order	Host species	DN	EU	SC	SI	Reference
Gaviiformes	Arctic Loon (*Gavia arctica*)	—	—	+	—	McDonald (1969)
	Red-throated Loon (*Gavia stellata*)	—	—	+	—	McDonald (1969)
Podicipediformes	Western Grebe (*Aechmophorus occidentalis*)	—	—	+	—	Denny (1969)
	Horned Grebe (*Podiceps auritus*)	—	—	+	—	Denny (1969)
	Great Crested Grebe (*Podiceps cristatus*)	—	—	+	—	McDonald (1969)
	Red-necked Grebe (*Podiceps grisegena*)	—	—	+	—	Denny (1969)
Pelecaniformes	Great Cormorant (*Phalacrocorax carbo*)	—	—	+	—	McDonald (1969)
Ciconiiformes	Great Bittern (*Botaurus stellaris*)	—	—	+	—	McDonald (1969)
Phoenicopteriformes	Chilean Flamingo (*Phoenicopterus chilensis*)	—	—	—	+	Fox et al. (1974)
Anseriformes	Fulvous Whistling-Duck (*Dendrocygna bicolor*)	—	+	—	—	Wood (1974)
	White-faced Whistling-Duck (*Dendrocygna viduata*)	—	+	—	—	McDonald (1969)
	West Indian Whistling-Duck (*Dendrocygna arborea*)	—	+	—	—	Wood (1974)
	Mute Swan (*Cygnus olor*)	—	+	—	—	McDonald (1969)
	Tundra Swan (*Cygnus columbianus*)	—	—	+	—	McDonald (1969)
	Whooper Swan (*Cygnus cygnus*)	—	+	+	—	McDonald (1969)
	Black-necked Swan (*Cygnus melancoryphus*)	—	+	—	—	McDonald (1969)
	Black Swan (*Cygnus atratus*)	—	+	—	—	McDonald (1969)
	Trumpeter Swan (*Cygnus buccinator*)	—	+	+	—	McDonald (1969)
	Greater White-fronted Goose (*Anser albifrons*)	—	+	—	—	McDonald (1969)
	Swan Goose (*Anser cygnoides*)	—	+	+	+	McDonald (1969)
	Emperor Goose (*Chen canagica*)	—	+	—	—	Wood (1974)
	Canada Goose (*Branta canadensis*)	—	+	—	—	McDonald (1969)
	Barnacle Goose (*Branta leucopsis*)	—	+	—	—	McDonald (1969)
	Brant (*Branta bernicla*)	—	+	—	—	Wood (1974)
	Red-breasted Goose (*Branta ruficollis*)	—	+	—	—	McDonald (1969)
	Hawaiian Goose (*Branta sandvicensis*)	—	+	+	—	Bailey and Black (1995) and Lapage (1961)

Orinoco Goose (*Neochen jubata*)	—	+	—	—	McDonald (1969)
Egyptian Goose (*Alopochen aegyptiaca*)	—	+	—	—	Wood (1974)
Ruddy Shelduck (*Tadorna ferruginea*)	—	+	+	—	McDonald (1969)
Muscovy Duck (*Cairina moschata*)	—	+	+	—	McDonald (1969) and Mason (1988)
White-winged Duck (*Cairina scutulata*)	—	+	—	—	Wood (1974)
Comb Duck (*Sarkidiornis melanotos*)	—	+	—	—	Wood (1974)
Hartlaub's Duck (*Pteronetta hartlaubii*)	—	+	—	—	McDonald (1969)
Cotton Pygmy-Goose (*Nettapus coromandelianus*)	—	+	—	—	Wood (1974)
African Pygmy-Goose (*Nettapus auritus*)	—	+	—	—	Wood (1974)
Ringed Teal (*Callonetta leucophrys*)	—	+	—	—	Wood (1974)
Mandarin Duck (*Aix galericulata*)	—	+	—	—	McDonald (1969)
Wood Duck (*Aix sponsa*)	—	+	—	—	McDonald (1969)
Blue Duck (*Hymenolaimus malacorhynchos*)	—	+	—	—	Wood (1974)
Northern Pintail (*Anas acuta*)	—	+	+	—	McDonald (1969)
White-cheeked Pintail (*Anas bahamensis*)	—	+	—	—	Lapage (1961)
American Wigeon (*Anas americana*)	—	+	+	—	McDonald (1969) and McLaughlin and McGurk (1987)
Eurasian Wigeon (*Anas penelope*)	—	+	+	—	McDonald (1969)
Chiloe Wigeon (*Anas sibilatrix*)	—	+	—	—	McDonald (1969)
Cape Teal (*Anas capensis*)	—	+	—	—	McDonald (1969)
Chestnut Teal (*Anas castanea*)	—	+	—	—	McDonald (1969)
Cinnamon Teal (*Anas cyanoptera*)	—	+	+	—	Lapage (1961) and McDonald (1969)
Northern Shoveler (*Anas clypeata*)	—	+	+	—	McDonald (1969)
Blue-winged Teal (*Anas discors*)	—	+	+	—	McDonald (1969) and McLaughlin and McGurk (1987)
Baikal Teal (*Anas formosa*)	—	—	+	—	McDonald (1969)
Falcated Duck (*Anas falcata*)	—	+	—	—	McDonald (1969)
Yellow-billed Pintail (*Anas georgica*)	—	+	—	—	McDonald (1969)
Mallard (*Anas platyrhynchos*)	—	+	+	+	McDonald (1969)
Silver Teal (*Anas versicolor*)	—	+	—	—	Wood (1974)

(*continues*)

Table 18.1. (*Continued*)

Host order	Host species	DN	EU	SC	SI	Reference
	Garganey (*Anas querquedula*)	—	+	+	—	McDonald (1969)
	American Black Duck (*Anas rubripes*)	—	+	—	—	McDonald (1969)
	African Black Duck (*Anas sparsa*)	—	+	—	—	Lapage (1961)
	Pacific Black Duck (*Anas superciliosa*)	—	+	—	—	Clark (1977)
	Philippine Duck (*Anas luzonica*)	—	+	—	—	Wood (1974)
	Gadwall (*Anas strepera*)	—	+	+	—	McDonald (1969)
	Spectacled Duck (*Anas specularis*)	—	+	—	—	McDonald (1969)
	Yellow-billed Duck (*Anas undulata*)	—	+	—	—	McDonald (1969)
	Red-crested Pochard (*Netta rufina*)	—	+	+	—	McDonald (1969)
	Rosy-Billed Pochard (*Netta peposaca*)	—	+	—	—	da Silveira et al. (2006)
	Lesser Scaup (*Aythya affinis*)	—	+	+	—	McDonald (1969)
	White-eyed Duck (*Aythya australis*)	—	+	—	—	Wood (1974)
	Greater Scaup (*Aythya marila*)	—	+	+	—	McDonald (1969)
	Redhead (*Aythya americana*)	—	+	+	—	McDonald (1969) and McLaughlin and McGurk (1987)
	Ferruginous Pochard (*Aythya nyroca*)	—	—	+	—	McDonald (1969)
	Tufted Duck (*Aythya fuligula*)	—	+	+	—	McDonald (1969) and Wood (1974)
	Canvas back (*Aythya valisineria*)	—	+	+	—	McDonald (1969)
	Common Eider (*Somateria mollissima*)	—	+	+	—	McDonald (1969)
	King Eider (*Somateria spectabilis*)	—	+	+	—	McDonald (1969); unpublished observations
	Harlequin Duck (*Histrionicus histrionicus*)	—	+	+	—	Wood (1974) and McDonald (1969)
	Long-tailed Duck (*Clangula hyemalis*)	—	—	+	—	McDonald (1969)
	White-winged Scoter (*Melanitta fusca*)	—	—	+	—	McDonald (1969)
	Black Scoter (*Melanitta nigra*)	—	—	+	—	McDonald (1969)
	Surf Scoter (*Melanitta perspicillata*)	—	—	+	—	McDonald (1969)
	Bufflehead (*Bucephala albeola*)	—	+	+	—	McDonald (1969) and McLaughlin and McGurk (1987)
	Barrow's Goldeneye (*Bucephala islandica*)	—	—	+	+	McDonald (1969)
	Smew (*Mergellus albellus*)	—	+	+	—	McDonald (1969)
	Hooded Merganser (*Lophodytes cucullatus*)	—	—	+	—	McDonald (1969)

	Common Merganser (*Mergus merganser*)	—	+	+	—	Wood (1974) and McDonald (1969)
	Red-breasted Merganser (*Mergus serrator*)	—	—	+	—	McDonald (1969)
	Ruddy Duck (*Oxyura jamaicensis*)	—	+	+	+	McLaughlin and McGurk (1987) and McDonald (1969)
Galliformes	Plain Chachalaca (*Ortalis vetula*)	+	—	—	—	Christensen and Pence (1977)
	Wild Turkey (*Meleagris gallopavo*)	+	—	—	—	Goble and Kutz (1945)
	Dusky Grouse (*Dendragapus obscurus*)	+	—	—	—	Bendell (1955)
	Ruffed Grouse (*Bonasa umbellus*)	+	—	—	—	Goble and Kutz (1945)
	Sharp-tailed Grouse (*Tympanuchus phasianellus*)	—	—	+	—	McDonald (1969)
	California Quail (*Callipepla californica*)	+	—	—	—	Moore et al. (1988)
	Northern Bobwhite (*Colinus virginianus*)	+	—	—	—	Goble and Kutz (1945)
	Barbary Partridge (*Alectoris barbara*)	+	—	—	—	Goble and Kutz (1945)
	Gray Partridge (*Perdix perdix*)	+	—	—	—	Goble and Kutz (1945)
	Common Quail (*Coturnix coturnix*)	+	—	—	—	Baruš and Sonin (1983)
	Red Junglefowl (*Gallus gallus*)	+	—	+	—	Goble and Kutz (1945) and McDonald (1969)
	Ring-necked Pheasant (*Phasianus colchicus*)	+	—	—	—	Goble and Kutz (1945)
	Golden Pheasant (*Chrysolophus pictus*)	+	—	—	—	Goble and Kutz (1945)
	Indian Peafowl (*Pavo cristatus*)	+	—	—	—	Goble and Kutz (1945)
	Helmeted guineafowl (*Numida meleagris*)	+	—	+	—	Goble and Kutz (1945) and McDonald (1969)
Gruiformes	Whooping Crane (*Grus americana*)	+	—	—	—	Spalding et al. (1996)
	Sandhill Crane (*Grus canadensis*)	+	—	—	—	Forrester et al. (1975)
	Eurasian Coot (*Fulica atra*)	—	—	+	—	McDonald (1969)
Charadriiformes	African Jacana (*Actophilornis africanus*)	+	—	—	—	Schulman et al. (1992)
	Eurasian Oystercatcher (*Haematopus ostralegus*)	—	—	+	—	Borgsteede et al. (1988)
	Northern Lapwing (*Vanellus vanellus*)	—	—	+	—	McDonald (1969)
	Snowy Plover (*Charadrius alexandrinus*)	—	—	+	—	McDonald (1969)
	Common Greenshank (*Tringa nebularia*)	—	—	+	—	McDonald (1969)

(*continues*)

Table 18.1. (*Continued*)

Host order	Host species	DN	EU	SC	SI	Reference
	Common Redshank (*Tringa totanus*)	—	—	+	—	McDonald (1969)
	Spotted Sandpiper (*Actitis macularius*)	—	—	+	—	McDonald (1969)
	Ruff (*Philomachus pugnax*)	—	+	+	—	McDonald (1969)
	European Herring Gull (*Larus argentatus*)	—	—	+	—	McDonald (1969)
	Mew Gull (*Larus canus*)	—	—	+	—	McDonald (1969)
	Great Black-headed Gull (*Larus ichthyaetus*)	—	—	+	—	McDonald (1969)
	Thayer's Gull (*Larus thayeri*)	—	—	+	—	Gibson (1968)
	Common Tern (*Sterna hirundo*)	—	—	+	—	McDonald (1969)
	Common Murre (*Uria aalge*)	—	—	+	—	McDonald (1969)
	Razorbill (*Alca torda*)	—	—	+	—	McDonald (1969)
	Pigeon Guillemot (*Cepphus columba*)	—	—	+	—	McDonald (1969)
	Horned Puffin (*Fratercula corniculata*)	—	—	+	—	McDonald (1969)
Columbiformes	Rock Pigeon (*Columba livia*)	+	—	—	—	Goble and Kutz (1945)
	Mourning Dove (*Zenaida macroura*)	+	—	—	—	Forrester et al. (1983)
	Luzon Bleeding-heart (*Gallicolumba luzonica*)	+	—	—	—	Lindquist and Strafuss (1980)
Psittaciformes	Alexandra's Parrot (*Polytelis alexandrae*)	+	—	—	—	Bolette (1998)b
Cuculiformes	Smooth-billed Ani (*Crotophaga ani*)	+	—	—	—	Macko et al. (1974)
Coraciiformes	European Roller (*Coracias garrulus*)	+	—	—	—	Goble and Kutz (1945)
Piciformes	Red-headed Woodpecker (*Melanerpes erythrocephalus*)	+	—	—	—	Cooper (1974)
	Yellow-bellied Sapsucker (*Sphyrapicus varius*)	+	—	—	—	Baruš (1971)
	Northern Flicker (*Colaptes auratus*)	+	—	—	—	Bolette (1998)c
Passeriformes	Least Flycatcher (*Empidonax minimus*)	+	—	—	—	Bolette (1998)a
	Carolina Wren (*Thryothorus ludovicianus*)	+	—	—	—	Goble and Kutz (1945)
	Long-billed Gnatwren (*Ramphocaenus melanurus*)	+	—	—	—	Zhang et al. (2004)
	Gray Catbird (*Dumetella carolinensis*)	+	—	—	—	Goble and Kutz (1945)

Northern Mockingbird (*Mimus polyglottos*)	+	—	—	—	Macko et al. (1974)
Eastern Bluebird (*Sialia sialis*)	+	—	—	—	Goble and Kutz (1945)
American Robin (*Turdus migratorius*)	+	—	—	—	Goble and Kutz (1945)
Clay-colored Robin (*Turdus grayi*)	+	—	—	—	Zhang et al. (2004)
Blue Jay (*Cyanocitta cristata*)	+	—	—	—	Rickard (1985)
Florida Scrub-Jay (*Aphelocoma coerulescens*)	+	—	—	—	Kinsella (1974)
American Crow (*Corvus brachyrhynchos*)	+	—	—	—	Goble and Kutz (1945)
Cuban Crow (*Corvus nasicus*)	+	—	—	—	Baruš and Garrido (1968)
European Starling (*Sturnus vulgaris*)	+	—	—	—	Goble and Kutz (1945)
House Sparrow (*Passer domesticus*)	+	—	—	—	Goble and Kutz (1945)
Prairie Warbler (*Dendroica discolor*)	+	—	—	—	Baruš and Garrido (1968)
Blackburnian Warbler (*Dendroica fusca*)	+	—	—	—	Barus and Garrido (1968)
Tennessee Warbler (*Vermivora peregrina*)	+	—	—	—	Zhang et al. (2004)
Gray-crowned Yellowthroat (*Geothlypis poliocephala*)	+	—	—	—	Zhang et al. (2004)
Northern Cardinal (*Cardinalis cardinalis*)	+	—	—	—	Rickard (1985)
Blue-gray Tanager (*Thraupis episcopus*)	+	—	—	—	Zhang et al. (2004)
Rose-breasted Grosbeak (*Pheucticus ludovicianus*)	+	—	—	—	Baruš and Garrido (1968)
Black-faced Grosbeak (*Caryothraustes poliogaster*)	+	—	—	—	Zhang et al. (2004)
Boat-tailed Grackle (*Quiscalus major*)	+	—	—	—	Rickard (1985)
Greater Antillean Grackle (*Quiscalus niger*)	+	—	—	—	Baruš and Garrido (1968)
Brown-headed Cowbird (*Molothrus ater*)	+	—	—	—	Goble and Kutz (1945)

Geographic range may include domestic or captive host populations. Host names follow Clements (2000).

retarded development, yet retain a ravenous appetite. When present in large numbers, *D. nasuta* may inhibit growth and cause anorexia and inability to thrive. Emaciation and death may follow (Cram 1928; Shanthikumar 1987).

PATHOGENESIS AND PATHOLOGY

Development of both juvenile and adult specimens of *D. nasuta* in the proventriculus causes dispharynxiasis. Lesions are produced when the worms bury their heads into the lamina propria of the proventriculus, causing an inflammatory response that leads to thickening of the mucosa and functional obstruction of the digestive tract (Schulman et al. 1992). This inflammatory response can result in death by starvation due to decreased digestive capability (Blasdel and Lasswell 1986).

At necropsy, characteristic lesions consist of ulceration and inflammation of the proventriculus, which may be as large as the gizzard (Shanthikumar 1987). The mucosa is often completely destroyed, and parasites are found buried in a mass of degenerated and necrotic tissue (Shanthikumar 1987).

The presence of *D. nasuta* can cause swelling, ulceration, cellular infiltration, and destruction of the proventricular glands (Figure 18.2).

As much as a threefold increase in the size of the proventriculus has been reported from infected Boat-tailed Grackles and other birds (Rickard 1985). In the vicinity of the worms, multifocal petechial hemorrhages are found, the mucosa of the proventriculus is thickened, and the worms are covered in an acellular, catarrhal exudate (Hwang et al. 1961; Rickard 1985; Schulman et al. 1992). The pathogenesis of *D. nasuta* in domestic chickens occurs in three phases that coincide with development of the worms (Nagy et al. 1977). In the first phase, 1–11 days PI, petechial hemorrhages and light hyperplastic processes are evident. In the second, 12–22 days PI, the proventriculus begins to increase in size, and congestion and hemorrhage become more marked. In the third phase at 23 days PI when mature females begin to shed eggs, the serosal surface of the proventriculus is covered with whitish areas from which a yellowish exudate emanates. These whitish regions have been described as necrotic ulcers and contain developing juveniles (Ramaswamy and Sundaram 1985).

In addition to the pathogenic effects described above, an extreme papillomatous proliferation of the mucosal surface has been seen in a naturally infected American Crow, a naturally infected Boat-tailed Grackle, and two naturally infected Northern Cardinals. In one case, proliferative tissue almost completely occluded the proventricular lumen (Rickard 1985).



Figure 18.2. *Dispharynx nasuta*. Proventriculus of a Rock Pigeon (*Columba livia*) with parasites in situ (arrows). Reproduced with permission from *Avian Diseases* (Hwang et al. 1961).

In histological sections, extensive desquamation of the superficial mucosa, hypersecretion of mucus, and cellular masses containing bacteria can be observed. At 8 days PI in domestic chickens, a severe nonkeratinizing squamous cell metaplasia of the lining epithelium occurs in the lamina propria, and proventricular glands containing juvenile worms are surrounded by necrotic cell debris and desquamated epithelial cells (Ramaswamy and Sundaram 1985). By 14 days PI, deep ulcers, necrotic foci, lymphoid hyperplasia, and severe cellular infiltration with granulocytes occur in the lamina propria. Many of these histological changes, including the occurrence of a mononuclear transmural infiltrate through the proventriculus, were also evident in experimentally infected domestic turkeys and a Brown-headed Cowbird (*Molothrus ater*) (Rickard 1985).

DIAGNOSIS

Both demonstration of the eggs of *D. nasuta* in feces and the presence of clinical signs can be used to make

an antemortem diagnosis. The eggs are ellipsoidal, 36–40 μm long and 21 μm wide, and are embryonated when passed in feces. These features distinguish eggs of *D. nasuta* from those of many other parasitic nematodes such as capillarids and ascarids. However, the possibility of infection with other spirurid nematodes cannot be excluded based on this type of egg morphology. Necropsy is necessary to recover adults from the proventriculus and to verify the presence of recurrent cordons.

Pathogenic effects of the worms can occur before the infections become patent and eggs may be passed sporadically. A possible method for antemortem diagnosis involves contrast radiography, whereby distinct radiographic changes of increased proventricular to ventricular size ratio and proventricular filling can be detected (Schulman et al. 1992). Postmortem observation of the characteristic proventriculitis and the presence of adults and juveniles of *D. nasuta* (Figure 18.2) can confirm diagnosis.

IMMUNITY

The immune response to *D. nasuta* has not been studied in detail. Experimental infections in domestic and wild birds have shown that some hosts appear to be more susceptible to dispharynxiasis than do others. This might suggest the presence of different strains of *D. nasuta*, some of which are more pathogenic (Rickard 1985), or may be reflective of a longer host–parasite coevolution of *D. nasuta* in some hosts.

Although survey data and information from published case reports are limited, infections appear to be more common in younger birds than in older ones. A list of infected galliform and passeriform birds by Goble and Kutz (1945) reported consistently higher prevalences of infection in juvenile birds than adults. Mechanisms of age resistance to infection, if present, are unknown.

ECHINURIA

SYNONYMS

Acuariasis, echinuriasis.

HISTORY

The genus *Echinuria* (family Acuariidae) was erected in 1912 by Soloviev, and *E. uncinata* (Rudolphi 1819) Soloviev, 1912 was eventually placed here (see list of synonyms below). The intermediate hosts of *E. uncinata* were discovered by Hamann (1893), who demonstrated development of the larvae in cladoceran crustaceans of the genus *Daphnia*. Descriptions of echinuriasis were given in the late nineteenth century (Hamann, 1893), and the disease has been documented frequently throughout the later half of the twentieth century (Buxton et al. 1952; Cornwell 1963; Gräfner and Graubmann 1967; Takla and Thiel 1983; Griffiths et al. 1985). In the latter period, studies of the disease using experimental infections with *E. uncinata* have provided additional information (Guilhon et al. 1971; Ould and Welch 1980).

ETIOLOGY

Taxonomic synonyms: Echinuria uncinata (Rudolphi 1819) Soloviev, 1912; *Spiroptera uncinata* Rudolphi, 1819; *Filaria uncinata* Schneider, 1866; *Dispharagus uncinatus* (Rud. 1819) Railliet, 1893; *Acuaria (Hamannia) uncinata* (Rud. 1819) Stiles and Hassall, 1912; *Echinuria jugadornata* Soloviev, 1912.

Approximately 14–15 species of *Echinuria* are recognized (Ali, 1968; McDonald, 1974). Members of the genus occur in the proventriculus of avian hosts. Although various *Echinuria* spp. have been reported from other birds (mostly waterfowl), *E. uncinata* from anatids is the most significant pathogen of the genus. This species, like other members of the genus, has recurving cordons extending posteriorly from the anterior end and, unlike *Dispharynx* species, has cuticular spines running along the length of the body (Figure 18.1b). Adult males are 8–10 mm in length and females are 12–18 mm long.

EPIZOOTIOLOGY

The ingestion of intermediate hosts containing infective larvae of *E. uncinata* results in the development of the parasites in the proventriculus. Cladoceran crustaceans, usually of the genus *Daphnia* (e.g., *D. pulex* and *D. magna*), are suitable intermediate hosts for *E. uncinata* (Anderson 2000). In addition to species of *Daphnia*, *Ceriodaphnia* spp., *Simocephalus vetulus*, *Gammarus lacustris*, the ostracod *Heterocypris incongruens*, and the conchostracan *Lynceus brachyurus* have also been shown to support development of third-stage larvae (Misiura 1970; Austin and Welch 1972; Anderson 2000). On the basis of studies of the life cycle in domestic ducks, embryonated eggs are ingested by intermediate hosts and the larvae penetrate through the digestive tract and undergo development in the body cavity of the cladoceran intermediate hosts (Guilhon et al. 1971). The larvae molt once approximately 8–9 days after ingestion and molt again to the third stage after 14–15 days, depending on the temperature. In temperate climates, numbers of larvae in intermediate hosts reach their highest numbers in midsummer (Austin and Welch 1972). Upon ingestion of infected species of *Daphnia* by the avian

definitive hosts, the third-stage larvae are liberated and disseminate throughout the proventriculus. Fully formed adults are noticeable after 45 days, and the prepatent period is approximately 30 days.

In Mallards (*Anas platyrhynchos*), parasites are located under the soft mucosa of the proventriculus and gizzard, usually along the region connecting the two organs. Groups of worms occur in fibrotic nodules and large granulomas form after 40–50 days. Sexually mature male worms are found 30 days PI, and mature females shed viable eggs 40 days PI (Austin and Welch 1972).

Some species of waterfowl are apparently more susceptible to infection with *E. uncinata* than others. On the basis of experimental infections of ducklings with infective larvae, Northern Shovelers (*Anas clypeata*), which feed frequently on *Daphnia* species, have very low susceptibility to infection (Austin and Welch 1972). Mallards, Gadwalls (*Anas strepera*), Common Eiders (*Somateria mollissima*), Northern Pintails (*Anas acuta*), and domestic geese are the most susceptible to infection with *E. uncinata*, while Northern Shovelers (*Anas clypeata*), Blue-winged Teal (*Anas discors*), Ruddy Ducks (*Oxyura jamaicensis*), and American Coots (*Fulica americana*) are generally resistant (Austin and Welch 1972). Experimental infections in *Anas* spp. generally yield higher average numbers of worms than in other experimentally infected waterfowl (Austin and Welch 1972).

CLINICAL SIGNS

Birds with infections of high intensity are usually in a weak and emaciated condition without apparent inappetance (Kock et al. 1987). Wild ducks have a prominent keel as a result of severe emaciation, fading, discoloring, and poor grooming of the feathers, an inability to sustain flight, or to fly at all, and a near absence of wariness (Cornwell 1963). Gräfner and Graubmann (1967) described the clinical signs of infection as weakness, awkward gait, and diminished appetite. Throughout the progression of the disease, the animals reeled, crossed their legs, and eventually developed lameness and difficulty in breathing. Decreased intake of food occurred. Attempts to eat resulted in retching or choking, and the birds became completely emaciated and soon died.

PATHOGENESIS AND PATHOLOGY

The presence of the nematodes in the proventriculus results in a strong immune response that leads to formation of nodules that contain the worms. In heavy infections, these nodules can become so numerous that the proventriculus becomes occluded, preventing successful feeding. Infection with *E. uncinata* can result in the formation of encapsulated lesions in the proventriculus (Figure 18.3), usually at its junction with the esophagus (Kock et al. 1987). The overall size of the proventriculus increases, and the proventricular mucosae can be covered by thick, white, slimy exudates (Griffiths et al. 1985). The nodules often contain creamy white caseous material, and worms are often not present. The nodules eventually become granulomas. In fatal infections, the nodules become so large that the proventriculus becomes occluded, preventing the passage of food. In histological sections, abscess-like lesions in the wall of the proventriculus are evident. These lesions are lined by cells resembling macrophages and giant cells and surrounded by extensive proliferation

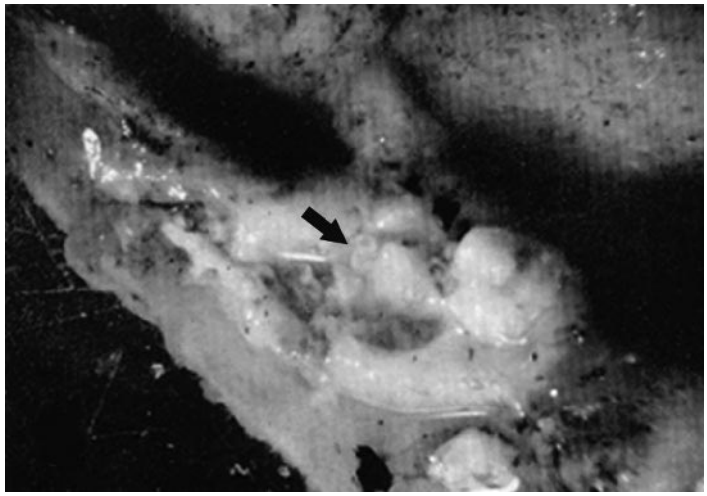


Figure 18.3. *Echinuria uncinata*. Ulcerative cyst in the proventriculus of a Trumpeter Swan (*Cygnus buccinator*). There is a caseous tissue reaction and some nematodes (arrow) are visible in situ. Reproduced with permission from *Canadian Journal of Zoology* (Cornwell 1963).

of fibrous tissue and inflammatory cells (Kock et al. 1987). There is some evidence of ulceration in the lining epithelium at the junction of the proventriculus and esophagus (Kock et al. 1987).

DIAGNOSIS

Methods for diagnosis of echinuriasis in live birds include the recovery of eggs from fecal samples. Eggs shed in the feces of infected birds are approximately 35 μm × 20 μm (Guilhon et al. 1971); however, eggs will not be present if the prepatent period has not been completed (Respaldiza et al. 1979). At postmortem, the characteristic enlarged proventriculus, proventricular lesions (0.5–2.5 cm nodules and granulomas), and presence of whole specimens of *E. uncinata* inside the nodules can confirm diagnosis.

Species of *Echinuria* differ from most other acuarioid nematodes in having cuticular spines that extend posteriorad from the anterior end (Figure 18.1b). Other nematodes that may occur in the proventriculus include other acuarioid nematodes such as *Streptocara* spp. as well as *Tetrameres* spp. and the trichostrongyloid *Amidostomum*. Adult morphology, including extreme sexual dimorphism, absence of cordons, and different lesions can be used to differentiate *Tetrameres* infections from those of *E. uncinata*. Species of *Streptocara* and *Amidostomum* differ morphologically from *Echinuria* and are more often found in the gizzard.

STREPTOCARA

SYNONYMS

Streptocariasis.

HISTORY

Species of *Streptocara* are acuarioid nematodes in the family Seuratiinae that occur in the gizzard and occasionally the proventriculus and esophagus of waterfowl. Records of various species of *Streptocara* in waterfowl have been known since the nineteenth century. The life cycle remained unknown, however, until Garkavi (1949) established that amphipods (*G. lacustris*) are suitable intermediate hosts. There have been scattered reports of outbreaks of streptocariasis in waterfowl throughout the twentieth century, but some of the most detailed studies of the disease were carried out in the 1980s and early 1990s (Sterner and Stackhouse 1987; Mason 1988; Laberge and McLaughlin 1991).

ETIOLOGY

Taxonomic synonyms: Streptocara crassicauda (Creplin 1829) Skrjabin, 1916; *Spiroptera crassicauda* Creplin, 1829; *Dispharagus crassicauda* (Creplin 1829) Molin, 1860; *Streptocara crassicauda crassicauda* (Creplin 1829) Gibson, 1968; *Spiroptera pectinifera* Neumann, 1900; *Streptocara pectinifera* (Neumann 1900) Skrjabin, 1916; *Streptocara crassicauda anseri* Skrjabin, 1916; *Streptocara crassicauda charadrii* Skrjabin, 1916; *Streptocara crassicauda skrjabini* Liubimov, 1927.

Adult *Streptocara* spp. have cordons that expand only on the cephalic region, forming a collarette (Figure 18.1c) (see also Chabaud, 1975). The deirids (cervical papillae) are located posterior to the cephalic region and have 5–9 teeth. Although up to 12 species have been listed in various monographs, only 4 species and 2 subspecies were included by Gibson (1968) in a review of the genus. Additional species have been described from various avian hosts since 1968.

One of the common species that has been shown to be pathogenic is *S. crassicauda*. Males of these thin, rather delicate parasites are 3.4–5.1 mm long and females are 5.5–12.9 mm in length (Gibson 1968). The cuticle has an annular pattern and is slightly expanded at the cephalic end (Figure 18.1c). Another species of *Streptocara*, *S. incognita* Gibson, 1968, has also been shown to be pathogenic in waterfowl. This species lacks the inflated cuticle and annuli between the collarette and the cervical papillae are also different from those of *S. crassicauda* (Gibson 1968; McDonald 1974).

EPIZOOTIOLOGY

Definitive hosts become infected by ingesting amphipod intermediate hosts such as species of *Gammarus* and *Hyalella* (Anderson 2000). Eggs of *S. crassicauda* are shed in the feces and develop in various marine and freshwater amphipod crustaceans. Development from eggs to infective third-stage larvae occurs in 19–25 days in *G. lacustris*, depending on the temperature (Garkavi 1949). Larvae from six species of fish were also infective to ducks (Kovalenko 1960). The amphipod, *Hyalella azteca*, also serves as a suitable intermediate host (Denny 1969; Laberge and McLaughlin 1989). First-stage larvae molt as early as 11 days PI at 18–20°C in these amphipods, with most molting after 15–17 days. Second-stage larvae molt as early as 15 days PI, with a peak on day 17 PI (Laberge and McLaughlin 1989). The earliest third-stage larvae are found 19 days PI. When infective larvae from *H. azteca* were fed to domestic ducks, female worms became gravid as early as 9 days PI. The first eggs detected in feces were observed 26 days PI (Laberge and McLaughlin 1989). The life cycle of *S. incognita* has not been determined, but is likely similar to that of *S. crassicauda*.

In comparing infections in Blue-winged Teal, Gadwall, and Lesser Scaup (*Aythya affinis*), Laberge and McLaughlin (1991) suggested that dabbling ducks such as Blue-winged Teal and Gadwall are suitable hosts for *S. crassicauda* but play a minor role in maintaining transmission of the parasite. Experimentally infected ducklings have short-lived infections and lesions in Blue-winged Teal and Gadwall are larger than those in diving ducks, such as Lesser Scaup, that feed primarily on amphipods (Laberge and McLaughlin 1991). This suggests that dabbling ducks are more likely to become infected where they overlap in distribution with Lesser Scaup (McLaughlin and McGurk 1987; Laberge and McLaughlin 1991). The smaller lesions in Lesser Scaup may be attributed to a greater degree of tolerance to infection with *Streptocara* spp.

Since fish can serve as paratenic hosts of *S. crassicauda* (Kovalenko 1960), other waterfowl, including fish-eating ducks, may have evolved a similar tolerance for the parasites. On the basis of the large number of fish-eating definitive hosts for *S. crassicauda* (Table 18.1), piscine paratenic hosts may play a significant role in transmitting infective larvae to these avian hosts. However, the role of paratenic hosts in the natural transmission of *Streptocara* to ducks and other birds is poorly understood.

CLINICAL SIGNS

Birds infected with *Streptocara* spp. may exhibit weakness, loss of appetite, and sneezing (Mason 1988). Weight loss and an inability to swallow have been observed as additional clinical signs (Dalton 1980).

PATHOGENESIS AND PATHOLOGY

Infective larvae of *Streptocara* spp. penetrate the cuticle of the gizzard and burrow into the mucosa. Their presence gives rise to local hemorrhage, ulceration, and necrosis (Boughton 1969). The burrowing of the worms into the mucosa results in local hemorrhage and later ulceration and necrosis of the tissues. Sloughing of the cornified layer of the gizzard can occur, leaving large areas unsuitable for grinding food (Sterner and Stackhouse 1987). Infections with *Streptocara* can cause a necrotizing esophagitis, an inability to swallow, and death from inanition (Dalton 1980). The presence of *S. crassicauda* in the gizzard and *S. incognita* in the gizzard, proventriculus, and esophagus can thus lead to destruction of tissue, inability of the host to feed properly, and death.

One- to two-cm-diameter ulcers have been reported at the junction of the gizzard and proventriculus and ulcerative lesions were also found on the proventriculus and gizzard of Chilean Flamingos (*Phoenicopterus chilensis*) infected with *S. incognita* (Fox et al. 1974). The primary lesion associated with infection with *Streptocara* spp. among a variety of species of ducks in Australia was a necrotic, diphtheritic, plaque-like mass overlying, and adherent to the larynx and pharynx and adjacent mucosae (Mason 1988). In these infections, the laryngeal opening was totally or severely obstructed by the plaque-like mass, and death was considered to be a result of asphyxiation. When parts of the digestive tract are damaged, the lesions can cause difficulty in swallowing and result in starvation.

In histological sections from Mallards infected with *S. crassicauda* and *S. incognita*, worms are surrounded by varying amounts of necrotic debris and a cellular infiltrate consisting of mononuclear and multinucleated phagocytes, lymphocytes, and heterophiles (Sterner and Stackhouse 1987). In addition, granulomatous inflammation surrounding amorphous debris was observed in the gizzard musculature. In Chilean Flamingos infected with *S. incognita*, necrotic cellular debris and polymorphonuclear cells formed a pseudomembrane over eroded portions of the tissues at the junction of the gizzard and proventriculus (Fox et al. 1974).

DIAGNOSIS

A combination of clinical signs and the presence of spirurid-type eggs in feces may be used for antemortem diagnosis of streptocariasis. The eggs of *S. crassicauda* are 37 μm long and 18 μm wide and those of *S. incognita* are 36 μm long and 20 μm wide (Gibson 1968). However, lesions may develop before infections become patent, making the detection of eggs in feces unreliable as a diagnostic method. At postmortem, the nematodes can be extracted from lesions and identified by their characteristic morphology (Figure 18.1c).

The morphology of adult species of *Streptocara* is characteristic and distinguishable from most of these other nematodes by the presence of a collarette at the cephalic end. With the exception of several closely related acuarioid nematodes, the collarette is absent in these other groups (Figure 18.1c). There is some confusion regarding which species of *Streptocara* cause disease. *Streptocara crassicauda* appears to be limited to the gizzard, while *S. incognita* is found in the esophagus and proventriculus in addition to the gizzard. On the basis of experimental infections in three species of ducks, Laberge and McLaughlin (1991) believe that *S. crassicauda* is unlikely to cause the esophageal and proventricular lesions reported in other studies and that *S. incognita* is more likely to be responsible for lesions occurring outside the gizzard. A recent report of parasitic esophagitis in Muscovy Ducks (*Cairina moschata*) in Italy caused by *S. incognita* supports this

hypothesis (Bano et al. 2005). Other nematode parasites that may occur in the gizzard include other acuarioid nematodes as well as the trichostrongyloid *Amidostomum* and the ascaridoid parasites, *Contracaecum* and *Porrocaecum*. Other nematodes that may also occur in the proventriculus and esophagus include ascarids and species of *Cyrnea*, *Echinuria*, *Tetrameres*, and *Capillaria*.

DISPHARYNX, ECHINURIA, AND *STREPTOCARA*

DOMESTIC ANIMAL HEALTH CONCERNS

Dispharynx nasuta is potentially pathogenic in domestic poultry. In experimentally infected chickens, Ramaswamy and Sundaram (1985) observed a mortality rate of 29.16% in chicks. Mortality due to dispharynxiasis was also observed in experimentally infected chickens in Cuba (Nagy et al. 1977). *Echinuria uncinata* is common in waterfowl occurring in the wild, and the cross-transmission to captive waterfowl populations occurring in outdoor facilities is a serious threat. There have been numerous outbreaks in domestic ducks that have usually resulted in the deaths of the animals (Buxton et al. 1952; Gräfner and Graubmann 1967; Respaldiza et al. 1979; Griffiths et al. 1985; Kock et al. 1987). This susceptibility also exists for outbreaks of streptocariasis (Boughton 1969; Fox et al. 1974; Mason 1988). Often, a search in the water of aviaries in which enzootics of streptocariasis have occurred will reveal the crustacean intermediate hosts.

WILDLIFE POPULATION IMPACTS

Dispharynxiasis can cause mortality in wild birds such as Ruffed Grouse (Goble and Kutz 1945). *Dispharynx* is considered to be one of the most important parasites of game birds in the eastern US (Wehr 1971), and fatal infections have been reported. The widespread range of *D. nasuta* is of particular importance in the management of declining wild species that may be susceptible to dispharynxiasis. Infections have recently been reported in experimentally introduced Whooping Cranes (*Grus americana*) in Florida (Spalding et al. 1996). Although infections were of low prevalence and intensity and no pathogenic effects were noted, the potential for *Dispharynx* to cause pathology in a broad range of avian hosts is a cause for concern. Careful monitoring of these birds and other wild populations is warranted. A species of *Dispharynx* has also been reported from domestic chickens in the Galapagos Islands, raising concerns about its potential transmission to endemic host species (Gottdenker et al. 2005).

Species of *Streptocara* and *Echinuria* have similar potential for causing disease in threatened or endangered populations of wild birds. The endangered Laysan Duck (*Anas laysanensis*) with a single population in the northwestern Hawaiian Islands is susceptible to infection with *E. uncinata*. Mortality caused by this parasite was recently reported (Work et al. 2004).

TREATMENT AND CONTROL

There are few good options for controlling infections with *Dispharynx*, *Echinuria*, and *Streptocara* in free-living and captive populations, since definitive and intermediate hosts have a cosmopolitan range. Although *D. nasuta*, *E. uncinata*, *and S. crassicauda* are common and widespread in their natural hosts, there is a serious risk of disease and mortality when captive birds in zoos, private collections, and other aviaries are exposed to these parasites. *Dispharynx nasuta* can be transmitted to many captive, valuable, and endangered birds (Lindquist and Strafuss 1980; Stauber and Schussman 1985; Blasdel and Lasswell 1986; Bolette 1998a, b). Parasite eggs shed from locally infected wild birds can result in the spread of infection to captive animals through the isopod intermediate hosts.

Preventing the spread of acuarioid nematodes in domestic birds and in zoo collections is dependent on minimizing exposure to infected wild birds and in reducing or eliminating intermediate hosts. House Sparrows and Rock Pigeons are hosts of *D. nasuta*, and both are often found in zoos with outdoor displays and in the vicinity of larger aviaries. As *E. uncinata* is globally widespread, it could occur in any zoos or private collections of waterfowl that are kept on open water where wild birds can deposit eggs of *E. uncinata* and where suitable intermediate hosts, particularly *Daphnia* species, can serve as intermediate hosts. Similarly, the overlap of captive and domestic birds with wild waterfowl should be avoided in order to prevent the introduction of *Streptocara* species into the captive populations.

Controlling outbreaks of dispharynxiasis in captive birds is dependent on elimination of the isopod intermediate hosts. This is particularly difficult in outdoor aviaries. However, the use of mild insecticides such as pyrethrin preparations may help to reduce their numbers (Shanthikumar 1987). Various anthelmintics can be used to treat dispharynxiasis. Schulman et al. (1992) treated infected African jacanas (*Actophilornis africanus*) successfully with ivermectin. Shedding of eggs stopped and preventricular swelling resolved following treatment. Other anthelmintics that are effective include thiabendazole, levamisole, and mebendazole (Stauber and Schussman 1985; Shanthikumar 1987).

One of the most reliable means of controlling echinuriasis in captive ducks is to reduce or eliminate the presence of the daphnid intermediate hosts in duck

ponds and holding facilities. Species of *Daphnia* survive well in stagnant ponds and lakes with little or no current, and increasing the flow of water should reduce their numbers (Wood 1974). Adult *E. uncinata* can overwinter in ducks, and it may be possible for larvae in the intermediate hosts to also survive winter (Austin and Welch 1972). Therefore, elimination or reduction of *Daphnia* or removal of birds from infested areas can control this parasite in captive host populations.

Early attempts to treat echinuriasis with anthelmintics were largely unsuccessful. More recently however, the treatment of Red-breasted Geese (*Branta ruficollis*) with ivermectin has shown promise (Kock et al. 1987). Additional treatment with antibiotics may control secondary bacterial infections, and granulomas may resolve after death of the worms.

Eradication of crustaceans with copper sulfate treatment has been suggested for control of streptocariasis (Fox et al. 1974). In zoological collections, birds should be carefully screened for possible infection with *Streptocara* by examination of feces before they are introduced to a new facility. Various anthelmintics including fenbendazole, levamisole, and mebendazole may be efficacious in treating streptocariasis (Denev et al. 1977; Dalton 1980). Administration of a liquid gruel diet may also assist recovery.

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19
Tracheal Worms

M. A. Fernando and John R. Barta

INTRODUCTION

Tracheal worms are cosmopolitan, strongylid nematodes that infect the respiratory tract of wild and domestic birds. Throughout this chapter we discuss gapeworms (*Syngamus* spp.) and cyathostomes (*Cyathostoma* spp.) separately. The widely reported gapeworm, *Syngamus trachea*, is relatively common in wild and range-reared domestic fowl such as turkeys, chickens, pheasants, and perching birds and has been found much less commonly in waterfowl, herons and storks, pelicans, and woodpeckers. Uncommonly, *S. trachea* has been shown to be a primary pathogen in young game birds, especially pheasants (Lister 1989). Among cyathostomes, the most commonly recorded species, *Cyathostoma bronchialis*, is likewise found globally, most commonly in waterfowl (Anseriformes). There are a few rare reports of mortality in wild populations (e.g., Karstad and Sileo 1971), but these parasites are usually not regarded as serious pathogens in the wild. However, confined rearing of susceptible hosts and the resulting enhanced transmission can make these serious parasites of young birds. *Cyathostoma (Hovorkonema) variegatum* has been recovered from wild birds in respiratory distress, suggesting that these parasites may be an unrecognized cause of wild bird mortality (Krone et al. 2007). Only a few worms are capable of causing dramatic clinical signs under some circumstances.

Many wild birds may act as reservoirs of infection to domestic animals (and vice versa), and transmission of both *S. trachea* and *C. bronchialis* between wild and domestic hosts has been demonstrated.

SYNONYMS

Gapes, gapeworm infection, cyathostomiasis, syngamiasis, tracheal worm infection.

HISTORY

Madsen (1950) gives a short history of the records of *S. trachea* from poultry and other birds. Originally recorded in 1779 from poultry by Weisenthal, as a "worm from poultry," it was named in 1811 by Montagu as *Fasciola trachea* from a pheasant and partridge. In 1886, Seibold recognized the worm as a nematode that he called *S. trachealis* and provided the first description. It was called *Syngamus trachealis* until Chapin (1925) corrected the nomenclature to *S. trachea*. Since this time, as noted below, *S. trachea* has been found in many wild and domestic gallinaceous and passeriform birds.

Some of these syngamid nematodes that did not reside in permanent copula were placed in the genus *Cyathostoma* (cf. *Syngamus* spp. that remain paired even when removed from the animal or placed in fixative, see Borgsteede and Okulewicz, 2001). The genus *Cyathostoma* was first described in 1849 by Blanchard on the basis of his description of *Cyathostoma lari* from the orbital cavity of the black-headed gull, *Larus ridibundus*. Since then, a number of *Cyathostoma* species have been described from the orbital cavity and throughout the respiratory tract of a wide range of wild and domestic waterfowl and a wide range of wild and captive raptors (Ali 1970).

ETIOLOGY

Tracheal worms of birds are parasitic nematodes belonging to the order Strongylida, superfamily Strongyloidea, family Syngamidae (Lichtenfels 1980). Species in the subfamily Syngaminae belong to the genera *Syngamus* or *Cyathostoma* and are usually considered to occur only in birds (Chapin 1925). *Cyathostoma* species have been assigned to one of two subgenera (e.g., *Cyathostoma* (*Cyathostoma*) spp. or *Cyathostoma* (*Hovorkonema*) spp.) on the basis of details of the male copulatory bursa and spicule length (Lichtenfels 1980; Borgsteede and Okulewicz 2001). Some confusion surrounds the naming of cyathostomes infecting birds in various surveys and case reports. For example, *Cyathostoma (Hovorkonema) variegatum* has been referred to as a *Hovorkonema* sp. in the literature

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(Krone et al. 2007 and others). Throughout this chapter, we refer to all cyathostomes as *Cyathostoma* spp. belonging to the subgenera *Hovorkonema* or *Cyathostoma* as appropriate (see Lichtenfels 1980; Borgsteede and Okulewicz 2001). A detailed examination of the taxonomic relationships among the various syngamid worms infecting birds is beyond the scope of this chapter. For the sake of simplicity, we discuss "gapeworms" (*Syngamus* species) that all reside in permanent copulation in the trachea of birds (Table 19.1) and the closely related "cyathostomes" (*Cyathostoma* spp.) that reside in the trachea, air sacs, or orbital sinuses of birds and that do not pair up permanently (Table 19.2).

Morphology

Gapeworms and cyathostomes in birds are typical strongylid nematodes with a moderate to large buccal capsule or mouth and prominent copulatory bursa in the male worms. These parasites are associated with the mucosal surface of the respiratory tract of birds. Determining whether syngamid worms found in the respiratory tract belong to the genera *Sygamus* or *Cyathostoma* is relatively straightforward; species of *Syngamus* are found in permanent copulation as adults and are found attached to the trachea of infected hosts, whereas species of *Cyathostoma* are not permanently joined in copulation and can be found in the trachea, air sacs, or orbital sinuses of their hosts. Although there are multiple species in each genus, the morphology of the most common species of each is described below. Differentiating species within each of these genera is difficult and relies on detailed morphological and morphometric observations of the adult worms. The general morphology of the remaining species in each genus is similar.

Syngamus trachea

Both the male and female are bright red, permanently in copulo, and their bodies form a Y shape (Figure 19.1a). The buccal capsule is large and well developed with eight (sometimes nine) teeth at the base, which allows them to live attached to the wall of the trachea (Figure 19.1b). Here, they often form nodules. The males are 2–6 mm long and 200 μm in diameter with a bursa and slender spicules 55–82 μm long at the posterior end. The females are 5–40 mm in length and 350 μm in diameter. They shed ellipsoidal eggs 85–90 μm × 50 μm that possess distinct opercula at each end.

Cyathostoma bronchialis

In contrast to *S. trachea*, these worms are not permanently in copulo. The buccal capsule is cup shaped with six to seven teeth at the base (Figure 19.1c). The males are 4–6 mm long, the bursa is well developed and the spicules are about 0.5 mm in length. The females are 16–30 mm in length and their eggs measure 75–83 μm × 50–62 μm with a single indistinct operculum at the narrow end of the egg.

DISTRIBUTION AND HOST RANGES

Gapeworms (*Syngamus* spp.) have been reported mainly from Europe and North America but likely infect birds globally in tropical and temperate to cold temperate climates (Table 19.1). Not surprisingly, many of the reported host species are ground-feeding birds such as chickens, grouse, turkeys, and pheasants or invertebrate-feeding birds such as robins, herons, cranes, gulls, and jays. Surprisingly, cyathostomes have been reported quite commonly from predatory birds such as merlins, hawks, and owls (Table 19.2) that do not generally feed directly on the invertebrates that act as intermediate hosts for these worms. Several authors have suggested that predatory birds acquire infections by eating rodents or smaller birds with infected intermediate hosts containing infective larvae of *Cyathostoma* spp. in their alimentary tracts (Simpson and Harris 1992; Lavoie et al. 1999).

LIFE CYCLE AND EPIZOOTIOLOGY

Syngamus trachea

Eggs laid by adult female *S. trachea* within the trachea are coughed up, usually swallowed, and passed in the feces of the host. Under optimal conditions at 29°C, the larvae take about a week to develop to the infective third stage within the eggs. The larvae can hatch spontaneously. Development is much longer under less favorable conditions. Eggs do not develop at ambient temperatures at or below 16°C (Baruš 1966b). Larvae developing within the eggshell reach infectivity in 42 days at 17°C, 25–28 days at 19°C, 16 days at 21°C, 13–14 days at 25°C, and only 9 days at 27°C. Hatched larvae are susceptible to desiccation (Ortlepp 1923). Both hatched infective larvae and eggs containing infective larvae are infective when ingested by the host.

In addition to direct transmission via ingestion of embryonated eggs or larvae from the environment, *S. trachea* frequently incorporates an optional transport (paratenic) host in its life cycle. Earthworms act as transport hosts and may be the principal means for infective larvae to overwinter in colder climates. The larvae of *S. trachea* have been shown to remain viable and infective for more than 3 years, encapsulated in earthworm muscle. In experimentally infected earthworms (*Eisenia foetida*), encysted third-stage larvae

Table 19.1. Examples of *Syngamus* species reported from wild birds.

Syngamus species	Host common name	Host species	Country reported	Reference
Syngamus trachea	Ring-necked Pheasant	*Phasianus colchicus*	Commercially reared in Serbia	Pavlovic et al. (2003)
			Commercial flock, BC, Canada	Moynihan and Musfeldt (1950)
			Britain	Campbell (1935)
	Willow Ptarmigan	*Lagopus lagopus*	Norway	Wissler and Halvorsen (1975)
	Carrion Crow	*Corvus corone*	Britain	Campbell (1935)
	Rook	*Corvus frugilegus*		
	Eurasian Jackdaw	*Corvus monedula*		
	Eurasian Magpie	*Pica pica*		
	European Starling	*Sturnus vulgaris*		
	House Sparrow	*Passer domesticus*		
	Purple Sandpiper	*Calidris maritima*		
	Greater Rhea	*Rhea americana*	USA	De Wit (1995)
	Wild Turkey	*Meleagris gallopavo*	Commercially reared in Eastern Europe	Zavadil (1966); Baruš (1966a)
	Red-and-yellow Barbet	*Trachyphonus erythrocephalus*	In aviary in USA	Nevarez et al. (2002)
	Song Thrush	*Turdus philomelos*	Britain	Campbell (1935)
	Redwing	*Turdus iliacus*		
	Eurasian Blackbird	*Turdus merula*		
	American Robin	*Turdus migratorius*	Delaware, USA	Welte and Kirkpatrick (1986)
Syngamus alcyone	Belted Kingfisher	*Megaceryle alcyon*	Massachusetts, USA and Ontario, Canada	Boyd and Fry (1971)
Syngamus gibbocephalus	Pintail Snipe	*Gallinago stenura*	Russia	Ryzhikov (1949)
Syngamus microspiculum	Great Cormorant	*Phalacrocorax carbo*	Turkestan	Skrjabin (1915)
Syngamus palustris	Ruff	*Philomachus pugnax*	Russia	Ryzhikov (1949)
	Spotted Redshank	*Tringa erythropus*		
Syngamus taiga	Eurasian Nutcracker	*Nucifraga caryocatactes*	Russia	Ryzhikov (1949)
	Sedge Warbler	*Acrocephalus schoenobaenus*		
	White Wagtail	*Motacilla alba*		
Syngamus sp. (eggs only)	Magnificent Bird-of-paradise	*Cicinnurus magnificus*	Papua New Guinea	Varghese (1987)

Note: Syngamus trachealis Siebold 1936, *Syngamus parvus* Chapin 1925, *Syngamus gracilis* Chapin 1925, and *Syngamus merulae* Baylis 1926 are considered to be synonyms of *Syngamus trachea* (Montagu, 1811) as discussed in detail by Madsen (1950). Also called "the gapeworm."

Table 19.2. Examples of *Cyathostoma* species reported from wild birds.

Species of Cyathostoma	Host common name	Host species	Country	Reference
C. lari	Black-headed Gull	*Larus ridibundus*	Britain	Pemberton (1959)
	Rook	*Corvus frugilegus*		
	Eurasian Jackdaw	*Corvus monedula*		
	European Starling	*Sturnus vulgaris*		
	Gray Heron	*Ardea cinerea*		
	Eurasian Kestrel	*Falco tinnunculus*	Britain	Simpson and Harris (1992)
	Eurasian Buzzard	*Buteo buteo*		
	Eurasian Sparrow Hawk	*Accipiter nisus*		
	European Herring Gull	*Larus argentatus*	Britain	Threlfall (1965)
C. (Hovorkonema) americanum	Northern Goshawk	*Accipiter gentilis*	The Netherlands	Borgsteede and Okulewicz (2001)
	Eurasian Buzzard	*Buteo buteo*		
	Rough-legged Hawk	*Buteo lagopus*		
	Broad-winged Hawk	*Buteo platypterus*	Quebec, Canada	Lavoie et al. (1999)
	Northern Goshawk	*Accipiter gentilis*		
C. bronchialis	Canada Goose	*Branta canadensis*	Ontario, Canada	Fernando et al. (1973)
	Swan Goose	*Anser cygnoides*		Karstad and Sileo (1971)
	Geese	Various *Anser* spp.	Europe and USA	Chapin (1925)
	Mallard	*Anas platyrhynchos* and others		
C. (Hovorkonema) variegatum	Eurasian Buzzard	*Buteo buteo*	Spain	Sanmartin et al. (2004)
	Northern Goshawk	*Accipiter gentilis*	Germany	Krone et al. (2007)
	Eurasian Sparrow Hawk	*Accipiter nisus*		
	Eurasian Buzzard	*Buteo buteo*		
	Western Marsh-Harrier	*Circus aeruginosus*		
	White-tailed Eagle	*Haliaeetus albicilla*		
	Common Crane	*Grus grus*		
	Whooping Crane	*Grus americana*	Florida, USA	Spalding et al. (1996)
Cyathostoma sp.	Barred Owl	*Strix varia*	Quebec, Canada	Lavoie et al. (1999)
	Snowy Owl	*Bubo scandiacus*		
	Northern Harrier	*Circus cyaneus*		
	Northern Goshawk	*Accipiter gentilis*		
	Broad-winged Hawk	*Buteo platypterus*		
	Belted Kingfisher	*Megaceryle alcyon*	Massachusetts, USA.	Boyd and Fry (1971)

of *S. trachea* are found in the body wall of the earthworm, deeply embedded in the muscle in a thin hyaline cyst. Encysted larvae do not affect vitality of the earthworms and hence they can act as cumulative reservoirs (Clapham 1934).

Slugs, ants, beetles, and many other invertebrates are capable of acting as paratenic hosts for this parasite and these may provide additional opportunities for dissemination of infections.

When eggs of *S. trachea* are ingested by a suitable avian host, larvae hatch within the intestines and can be recovered from the liver shortly thereafter (Fernando et al. 1971). This suggests that most larvae arrive in the lungs via the bloodstream. The larvae are seen in the pulmonary connective tissue and air sac capillaries as early as 4 h postinfection (PI) and the atria of the lungs by 24 h (Figure 19.2a). Worms are recovered from the lungs up to 7 days PI and from the trachea as early as 7 days PI. Most worms are in the trachea by 11 days PI and the females are fertile by 14 days PI. Worms have been found attached to the cartilage of the tracheal rings at day 14 PI. Male worms are always found permanently attached to the tracheal mucosa. This is normal. In some instances, they penetrate the tracheal rings and the host response forms a nodule around the anterior end of the male worm. The females are attached to the males in permanent copula but are not attached permanently to the tracheal mucosa. Female worms feed at multiple locations around the attachment site of their male partner. The first eggs are found in the feces 17–20 days PI (Wehr 1937; Clapham 1939; Fernando et al. 1971).

Gapeworm infection has been documented in commercially raised pheasants (Moynihan and Musfeldt 1950; Pavlovic et al. 2003) and turkeys (Zavadil 1966). In Serbia, *S. trachea* is the most prevalent nematode species (37.2%) in commercially raised pheasants under the age of 14 weeks (Pavlovic et al. 2003). Commercially raised turkeys may be particularly susceptible throughout life to infection with *S. trachea* resulting in "mass-perishing" of these birds (Zavadil 1966). In a longitudinal study of seasonally range-reared breeding turkeys in Eastern Europe, newly acquired infections arose each spring upon access to infected earthworms. Transmission peaked in late summer, possibly because larval maturation within eggs occurs most quickly at higher temperatures and because earthworm populations are larger or more available (Baruš 1966a). In the same study, almost 7% of the earthworms collected within the turkey runs contained 1–12 infective larvae of *S. trachea*.

Free-living hosts of *Syngamus* spp. include Rooks (*Corvus frugilegus*), crows, pheasants, robins, jays, magpies, starlings, and many other species (Campbell 1935). Prevalence of this parasite varies widely by host

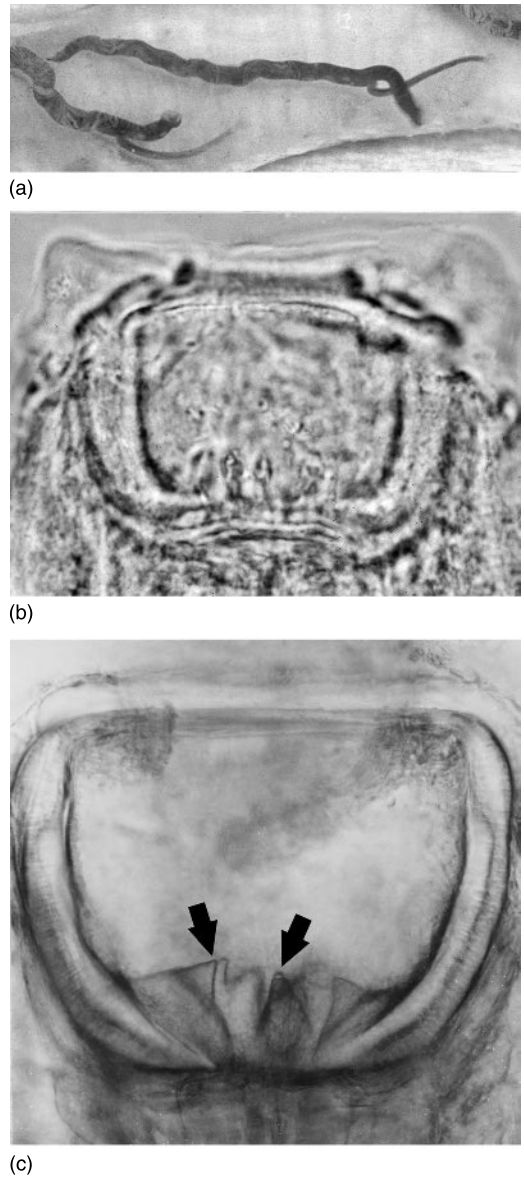
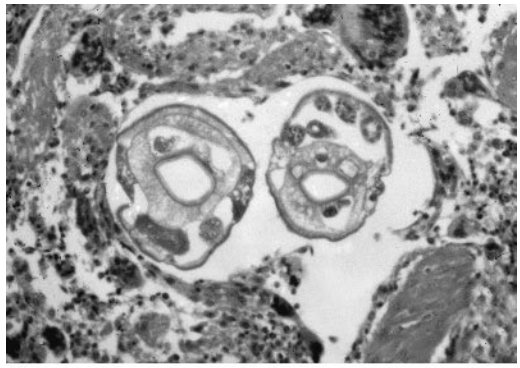
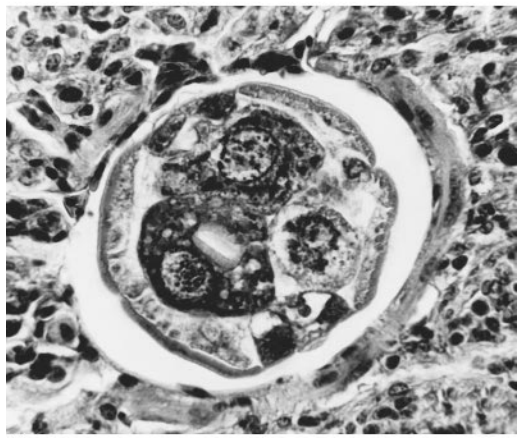


Figure 19.1. (a) and (b) *Syngamus trachea* from a Ring-necked Pheasant (*Phasianus colchicus*). (a) Pairs of male and female worms in copulo. (b) Buccal capsule of young adult (L5) recovered 5 days postinfection (PI). (c) *Cyathostoma bronchialis* from a Canada Goose (*Branta canadensis*). Buccal capsule of young adult (L5) recovered 5 days PI. Note triangular teeth (arrows) at the base of the buccal cavity. Reproduced from Fernando et al. (1973), with permission from the *Journal of Parasitology*.



(a)



(b)

Figure 19.2. (a) *Syngamus trachea* in lung of a Ring-necked Pheasant (*Phasianus colchicus*) at 24 h postinfection (PI). (b) Larvae of *Cyathostoma bronchialis* distending parabronchus in lung of a Canada Goose (*Branta canadensis*) at 3 days PI. Reproduced from Fernando et al. (1973), with permission from the *Journal of Parasitology*.

species and host age class. For example, in a series of examinations of wild birds over a number of years in the UK, starlings had a prevalence of about 18–35% (Lewis 1925, 1926; Taylor 1928; Campbell 1935) with intensity of infections ranging from 1 to 5 pairs of worms per bird. In a large survey examining gapeworm infections in more than a thousand birds of 53 species, from 1 to 82% of individuals of 13 different species of birds were infected with gapeworms (Campbell 1935). In this same study, the prevalence of gapeworm infections and their intensities in most hosts were shown to decrease with the age of the host. The most striking example of this was observed in the Rook in which the young of the year had a prevalence of 99%, 1-year-old birds 40%, and adults 8.6% (Campbell 1935). This drop in prevalence occurs even over a single breeding season. Prevalence of *S. trachea* in young Rooks was 98% in May, 85% in June, and 62% in August, suggesting that Rooks are able to expel the worms after a period of infection (Rice 1929). Intensity of infection with *S. trachea* also decreased as the birds aged. Young Rooks had up to 40 pairs of *S. trachea* in their trachea but most infections in adult birds had only 1 or a few pairs of worms (Elton and Buckland 1928). In contrast to most wild birds, prevalence of infection in adult and juvenile Ring-necked Pheasants (*Phasianus colchicus*) is similar (Campbell 1935).

Infections with *S. trachea* in wild birds have been implicated as a cause of outbreaks on game bird farms, particularly pheasant farms, as well as in range poultry (Elton and Buckland 1928; Clapham 1934; Campbell 1935).

Cyathostoma bronchialis

Eggs of *C. bronchialis* are coughed up, swallowed, and passed in the feces of the host. Under favorable environmental conditions, larvae take about 10 days to develop and then the eggs hatch spontaneously to release larvae. In contrast to the larvae of *S. trachea*, hatched larvae or eggs containing third-stage larvae are not directly infective to captive Snow Geese (*Chen caerulescens*) or Canada Geese (*Branta canadensis*) (Cram 1927; Fernando et al. 1973). Earthworms containing infective *C. bronchialis* larvae are, however, sources of infection when fed to susceptible geese (Fernando et al. 1973). Earthworms are necessary for transmission and are therefore obligate intermediate hosts. Like larvae of *S. trachea*, infective larvae of *C. bronchialis* can survive and overwinter in earthworms for several years but details about this host–parasite association are not known.

It is perhaps surprising to find cyathostome infections in birds that do not generally feed on invertebrates such as the Merlin (*Falco columbarius*), the Northern Goshawk (*Accipiter gentilis*), and the Snowy Owl (*Bubo scandiacus*) (Lavoie et al. 1999). In these cases, infections are most likely transmitted through infected invertebrates present in the alimentary tract of normal prey species. Sparrow hawks may acquire *Cyathostoma* infections in a similar manner (Simpson and Harris 1992).

After an infected earthworm is ingested, larvae of *C. bronchialis* can be recovered from abdominal air sacs and lungs as early as 1–4 h PI (Figure 19.2b). The route of migration from the intestinal tract to the pulmonary spaces is not known. Worms move to the trachea by 6 days PI. By 13 days PI, most males and females attain their maximum size, have mated, and

eggs are found in the tracheal mucus. Eggs are shed in the feces shortly thereafter (Fernando et al. 1973).

Wild birds are usually not heavily or commonly infected with cyathostomes. For example, infections with cyathostomes were found in only 7 of 20 species of wild birds during an extended study at three different locations in Germany. Prevalence of infection ranged from <1% to almost 20%, but intensities of infection ranged from only 1 to 5 worms per bird (Krone et al. 2007). In Canada, 394 wild-caught birds of prey belonging to 24 species were examined for cyathostome infections. Seven species of hawks and owls were found to be infected at prevalences that ranged from 4 to 33% (Lavoie et al. 1999). In the latter study, intensity of infection varied widely from 2 to approximately 100 worms per bird. Most birds were infected with only a few worms.

CLINICAL SIGNS

Clinical signs of infection with *S. trachea* vary depending on the size of the bird and the intensity of infection. Although the parasite infects a wide range of avian species, pheasants appear to be particularly susceptible to infection and clinical outbreaks of disease have been recorded (Pavlovic et al. 2003). Young birds are severely affected by migration of larvae through the lungs and develop pneumonia. Approximately 2 weeks after infection adult worms can block the trachea, giving rise to the typical clinical sign of "gaping" or gasping for air with outstretched necks and open mouths (Figure 19.3). Head shaking and bouts of coughing are seen in some birds. Severely affected young birds may stop drinking, deteriorate rapidly, and die (Clapham 1934; Levine 1968). Adult birds show few clinical signs except an occasional cough.



Figure 19.3. *Syngamus trachea.* Ring-necked Pheasant (*Phasianus colchicus*) with intense infection at 2 weeks postinfection. The outstretched neck and open mouth are typical clinical signs of "gaping" or gasping for air.

Even though *C. bronchialis* can be a significant pathogen in young captive waterfowl (Karstad and Sileo 1971), most wild birds infected with cyathostomes show no signs of disease. In those rare instances where clinical signs are present, they include emaciation and/or dyspnea resulting from impaired respiratory function associated with air sacculitis (Lavoie et al. 1999). Clinically affected birds had higher numbers of worms in their respiratory tracts.

PATHOGENESIS AND PATHOLOGY

Gapeworms

The pathogenesis of the lesions induced by *S. trachea* in the lungs and trachea in chickens and pheasants has been studied in detail (Wetzel and Fortmeyer 1964; Baruš and Blazek 1965). Grossly visible white foci, approximately 0.2 mm in diameter, do not appear in the lungs of pheasants until 7–9 days after experimental infection. However, microscopic lesions are seen as early as 3 days PI (Fernando et al. 1971). Early pulmonary lesions associated with larval migration through the lungs include an increase in the number of lymphocytes within the connective tissue and the parabronchi, cuffing of larger vessels by lymphocytes through migration of lymphocytes and heterophils from the blood stream, disappearance of the normal architecture of the air capillaries, and consolidation of pulmonary lobules. Giant cells and granulocytes fill the atria by 4 days PI and the lumina of the parabronchi by 7 days PI. Infiltration of the lamina propria of the secondary and primary bronchi and collapse of some of the secondary bronchi may be seen from the seventh day onward. Most pathogenic changes appear associated with an inflammatory host response to the presence of larvae and their antigens.

Worms in the trachea and bronchi give rise to hemorrhagic tracheitis and bronchitis. Worms are found attached to the cartilage of the tracheal rings by day 14 PI in pheasants (Figure 19.4a). Worms cause direct mechanical damage to the mucosa and produce hemorrhage as they feed on blood. Host-mediated inflammatory responses to these mechanical lesions and perhaps worm antigens and/or secretory products exacerbate the mechanical damage.

In Ring-necked Pheasants, pale tracheal nodules form at the point of attachment of the anterior end of male worms and may be visible on the interior or exterior surfaces (Figures 19.4b and 19.4c). These nodules are mainly composed of hyperplastic peritracheal connective tissue (Fernando et al. 1971). Male worms can perforate the mucosa and attach to the tracheal cartilage

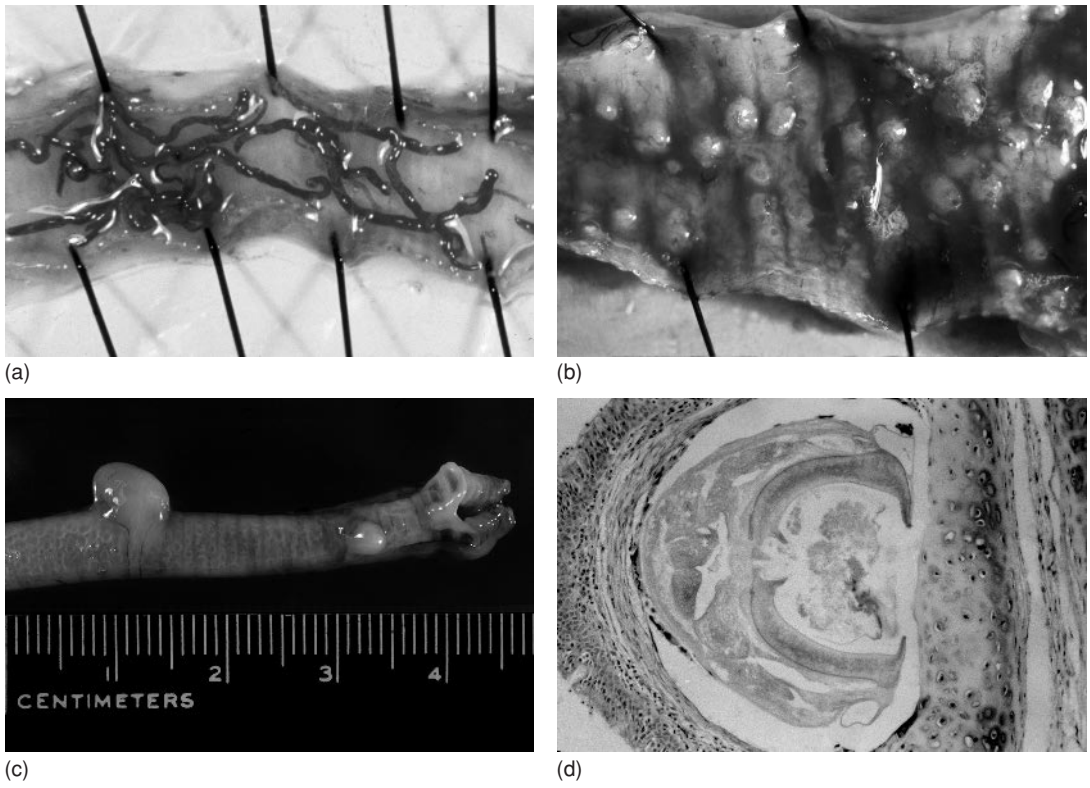


Figure 19.4. *Syngamus trachea* from ring-necked pheasant (*Phasianus colchicus*). (a) Worms blocking trachea at 14 days postinfection (PI). (b) Pea-sized nodules on the mucosal surface of the trachea at the points of attachment of the anterior end of male worms at 14 days after experimental infection. (c) Large nodule on the outer surface of the trachea 6 weeks after experimental infection. The head and anterior half of the body of the male worm is deeply embedded in the connective tissue of the nodule. (d) Buccal capsule attached to tracheal cartilage 14 days PI.

(Figure 19.4d). Male worms sometimes penetrate so deeply that they cause rupture and proliferation of the perichondrium, lysis of tracheal cartilage, and, not uncommonly, perforation of the tracheal rings (Clapham 1935; Carrara 1961; Fernando et al. 1971).

Cyathostomes

Cyathostoma bronchialis causes bronchitis of the primary, secondary, and tertiary bronchi during development in geese (Fernando et al. 1973). Worms move up the trachea by day 6 PI, so they exert the most pathogenic effects in the lower respiratory tract on the day 5 and day 6 PI (Fernando et al. 1973). Lymphoid cuffing of interlobular arteries and lymphoid replacement of air capillaries appears to be a reaction to presence of worms in the parabronchi and superficial secondary bronchi. Severe bronchitis in the secondary and primary bronchi probably also reflects the period of most rapid growth of the developing worms. By 14 days PI, the bronchial lesions are completely resolved.

Varied lesions may be seen in the bronchi at later stages of infection (28 days PI in experimental studies) and presumably are caused by aspiration of adult worms and eggs. Eggs, cuticular material shed by developing worms, and whole worms elicit a pyogranulomatous bronchitis or pneumonia in the lungs. Aspiration pneumonia in goslings with patent infections may also be caused by aspiration of eggs and adult worms. This contrasts with the behavior of *S. trachea*, which either remains in the trachea after migration through the lungs or is coughed out. Concurrent lesions of aspergillosis have been reported in a "blue goose" (presumably a Snow Goose, *Chen caerulescens*), a Canada Goose and goslings, and goslings of domestic

geese (Christenson 1932; Fernando et al. 1973). These observations suggest that *C. bronchialis* may predispose birds to other respiratory infections. The association of fungal hyphae with cuticular remnants in the parabronchi and secondary bronchi of these birds supports this possibility.

Cyathostoma americanum and other unidentified *Cyathostoma* spp. have been implicated as causes of diffuse pyogranulomatous air sacculitis, pneumonia, and bronchitis in wild raptors in North America (Lavoie et al. 1999) and elsewhere. Lesions are generalized and appear to be associated primarily with inflammatory reactions to eggs in the air sacs (Hunter et al. 1993). Prevalence of *Cyathostoma (Hovorkonema) variegatum* was 3% among raptors and 9% in Goshawks, and this parasite was associated with thickened air sac walls and granulomatous lesions at the site of infection in 7 of 12 cases (Krone et al. 2007).

DIAGNOSIS

Syngamus trachea

A diagnosis is usually made on the basis of the typical clinical signs and confirmed by observation of worms in the trachea during necropsy. Characteristic eggs will be found in the feces. Eggs of *S. trachea* are approximately 80–100 μm × 50–60 μm and are bipolar with clearly visible opercula at both ends. Each egg contains a morula (about an eight-cell embryo) when freshly passed. Eggs of *S. trachea* superficially resemble those of *Capillaria* spp. However, the eggs of *Capillaria* spp. are usually smaller (<60 μm in length), have a thicker (and frequently brownish) eggshell, possess pronounced polar plugs, and contain a single cell when passed.

Cyathostoma bronchialis

Clinical signs related to infections with cyathostomes are neither common nor diagnostic. On necropsy, finding nematodes morphologically consistent with cyathostomes within the trachea and bronchi is diagnostic. Antemortem, patent infections can be diagnosed by finding ovoid morulated eggs with an indistinct polar operculum at the narrow end measuring 75–90 μm × 45–60 μm in feces.

IMMUNITY

In infections with either *S. trachea* or *C. bronchialis*, younger birds are more susceptible to the pathogenic effects of infection. Following an initial infection with *S. trachea*, pheasants acquire at least partial immunity to further infection (Olivier 1942). Immunity persists after the adult worms are lost. Turkeys are, however, susceptible throughout life to gapeworm infection (Varga 1971). Prevalence of infection with *Syngamus* drops considerably in free-living populations as birds age (see above), suggesting that most natural hosts of these parasites acquire resistance to infection as the birds mature.

Approximately 95% of chickens vaccinated with irradiated embryonated eggs and larvae of *S. trachea* are resistant on challenge (Zeigler 1966, 1968). Development of the worms is inhibited before they enter the trachea, suggesting that the host immune response acts on the migratory phase of the parasite. This immunity may reduce the number of adult worms that become established in the trachea, even in conditions where birds are exposed to significant numbers of larvae.

PUBLIC HEALTH CONCERNS

Gapeworms of birds (*Syngamus* or *Cyathostoma* species) are not known to infect mammals, including humans, and therefore pose no public health concerns. Tracheal worms infecting mammals (species of *Mammomanogamus*) are relatively common globally and have been reported as accidental infections of humans. Previously, these mammalian tracheal worms were considered to be species of *Syngamus* and thus there are a number of case reports of "syngamosis" in humans (e.g., Leers et al. 1985). No avian gapeworm has ever been implicated in a human infection.

WILDLIFE POPULATION IMPACTS

Clinical gapeworm and cyathostome infections in wild populations are uncommon, although subclinical infections with these parasites are frequent. For example, 23 of 107 wild raptors in Quebec, Canada, were infected with species of *Cyathostoma*, but clinical signs of emaciation and dyspnea attributable to gapeworm infection were evident in only 4 cases (Lavoie et al. 1999). All the clinically affected birds had a severe diffuse pyogranulomatous air sacculitis. Reports of birds clinically affected by species of *Syngamus* are equally uncommon despite high prevalence and high intensity in some hosts (e.g., young Rooks in Britain, see Campbell 1935).

DOMESTIC ANIMAL HEALTH CONCERNS

Wild birds including Rooks, pheasants, robins, jays, crows, magpies, and starlings can act as reservoir hosts of *S. trachea*. These reservoirs have been implicated as the source of outbreaks on game bird farms, particularly pheasant farms, as well as range poultry (LeaMaster 2007). Gapeworms (*Syngamus* spp.) are not host specific and *S. trachea*, at least, can be

transmitted among many orders and families of birds. Likewise, wild waterfowl are expected to act as reservoirs for species of *Cyathostoma* that infect domestic ducks and geese. Earthworms and perhaps other invertebrates are an important source of infection where poultry and game birds are reared on soil.

TREATMENT AND CONTROL

Gapeworm infections can be treated with a number of anthelmintic medications. Benzimidazole anthelmintics such as thiabendazole, mebendazole, flubendazole, and fenbendazole have demonstrated efficacy against gapeworm infections (Wehr and Hwang 1967; Ssenyonga 1982; Draycott et al. 2006). In the US, thiabendazole is registered for the control of S. *trachea* in pheasants. In Peru, Agrovet Market S.A. produces a broad-spectrum anthelmintic, Gallomec Plus®; each tablet contains 0.2 mg ivermectin, 30 mg fenbendazole, and 10 mg praziquantel, and is used to control *S. trachea* in poultry, particularly younger birds. Extralabel use of injectable ivermectin has been used with variable success to treat gapeworm and cyathostome infections in poultry and wild birds, including raptors.

Despite treatment, clinical signs of coughing may persist for a period of time because of the presence of dead and dying worms in the respiratory tract. In heavy infections with species of *Cyathostoma*, treatment may produce fatal pneumonia from the aspiration of dead and dying worms and inflammatory responses to the dead parasites. Opportunistic secondary bacterial or fungal pneumonias may require specific treatment in addition to anthelmintics to remove the worms.

Although gapeworm and cyathostome infections appear to be of minor significance in free-living wild birds, they can be a serious problem in commercially reared pheasants, chukars, and waterfowl as well as range-reared turkeys and chickens. The presence of large numbers of susceptible young birds in a relatively confined area can lead to severe and clinically apparent infections and mortality. Restricting access of young birds to heavily contaminated yards, older birds, or wild bird reservoir hosts will reduce the intensity of infections and thereby reduce clinically significant infections. Earthworms are particularly important in maintaining an infective environment because third-stage larvae of gapeworms can survive in earthworms for extended periods (Baruš 1966a) and earthworms are obligate intermediate hosts for cyathostomes. For this reason, restricting access of young birds to open ground may reduce the impact of these parasites in domestic species.

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20
Amidostomum and *Epomidiostomum*

Alan M. Fedynich and Nancy J. Thomas

INTRODUCTION

Gizzard worms of the genera *Amidostomum* and *Epomidiostomum* are commonly found in waterfowl within the family Anatidae. They have direct life cycles and are classified in the order Strongylida (bursate nematodes). Adult worms of both genera live under the koilin lining of the gizzard (ventriculus) and feed on blood. Pathology caused by ventricular nematodiasis has been reported in wild geese (Jerstad 1937; Herman and Wehr 1954; Harradine 1982; Tuggle and Crites 1984), ducks (MacNeill 1970; Crichton and Welch 1972; Turner and Threlfall 1975), and swans (MacNeill 1970). Intense infections can result in damage to the koilin lining and associated muscle and lead to gizzard dysfunction, emaciation, weakness, and potentially poor growth rates of juveniles (MacNeill 1970; Tuggle and Friend 1999). Debilitated hosts may be more susceptible to predation or infection by other pathogens (Tuggle and Friend 1999). Additionally, migratory birds in poor condition from ventricular nematodiasis may be unable to cope with the demands of migration and increased competition for limited food resources during winter (Herman and Wehr 1954).

SYNONYMS

Gizzard worm, ventricular nematodiasis, amidostomiasis, epomidiostomiasis.

HISTORY

Species of *Amidostomum* and *Epomidiostomum* were first reported from gizzards of anatids in Europe beginning as early as 1791 when Froelich described *Strongylus mucronatus* (=*Amidostomum anseris*). Linstow, Lundahl, Molin, Rudolphi, and Zeder in the 1800s and Baylis, Boulenger, Maplestone, Seurat, Skrjabin, Travassos, Wehr, and Wetzel in the 1900s found and described species in both genera (Yamaguti 1961; McDonald 1969).

In North America, *A. anseris* was first reported from domestic geese in New York (Cram 1925), followed by a report of *Amidostomum* sp. in wild Canada Geese (*Branta canadensis*), Snow Geese (*Chen caerulescens*), Ross' Geese (*Chen rossii*), Northern Pintail (*Anas acuta*), and Green-winged Teal (*Anas carolinensis*) in California (O'Roke 1928). Shortly thereafter, *Amidostomum spatulatum* was reported from Canada Geese (wild or captive not reported), which were also infected with *Epomidiostomum crami* (Wehr 1933a).

Although early literature from the Old World clearly established that *A. anseris* is pathogenic in anatids, evidence of morbidity and mortality in hosts from North America first emerged in 1926 when Canada Goose goslings were found to be particularly susceptible (Wickware 1941). Subsequent observations indicated that *A. anseris* could be a contributing factor in mortalities observed in wintering Canada Geese (Herman and Wehr 1954).

In the early twentieth century, parasitological surveys of birds (particularly game birds) began to provide a better picture of the worldwide distributions of species of *Amidostomum* and *Epomidiostomum* in their respective hosts. Later, a few studies focused on understanding the life histories of several species of gizzard worms occurring in North America (Cowan 1955; Leiby and Olsen 1965). However, the life histories of most species in both genera have not been thoroughly examined.

DISTRIBUTION AND HOST RANGE

Species of *Amidostomum* and *Epomidiostomum* have been found in at least 96 and 60 species of wild birds, respectively (Tables 20.1 and 20.2). Both *Amidostomum* and *Epomidiostomum* occur principally in the host family Anatidae within the order Anseriformes, which includes the ducks, geese, and swans. The distribution of these parasites reflects the geographic ranges of their hosts. In migratory waterfowl, *Amidostomum acutum*, *A. anseris*, *A. spatulatum*, and *Epomidiostomum uncinatum* are widely distributed,

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Table 20.1. Avian hosts infected with *Amidostomum* spp., geographic location where infection was reported, and reporting authors.

Host	Parasite	Location	Reference
Anseriformes: Anatidae			
African Black Duck (*Anas sparsa*)	*Amidostomum anseris*	NR	McDonald (1969)
American Black Duck (*Anas rubripes*)	*Amidostomum acutum*	Canada	Mahoney and Threlfall (1978)
	Amidostomum anseris	NR	McDonald (1969)
American Wigeon (*Anas americana*)	*Amidostomum acutum*	Canada	McLaughlin and McGurk (1987)
	Amidostomum anseris	Washington, USA	Jerstad (1937)
Ashy-headed Goose (*Chloephaga poliocephala*)	*Amidostomum anseris*	NR	Gower (1939)
Baikal Teal (*Anas formosa*)	*Amidostomum acutum*	NR	Czapliński (1962)
Bar-headed Goose (*Anser indicus*)	*Amidostomum spatulatum*	NR	McDonald (1969)
Barnacle Goose (*Branta leucopsis*)	*Amidostomum anseris*	The Netherlands	Borgsteede et al. (2006)
Barrow's Goldeneye (*Bucephala islandica*)	*Amidostomum anseris*	Canada	MacNeill (1970)
Taiga Bean-Goose (*Anser fabalis*)	*Amidostomum anseris*	Europe	Balicka-Ramisz et al. (2000) and Borgsteede et al. (2006)
	Amidostomum spatulatum	Europe	Baylis (1932) and Macko et al. (2002)
Black-bellied Whistling-Duck (*Dendrocygna autumnalis*)	*Amidostomum acutum*	Texas, USA	Fedynich et al. (1996a)
Black Scoter (*Melanitta nigra*)	*Amidostomum acutum*	Canada	Bourgeois and Threlfall (1982)
		The Netherlands	Borgsteede et al. (2006)
	Amidostomum anseris	NR	Gower (1939)
Black Swan (*Cygnus atratus*)	*Amidostomum cygni*	Australia	Mawson (1980)
Blue-winged Teal (*Anas discors*)	*Amidostomum acutum*	North America	Turner and Threlfall (1975) and Wallace and Pence (1986)
	Amidostomum anseris	NR	McDonald (1969)
Brant (*Branta bernicla*)	*Amidostomum anseris*	The Netherlands	Borgsteede et al. (2006)
Bufflehead (*Bucephala albeola*)	*Amidostomum acutum*	Canada	McLaughlin and McGurk (1987)
Canada Goose (*Branta canadensis*)	*Amidostomum acutum*	Wyoming, USA	NWHC (voucher no. 48808)*
	Amidostomum anseris	North America	Jerstad (1937) and Wehr and Herman (1954)
	Amidostomum spatulatum	North America	Wehr (1933a) and Purvis et al. (1997)
Canvasback (*Aythya valisineria*)	*Amidostomum acutum*	Canada	McLaughlin and McGurk (1987)
Cape Barren Goose (*Cereopsis novaehollandiae*)	*Amidostomum anseris*	Australia	Mawson (1980)
Chiloe Wigeon (*Anas sibilatrix*)	*Amidostomum acutum*	NR	McDonald (1969)
Cinnamon Teal (*Anas cyanoptera*)	*Amidostomum acutum*	NR	McDonald (1969)
Common Eider (*Somateria mollissima*)	*Amidostomum acutum*	Newfoundland	Bishop and Threlfall (1974)
	Amidostomum anseris	Sweden	Persson et al. (1974)
	Amidostomum fulicae	Massachusetts, USA	NWHC (voucher no. 48809)

Common Goldeneye (*Bucephala clangula*)	*Amidostomum acutum*	Europe	Czapliński (1962)
		Canada	Mahoney and Threlfall (1978)
	Amidostomum anseris	Canada	MacNeill (1970)
Common Pochard (*Aythya ferina*)	*Amidostomum acutum*	Europe	Czapliński (1962)
	Amidostomum anseris	NR	McDonald (1969)
Common Shelduck (*Tadorna tadorna*)	*Amidostomum acutum*	The Netherlands	Borgsteede et al. (2006)
	Amidostomum anseris	NR	McDonald (1969)
Cotton Pygmy-goose (*Nettapus coromandelianus*)	*Amidostomum acutum*	NR	Czapliński (1962)
Eurasian Wigeon (*Anas penelope*)	*Amidostomum acutum*	Europe	Petrova (1987) and Borgsteede et al. (2006)
	Amidostomum anseris	NR	Gower (1939)
Falcated Duck (*Anas falcata*)	*Amidostomum acutum*	NR	McDonald (1969)
Ferruginous Pochard (*Aythya nyroca*)	*Amidostomum acutum*	Europe	Czapliński (1962)
	Amidostomum fulicae	Europe	Czapliński (1962)
Freckled Duck (*Stictonetta naevosa*)	*Amidostomum acutum*	Australia	Mawson (1980)
Gadwall (*Anas strepera*)	*Amidostomum acutum*	Canada	McLaughlin and McGurk (1987)
		The Netherlands	Borgsteede et al. (2006)
	Amidostomum anseris	NR	McDonald (1969)
	Amidostomum fulicae	NR	McDonald (1969)
Garganey (*Anas querquedula*)	*Amidostomum acutum*	Europe	Czapliński (1962)
	Amidostomum anseris	NR	Gower (1939)
	Amidostomum auriculatum	Europe	Lomakin (1988)
Gray Teal (*Anas gracilis*)	*Amidostomum acutum*	Australia	Mawson (1980)
Greater Scaup (*Aythya marila*)	*Amidostomum acutum*	The Netherlands	Borgsteede et al. (2006)
	Amidostomum anseris	NR	Gower (1939)
Greater White-fronted Goose (*Anser albifrons*)	*Amidostomum acutum*	Europe	Czapliński (1962)
	Amidostomum anseris	Texas, USA	Purvis et al. (1997)
		Europe	Balicka-Ramisz et al. (2000) and Borgsteede et al. (2006)
	Amidostomum spatulatum	Europe	Czapliński (1962)
		Texas, USA	Purvis et al. (1997)
Green-winged Teal (*Anas carolinensis*)	*Amidostomum acutum*	Europe	Czapliński (1962)
		Taiwan	Schmidt and Kuntz (1972)
		Canada	Turner and Threlfall (1975)
	Amidostomum anseris	Texas, USA	Canaris et al. (1981)
	Amidostomum auriculatum	Europe	Lomakin (1988)

(*continues*)

Table 20.1. (*Continued*)

Host	Parasite	Location	Reference
	Amidostomum fulicae	NR	McDonald (1969)
	Amidostomum henryi	NR	McDonald (1969)
Greylag Goose (*Anser anser*)	*Amidostomum acutum*	NR	McDonald (1969)
	Amidostomum anseris	Europe	Petrova (1987) and Bolte et al. (2000)
	Amidostomum spatulatum	NR	McDonald (1969)
Harlequin Duck (*Histrionicus histrionicus*)	*Amidostomum acutum*	NR	McDonald (1969)
	Amidostomum anseris	NR	McDonald (1969)
Hawaiian Goose (*Branta sandvicensis*)	*Amidostomum anseris*	Hawaii, USA	Bailey and Black (1995)
King Eider (*Somateria spectabilis*)	*Amidostomum acutum*	NR	Czapliński (1962)
	Amidostomum anseris	NR	McDonald (1969)
	Amidostomum cygni	North America	Czapliński (1962)
Lesser Scaup (*Aythya affinis*)	*Amidostomum acutum*	Canada	McLaughlin and McGurk (1987)
	Amidostomum anseris	NR	McDonald (1969)
Lesser White-fronted Goose (*Anser erythropus*)	*Amidostomum anseris*	NR	McDonald (1969)
	Amidostomum spatulatum	NR	McDonald (1969)
Long-tailed Duck (*Clangula hyemalis*)	*Amidostomum acutum*	NR	Czapliński (1962)
Mallard (*Anas platyrhynchos*)	*Amidostomum acutum*	Europe	Czapliński (1962)
		Australia	Mawson (1980)
		North America	McLaughlin and McGurk (1987) and Gray et al. (1989)
	Amidostomum anseris	Canada	Crichton and Welch (1972)
		Bulgaria	Petrova (1987)
		Japan	Nakamura and Asakawa (2001)
	Amidostomum fulicae	NR	McDonald (1969)
Mandarin Duck (*Aix galericulata*)	*Amidostomum acutum*	NR	McDonald (1969)
Maned Duck (*Chenonetta jubata*)	*Amidostomum anseris*	NR	McDonald (1969)
Marbled Teal (*Marmaronetta angustirostris*)	*Amidostomum acutum*	NR	McDonald (1969)
Mottled Duck (*Anas fulvigula*)	*Amidostomum acutum*	Florida, USA	Kinsella and Forrester (1972)
		Texas, USA	Fedynich et al. (1996b)
Musk Duck (*Biziura lobata*)	*Amidostomum acutum*	Australia	Mawson (1980)
Mute Swan (*Cygnus olor*)	*Amidostomum anseris*	The Netherlands	Borgsteede et al. (2006)
	Amidostomum cygni	Belgium	Yamaguti (1961)
Northern Pintail (*Anas acuta*)	*Amidostomum acutum*	Canada	McLaughlin and McGurk (1987)
		Europe	Petrova (1987) and Borgsteede et al. (2006)
	Amidostomum anseris	Canada	Crichton and Welch (1972)

Northern Shoveler (*Anas clypeata*)	*Amidostomum acutum*	North America	Broderson et al. (1977) and McLaughlin and McGurk (1987)
		Europe	Petrova (1987) and Borgsteede et al. (2006)
	Amidostomum anseris	NR	McDonald (1969)
Pacific Black Duck (*Anas superciliosa*)	*Amidostomum acutum*	Australia	Mawson (1980)
Pink-footed Goose (*Anser brachyrhynchus*)	*Amidostomum anseris*	NR	McDonald (1969)
	Amidostomum spatulatum	NR	McDonald (1969)
Radjah Shelduck (*Tadorna radjah*)	*Amidostomum acutum*	Australia	Mawson (1980)
Red-breasted Goose (*Branta ruficollis*)	*Amidostomum ryzhikovi*	Russia (zoo)	Kosupko and Lomakin (1985)
Red-crested Pochard (*Netta rufina*)	*Amidostomum acutum*	NR	Czapliński (1962)
	Amidostomum fulicae	NR	McDonald (1969)
Redhead (*Aythya americana*)	*Amidostomum acutum*	Canada	McLaughlin and McGurk (1987)
	Amidostomum anseris	Washington, USA	Jerstad (1937)
Ring-necked Duck (*Aythya collaris*)	*Amidostomum acutum*	Canada	McLaughlin and McGurk (1987)
Ross' Goose (*Chen rossii*)	*Amidostomum anseris*	Texas, USA	Fedynich et al. (2005)
	Amidostomum spatulatum	NR	McDonald (1969)
Ruddy Duck (*Oxyura jamaicensis*)	*Amidostomum acutum*	Canada	McLaughlin and McGurk (1987)
Ruddy Shelduck (*Tadorna ferruginea*)	*Amidostomum acutum*	NR	McDonald (1969)
	Amidostomum anseris	NR	McDonald (1969)
Smew (*Mergellus albellus*)	*Amidostomum acutum*	NR	McDonald (1969)
Snow Goose (*Chen caerulescens*)	*Amidostomum anseris*	North America	Tuggle and Crites (1984) and Purvis et al. (1997)
	Amidostomum cygni	NR	McDonald (1969)
	Amidostomum spatulatum	North America	Tuggle and Crites (1984) and Purvis et al. (1997)
Spectacled Duck (*Anas specularis*)	*Amidostomum anseris*	NR	McDonald (1969)
Spectacled Eider (*Somateria fischeri*)	*Amidostomum fulicae*	Alaska, USA	NWHC (voucher no. 48802, 48803)
Spot-billed Duck (*Anas poecilorhyncha*)	*Amidostomum acutum*	India	Dubey and Pande (1964)
	Amidostomum fulicae	NR	McDonald (1969)
Surf Scoter (*Melanitta perspicillata*)	*Amidostomum acutum*	Canada	Bourgeois and Threlfall (1982)
	Amidostomum fulicae	Virginia, USA	NWHC (voucher no. 48810)
Trumpeter Swan (*Cygnus buccinator*)	*Amidostomum anseris*	Canada	MacNeill (1970)
Tufted Duck (*Aythya fuligula*)	*Amidostomum acutum*	The Netherlands	Czapliński (1962) and Borgsteede et al. (2006)
	Amidostomum anseris	NR	Gower (1939)

(*continues*)

Table 20.1. (*Continued*)

Host	Parasite	Location	Reference
Tundra Swan (*Cygnus columbianus*)	*Amidostomum acutum*	NR	McDonald (1969)
	Amidostomum anseris	Canada	MacNeill (1970)
		Japan	Nakamura and Asakawa (2001)
	Amidostomum cygni	Washington DC, USA (zoo)	Wehr (1933b)
		Europe	Czapliński (1962)
	Amidostomum spatulatum	NR	McDonald (1969)
Upland Goose (*Chloephaga picta*)	*Amidostomum anseris*	Falkland Islands	Harradine (1982)
White-headed Duck (*Oxyura leucocephala*)	*Amidostomum anseris*	NR	McDonald (1969)
White-winged Scoter (*Melanitta fusca*)	*Amidostomum acutum*	Canada	Bourgeois and Threlfall (1982)
		The Netherlands	Borgsteede et al. (2006)
	Amidostomum anseris	NR	Gower (1939)
Whooper Swan (*Cygnus cygnus*)	*Amidostomum acutum*	NR	McDonald (1969)
	Amidostomum anseris	NR	McDonald (1969)
	Amidostomum cygni	Bulgaria	Petrova (1987)
		Japan	Nakamura and Asakawa (2001)
	Amidostomum spatulatum	NR	Czapliński (1962)
Wood Duck (*Aix sponsa*)	*Amidostomum acutum*	North America	Thul et al. (1985)
	Amidostomum anseris	NR	McDonald (1969)
Yellow-billed Duck (*Anas undulata*)	*Amidostomum acutum*	Africa	Alexander and McLaughlin (1997)
Yellow-billed Pintail (*Anas georgica*)	*Amidostomum acutum*	NR	McDonald (1969)
Galliformes: Phasianidae			
Black-billed Capercaillie (*Tetrao parvirostris*)	*Amidostomum acutum*	Europe	Baruš et al. (1984)
Eurasian Capercaillie (*Tetrao urogallus*)	*Amidostomum acutum*	Europe	Baruš et al. (1984)
Hazel Grouse (*Bonasa bonasia*)	*Amidostomum acutum*	NR	Czapliński (1962)
Rock Ptarmigan (*Lagopus muta*)	*Amidostomum acutum*	NR	Czapliński (1962)
Gaviiformes: Gaviidae			
Red-throated Loon (*Gavia stellata*)	*Amidostomum cygni*	North America	Czapliński (1962)
Podicipediformes: Podicipedidae			
Little Grebe (*Tachybaptus ruficollis*)	*Amidostomum anseris*	NR	McDonald (1969)
Gruiformes: Aramidae			
Limpkin (*Aramus guarauna*)	*Amidostomum acutum*	Florida, USA	Conti et al. (1985)

Gruiformes: Rallidae			
American Coot (*Fulica americana*)	*Amidostomum acutum*	NR	Roudabush (1942)
	Amidostomum fulicae	NR	McDonald (1969)
Black-tailed Native-hen (*Gallinula ventralis*)	*Amidostomum tribonyx*	Australia	Mawson (1980)
Common Moorhen (*Gallinula chloropus*)	*Amidostomum anseris*	NR	Gower (1939)
	Amidostomum fulicae	Spain	Acosta et al. (1992)
	Amidostomum quasifulicae	Bulgaria	Petrova (1987)
Eurasian Coot (*Fulica atra*)	*Amidostomum acutum*	NR	Czapliński (1962)
	Amidostomum anseris	NR	Gower (1939)
	Amidostomum fulicae	California	Yamaguti (1961)
		Iraq	Mahmoud and Mohammad (1989)
		Europe	Borgsteede et al. (2006)
Spotted Crake (*Porzana porzana*)	*Amidostomum fulicae*	NR	Czapliński (1962)
Charadriiformes: Charadriidae			
Northern Lapwing (*Vanellus vanellus*)	*Amidostomum henryi*	Bulgaria	Petrova (1987)
Charadriiformes: Recurvirostridae			
Black-winged Stilt (*Himantopus himantopus*)	*Amidostomum acutum*	France	Czapliński (1962)
Pied Avocet (*Recurvirostra avosetta*)	*Amidostomum acutum*	The Netherlands	Borgsteede et al. (2006)
Pied Stilt (*Himantopus leucocephalus*)	*Amidostomum acutum*	Australia	Mawson (1980)
Charadriiformes: Scolopacidae			
Common Snipe (*Gallinago gallinago*)	*Amidostomum acutum*	Canada	Threlfall (1970)
		Taiwan	Schmidt and Kuntz (1972)
Wood Sandpiper (*Tringa glareola*)	*Amidostomum acutum*	NR	McDonald (1969)
Columbiformes: Columbidae			
Eurasian Collared-Dove (*Streptopelia decaocto*)	*Amidostomum anseris*	Czech Republic	Ryšavý et al. (1955)

Note: Some host–parasite reports found only in the review literature did not include geographic location and often included hosts from wild, domestic, and private and public zoological collections. NR, not reported.

* NWHC = USGS National Wildlife Health Center, Diagnostic Parasitology Laboratory, Madison, WI, USA; specimens are deposited at the Harold W. Manter Laboratory of Parasitology at the University of Nebraska State Museum, Lincoln, NE, USA; specimen voucher numbers are provided.

Table 20.2. Avian hosts infected with *Epomidiostomum* spp., geographic location where infection was reported, and reporting authors.

Host	Parasite	Location	Reference
Anseriformes: Anatidae			
American Black Duck (*Anas rubripes*)	*Epomidiostomum uncinatum*	Canada	Mahoney and Threlfall (1978)
American Wigeon (*Anas americana*)	*Epomidiostomum uncinatum*	North America	Shaw and Kocan (1980) and McLaughlin and McGurk (1987)
Baikal Teal (*Anas formosa*)	*Epomidiostomum uncinatum*	NR	McDonald (1969)
Bar-headed Goose (*Anser indicus*)	*Epomidiostomum skrjabini*[*]	India	Ali (1971b)
Barnacle Goose (*Branta leucopsis*)	*Epomidiostomum orispinum*	NR	McDonald (1969)
Taiga Bean-Goose (*Anser fabalis*)	*Epomidiostomum crami*	NR	McDonald (1969)
	Epomidiostomum orispinum	Europe	Macko et al. (2002)
	Epomidiostomum uncinatum	NR	McDonald (1969)
Black Scoter (*Melanitta nigra*)	*Epomidiostomum orispinum*	NR	Gower (1939)
	Epomidiostomum uncinatum	NR	Gower (1939)
Black-bellied Whistling-Duck (*Dendrocygna autumnalis*)	*Epomidiostomum uncinatum*	Texas, USA	Fedynich et al. (1996a)
Black-necked Swan (*Cygnus melancoryphus*)	*Epomidiostomum vogelsangi*[†]	Argentina	Oliveira (1970)
Blue-winged Teal (*Anas discors*)	*Epomidiostomum uncinatum*	North America	Turner and Threlfall (1975) and Shaw and Kocan (1980)
Brant (*Branta bernicla*)	*Epomidiostomum crami*[*]	New Jersey, USA	NWHC (voucher no. 48801)[‡]
Canada Goose (*Branta canadensis*)	*Epomidiostomum crami*	North America	Wetzel (1931) and Purvis et al. (1997)
	Epomidiostomum uncinatum	NR	McDonald (1969)
Canvasback (*Aythya valisineria*)	*Epomidiostomum uncinatum*	Canada	McLaughlin and McGurk (1987)
Cape Shoveler (*Anas smithii*)	*Epomidiostomum uncinatum*	Africa	Alexander and McLaughlin (1997)
Cape Teal (*Anas capensis*)	*Epomidiostomum uncinatum*	Africa	Alexander and McLaughlin (1997)
Cinnamon Teal (*Anas cyanoptera*)	*Epomidiostomum uncinatum*	NR	McDonald (1969)
Comb Duck (*Sarkidiornis melanotos*)	*Epomidiostomum asymmetricum*	India	Jairajpuri and Siddiqi (1970)
	Epomidiostomum sarkidiorni	India	Jairajpuri and Siddiqi (1970)
	Epomidiostomum sultanai	India	Ali (1971a)
	Epomidiostomum uncinatum	NR	McDonald (1969)
Common Eider (*Somateria mollissima*)	*Epomidiostomum orispinum*	Sweden	Persson et al. (1974)
Common Goldeneye (*Bucephala clangula*)	*Epomidiostomum crami*	Canada	Mahoney and Threlfall (1978)
	Epomidiostomum orispinum	NR	McDonald (1969)
Common Pochard (*Aythya ferina*)	*Epomidiostomum orispinum*	NR	Gower (1939)
	Epomidiostomum penelopi	Bulgaria	Petrova (1989)

	Epomidiostomum serratum	China	Shen (1981)
	Epomidiostomum uncinatum	NR	McDonald (1969)
Common Shelduck (*Tadorna tadorna*)	*Epomidiostomum crami*	NR	McDonald (1969)
	Epomidiostomum uncinatum	NR	McDonald (1969)
Emperor Goose (*Chen canagica*)	*Epomidiostomum crami*	Alaska, USA	NWHC (voucher no. 48804)
Eurasian Wigeon (*Anas penelope*)	*Epomidiostomum crami*	NR	McDonald (1969)
	Epomidiostomum orispinum	NR	Gower (1939)
	Epomidiostomum subquadratum	China	Shen and Wu (1973)
	Epomidiostomum uncinatum	Bulgaria	Petrova (1989)
Falcated Duck (*Anas falcata*)	*Epomidiostomum subquadratum*	China	Shen and Wu (1973)
	Epomidiostomum uncinatum	NR	McDonald (1969)
Ferruginous Pochard (*Aythya nyroca*)	*Epomidiostomum orispinum*	NR	Ali (1971b)
	Epomidiostomum sultanai	India	Ali (1971a)
	Epomidiostomum uncinatum	NR	McDonald (1969)
Fulvous Whistling-Duck (*Dendrocygna bicolor*)	*Epomidiostomum uncinatum*	India	Ali (1971a)
		Texas, USA	Fedynich et al. (1996a)
Gadwall (*Anas strepera*)	*Epomidiostomum subquadratum*	China	Shen and Wu (1973)
	Epomidiostomum uncinatum	North America	Buscher (1965)
Garganey (*Anas querquedula*)	*Epomidiostomum subquadratum*	China	Shen and Wu (1973)
	Epomidiostomum uncinatum	Bulgaria	Petrova (1989)
Gray Teal (*Anas gracilis*)	*Epomidiostomum uncinatum*	NR	McDonald (1969)
Greater Scaup (*Aythya marila*)	*Epomidiostomum uncinatum*	NR	McDonald (1969)
Greater White-fronted Goose (*Anser albifrons*)	*Epomidiostomum crami*	Texas, USA	Purvis et al. (1997)
	Epomidiostomum orispinum	Bulgaria	Petrova (1989)
	Epomidiostomum skrjabini	Russia	Yamaguti (1961)
	Epomidiostomum uncinatum	NR	Gower (1939)
Green-winged Teal (*Anas carolinensis*)	*Epomidiostomum orispinum*	NR	Gower (1939)
	Epomidiostomum querquedulae	Egypt	Yamaguti (1961)
	Epomidiostomum subquadratum	China	Shen and Wu (1973)
	Epomidiostomum uncinatum	Taiwan	Schmidt and Kuntz (1972)
		Canada	Turner and Threlfall (1975)
		Bulgaria	Petrova (1989)
Greylag Goose (*Anser anser*)	*Epomidiostomum crami*	NR	McDonald (1969)

(*continues*)

Table 20.2. (*Continued*)

Host	Parasite	Location	Reference
	Epomidiostomum orispinum	Bulgaria	Petrova (1989)
	Epomidiostomum skrjabini	Russia	Yamaguti (1961)
	Epomidiostomum uncinatum	NR	McDonald (1969)
Lesser White-fronted Goose (*Anser erythropus*)	*Epomidiostomum orispinum*	NR	McDonald (1969)
Long-tailed Duck (*Clangula hyemalis*)	*Epomidiostomum uncinatum*	NR	McDonald (1969)
Mallard (*Anas platyrhynchos*)	*Epomidiostomum crami*	NR	McDonald (1969)
	Epomidiostomum orispinum	Bulgaria	Petrova (1989)
	Epomidiostomum petalum§	China	Yen and Wu (1959)
	Epomidiostomum serratum	China	Shen (1981)
	Epomidiostomum uncinatum	North America	Crichton and Welch (1972)
		Europe	Birová et al. (1990)
Mandarin Duck (*Aix galericulata*)	*Epomidiostomum uncinatum*	NR	McDonald (1969)
Marbled Teal (*Marmaronetta angustirostris*)	*Epomidiostomum uncinatum*	NR	McDonald (1969)
Mottled Duck (*Anas fulvigula*)	*Epomidiostomum uncinatum*	Florida, USA	Kinsella and Forrester (1972)
		Texas, USA	Fedynich et al. (1996b)
Mute Swan (*Cygnus olor*)	*Epomidiostomum cygni*	China	Shen and Wu (1964)
	Epomidiostomum querquedulae	NR	McDonald (1969)
	Epomidiostomum uncinatum	NR	McDonald (1969)
Northern Pintail (*Anas acuta*)	*Epomidiostomum alii*	India	Ali (1971a)
	Epomidiostomum crami	NR	McDonald (1969)
	Epomidiostomum querquedulae	NR	McDonald (1969)
	Epomidiostomum uncinatum	North America	Buscher (1965)
		Bulgaria	Petrova (1989)
Northern Shoveler (*Anas clypeata*)	*Epomidiostomum serratum*	China	Shen (1981)
	Epomidiostomum uncinatum	North America	Buscher (1965)
Red-billed Duck (*Anas erythrorhyncha*)	*Epomidiostomum uncinatum*	Africa	Alexander and McLaughlin (1997)
Red-crested Pochard (*Netta rufina*)	*Epomidiostomum crami*	NR	McDonald (1969)
	Epomidiostomum querquedulae	India	Ali (1971a)
	Epomidiostomum subquadratum	China	Shen and Wu (1973)
	Epomidiostomum uncinatum	NR	McDonald (1969)
Redhead (*Aythya americana*)	*Epomidiostomum uncinatum*	Canada	McLaughlin and McGurk (1987)
Ring-necked Duck (*Aythya collaris*)	*Epomidiostomum crami*	Canada	Noseworthy and Threlfall (1978)

Ross' Goose (*Chen rossii*)	*Epomidiostomum crami*	Texas, USA	Fedynich et al. (2005)
Ruddy Duck (*Oxyura jamaicensis*)	*Epomidiostomum uncinatum*	Canada	McLaughlin and McGurk (1987)
Ruddy Shelduck (*Tadorna ferruginea*)	*Epomidiostomum querquedulae*	India	Ali (1971a)
	Epomidiostomum uncinatum	NR	McDonald (1969)
Snow Goose (*Chen caerulescens*)	*Epomidiostomum crami*	North America	Purvis et al. (1997) and Tuggle and Crites (1984)
Spot-billed Duck (*Anas poecilorhyncha*)	*Epomidiostomum petalum*	NR	McDonald (1969)
	Epomidiostomum serratum	China	Shen (1981)
	Epomidiostomum subquadratum	China	Shen and Wu (1973)
	Epomidiostomum uncinatum	NR	McDonald (1969)
Tufted Duck (*Aythya fuligula*)	*Epomidiostomum orispinum*	NR	Gower (1939)
	Epomidiostomum uncinatum	NR	McDonald (1969)
Tundra Swan (*Cygnus columbianus*)	*Epomidiostomum crami*	NR	McDonald (1969)
White-headed Duck (*Oxyura leucocephala*)	*Epomidiostomum uncinatum*	NR	McDonald (1969)
White-winged Scoter (*Melanitta fusca*)	*Epomidiostomum orispinum*	NR	McDonald (1969)
	Epomidiostomum uncinatum	Canada	Bourgeois and Threlfall (1982)
Whooper Swan (*Cygnus cygnus*)	*Epomidiostomum uncinatum*	NR	McDonald (1969)
Wood Duck (*Aix sponsa*)	*Epomidiostomum uncinatum*	North America	Thul et al. (1985)
Yellow-billed Duck (*Anas undulata*)	*Epomidiostomum uncinatum*	Africa	Alexander and McLaughlin (1997)
Gruiformes: Rallidae			
Eurasian Coot (*Fulica atra*)	*Epomidiostomum orispinum*	NR	Gower (1939)
Charadriiformes: Charadriidae			
Northern Lapwing (*Vanellus vanellus*)	*Epomidiostomum orispinum*	Bulgaria	Petrova (1989)

Note: Some host–parasite reports found only in the review literature did not include geographic location and often included hosts from wild, domestic, and private and public zoological collections. NR, not reported.

*Ali (1971b) redescribed *E. skrjabini*, which has been considered a synonym of *E. orispinum* (McDonald 1969), and regards *E. crami* to be a synonym of *E. skrjabini*.

†Some references (Yamaguti 1961; McDonald 1969) report *E. vogelsangi* as a synonym of *E. orispinum*; however, Ali (1971b) considers *E. vogelsangi* a valid species.

‡NWHC = USGS National Wildlife Health Center, Diagnostic Parasitology Laboratory, Madison, WI, USA; specimens are deposited at the Harold W. Manter Laboratory of Parasitology at the University of Nebraska State Museum, Lincoln, NE, USA; specimen voucher numbers are provided.

§Described from domestic Mallard (*A. platyrhynchos* var. *domestica*).

occurring on 4–5 continents (McDonald 1969; Tables 20.1 and 20.2). Other species such as *Amidostomum tribonyx*, *Epomidiostomum alii*, *Epomidiostomum asymmetricum*, *Epomidiostomum sarkidiorni*, *Epomidiostomum serratum*, and *Epomidiostomum sultanai* have been reported in relatively few hosts and seem to be restricted to specific geographic regions (Tables 20.1 and 20.2).

Other species of *Amidostomum* have been reported infrequently in six other taxonomic orders of birds (Table 20.1). *Amidostomum fulicae* is a parasite of the Rallidae in the order Gruiformes, which is found infrequently in anatids (Table 20.1).

Species of *Epomidiostomum* appear more restricted to the Anatidae, and have been reported in one host species each from the Gruiformes and Charadriiformes (Table 20.2). With the notable exception of certain dove, grouse, and ptarmigan species, hosts infected with species of *Amidostomum* and *Epomidiostomum* are typically associated with aquatic habitats (Tables 20.1 and 20.2).

There appears to be some association between the two genera of gizzard worms and taxonomic tribe within the Anatidae. For example, *A. anseris* and *E. crami* are principally found in Anserini, *A. acutum* and *E. uncinatum* occur mainly in Anatini and Aythyini, and *Amidostomum cygni* is found primarily in Cygnini (Czapliński 1962; McDonald 1969). However, some early authors did not distinguish between *A. acutum* and *A. anseris*, particularly in species of ducks, so the affinities these two species of nematodes may have for specific tribes is masked in the summarized scientific literature (Czapliński 1962; Table 20.1). It remains unclear whether associations are related to host specificity or other factors such as differences in host diet, feeding strategies, or specific life cycle requirements of the parasites. For example, successful experimental infections in hatchling chickens (Phasianidae) with *Amidostomum raillieti* (=*Amidostomum fulicae*, normally found in coots) and in hatchling chickens and pigeons (Columbidae) with *E. uncinatum* (primarily found in ducks) suggest a lack of host specificity (Leiby and Olsen 1965). However, laboratory experiments using *A. anseris* were unsuccessful in infecting swan, coot, avocet, godwit, gull, blackbird, crow, dove, chicken, and turkey (Ruff 1984 citing Enigk and Dey-Hazra 1968a). In another experiment, chickens were infected with larval stages of *A. anseris*, but the worms did not mature, suggesting that they are abnormal hosts (Phuc and Varga 1975).

ETIOLOGY

Species of *Amidostomum* and *Epomidiostomum* are classified in the phylum Nemata (Nematoda), class Secernentea, order Strongylida (bursate nematodes), and family Amidostomatidae (Anderson 2000). The subfamily Amidostomatinae contains the genus *Amidostomum*, and the subfamily Epomidiostomatinae contains the genus *Epomidiostomum* (Anderson 2000). In 1962, the genus *Amidostomum* was revised by Czapliński (1962) and a number of species entered into synonymy, resulting in six recognized species: *Amidostomum acutum*, *A. anseris*, *A. cygni*, *A. fulicae*, *Amidostomum henryi*, and *A. spatulatum*. Since then, three additional species—*Amidostomum auriculatum*, *Amidostomum ryzhikovi*, and *Amidostomum tribonix*—have been described (Mawson 1980; Kosupko and Lomakin 1985; Lomakin 1988) and one (*Amidostomum biziurae*) redescribed and taken out of synonymy with *A. acutum* (Mawson 1980).

Recently, there has been an effort to revise the classification of species within the subfamily Amidostomatinae. On the basis of morphological characteristics, Lomakin (1993) proposed a revision of the genera in which *Amidostomum* (after Czapliński 1962) is expanded into four genera—*Amidostomum* with three species: *Amidostomum anseris*, *A. cygni*, and *A. spatulatum*; *Mesamidostomum* with one species: *Mesamidostomum skrjabini*; *Amidostomoides* with six species: *Amidostomum acutum*, *A. auriculatum*, *A. henryi*, *A. monodon*, *A. petrovi*, and *A. tribonix*; and *Quasiamidostomum* with one species: *Q. fulicae*. However, this revised classification has not been readily adopted, indicating a need for genetic analyses to help delineate species within the Amidostomatinae (Borgsteede et al. 2006).

Depending on the taxonomic authority, there are at least 14 species in the genus *Epomidiostomum* (Table 20.2) including *E. alii*, *E. asymmetricum*, *E. crami*, *Epomidiostomum cygni*, *Epomidiostomum orispinum*, *Epomidiostomum penelopi*, *Epomidiostomum petalum*, *Epomidiostomum querquedulae*, *E. sarkidiorni*, *E. serratum*, *Epomidiostomum subquadratum*, *E. sultanai*, *E. uncinatum*, and *Epomidiostomum vogelsangi*. As with *Amidostomum*, some species have been synonymized, then redescribed and separated, thereby making it difficult to determine the true number of species within this genus based solely on morphological differences.

Adult *Amidostomum* spp. are typically found in the koilin lining of the gizzard, whereas *Epomidiostomum* spp. can also invade the gizzard muscle (Tuggle and Crites 1984). They often appear white to red and are relatively long and narrow in shape, having a thread-like appearance. In both genera, adult males are shorter and thinner than females. Depending on the species, the males of *Amidostomum* range in length from 5.9 to 18 mm, and females range from 6.5 to 27.7 mm (Czapliński 1962). Males of *Epomidiostomum* range in length from 5.3 to 11 mm, and females range from 7.4 to 19 mm (Cram 1927; Wetzel 1931; Jairajpuri and

Siddiqi 1970; Ali 1971a, b; Macko et al. 2002). Descriptions and figures of morphological features of each species can be found in taxonomic reviews (Czapliński 1962; Ali 1971b; Petrova 1987; Lomakin 1993) and original species descriptions or redescriptions (see Literature Cited section).

EPIZOOTIOLOGY

Life Cycle

Species of *Amidostomum* and *Epomidiostomum* have a direct life cycle. Partially developed eggs are released from the adult female worm into the gastric lumen of the infected host and excreted into the environment with the feces.

Survivability of eggs and larvae in the environment varies. Eggs and larvae require moist conditions for survival (Anderson 2000). The inability of eggs to withstand desiccation helps to explain the close association with the Anatidae.

Winter temperatures will inhibit larval development within the egg (Stradowski 1971) and prevent hatching (Cowan 1955). Eggs of *A. anseris* are able to withstand periods of freezing, but most larvae are killed (Stradowski 1975). Warmer temperatures of 18–23°C increase the rate of embryo development to third-stage larvae, with eggs hatching in 23–72 h (Cowan 1955; Leiby and Olsen 1965; McDonald 1969). In *A. anseris*, the rate of embryo development and hatching is asynchronous, which may be a survival strategy for geographic regions that have unpredictable late winter and early spring conditions (Stradowski 1975). After a short free-living stage of about 4–9 days, third-stage larvae become infective to susceptible hosts and can remain viable up to 20 days under suitable environmental conditions (Leiby and Olsen 1965; McDonald 1969). One study suggests that larvae of *A. anseris* are infective to domestic geese (*Anser anser* dom.) within as little as an hour after hatching (Stradowski 1971). Susceptible hosts become infected when they ingest the infective third-stage larvae during feeding or drinking. Additionally, larvae of *A. anseris* can penetrate the skin and migrate to the lungs and trachea prior to invading the gizzard lining (Anderson 2000 citing Enigk and Dey-Hazra 1968b, 1969, 1970). Larvae penetrate the thinner portions of the gizzard epithelium at the junction of the gizzard and proventriculus after ingestion (Leiby and Olsen 1965; Anderson 2000). Development to the fourth stage requires 2–4 days (Leiby and Olsen 1965), and approximately 12–25 days are required for worms to mature to adults (McDonald 1969).

Host age can influence the rate at which worms mature. For example, the prepatent phase of *A. anseris* in geese is shorter in goslings than in adults (Cowan 1955; Stradowski 1977).

Prevalence, Intensity, and Abundance[1]

Gizzard worms are some of the most commonly found species of helminths infecting waterfowl. Parasitological surveys examining more than 50 host individuals of the same species have found prevalances of infection with *Amidostomum* spp. greater than 50% (Herman and Wehr 1954; Persson et al. 1974; Bourgeois and Threlfall 1982; Thul et al. 1985; McLaughlin and McGurk 1987; Fedynich and Pence 1994; Nowicki et al. 1995). Additionally, some studies have reported prevalences of greater than 90%. For example, 96% of 94 Blue-winged Teal (*Anas discors*) were infected with *A. acutum* (Wallace and Pence 1986), 98% of 329 Canada Geese were infected with *A. anseris* (Herman and Wehr 1954), and 100% of 117 Common Eiders (*Somateria mollissima*) were infected with *A. acutum* (Borgsteede et al. 2006).

A few studies have found prevalence of infection with species of *Epomidiostomum* that exceeds 50% (Kinsella and Forrester 1972; Wallace and Pence 1986; McLaughlin and McGurk 1987), but it appears that *Amidostomum* spp. often occur at a higher prevalence. Additional research is needed to determine why *Amidostomum* spp. may be more prevalent than *Epomidiostomum* spp.

Numerous host intrinsic and environmental extrinsic factors are likely to play a role in determining prevalence, intensity, and abundance of gizzard nematodes. Differences in one or more of these factors have been found among waterfowl tribes. For example, in a study conducted at Delta, Manitoba, Canada, during autumn 1979, *E. uncinatum* was found in each of seven species of dabbling ducks (Anatini), whereas only three of six species of diving ducks (Aythyini) were infected (McLaughlin and McGurk 1987). Rapid development of infective stages in shallow wetlands, coupled with generalistic and intermediate foraging strategies of *Anas* spp., may account for these observed differences in prevalence (McLaughlin and McGurk 1987).

Within host species, the most commonly examined variables (and most easily measured) include host age (usually juvenile and adult), host sex, geographic location, and season of collection. Several studies have reported age-related effects. For example, prevalence, mean intensity, and abundance of *A. acutum* and *E. uncinatum* were higher in juvenile Mallards (*Anas platyrhynchos*) than in adults during autumn 1979 at Delta, Manitoba (McLaughlin and McGurk 1987). Rank abundance of *A. acutum* in Mallards from the

[1]Studies reporting prevalence, intensity, and abundance represent observations made at specific points in time and, thus, do not necessarily indicate present dynamics of hosts and parasites.

Southern High Plains was higher in juveniles than in adults during two consecutive winter and summer periods (Fedynich and Pence 1994). Rank abundance of *A. acutum* in Blue-winged Teal collected from the Texas panhandle did not vary by age, but *E. uncinatum* was higher in juveniles than in adults (Wallace and Pence 1986). Juvenile geese and swans often are more severely infected than adults (Herman and Wehr 1954; Pennycott 1998). Although age-dependent immunity is thought to influence infections of certain helminths (Wehr and Herman 1954), research is needed to elucidate the relationship, if any, between host age and the ability to suppress infections of gizzard nematodes.

Host sex, coupled with age, has been found to influence prevalence, mean intensity, and abundance of some gizzard nematodes. For example, abundance of *A. acutum* in adult male Lesser Scaup (*Aythya affinis*) was higher than that found in adult females (McLaughlin and McGurk 1987). Mean intensity of *E. uncinatum* was higher in adult female and juvenile female American Wigeon (*Anas americana*) than in adult males and juvenile males (McLaughlin and McGurk 1987). It is uncertain why differences in prevalence or intensity of gizzard worms is associated by host sex in some species and not others. There is some speculation that acquisition of helminth species may be influenced by variation in habitat use, foraging time, and/or diet (Fedynich et al. 2005), which permits different exposure probabilities to infective parasitic stages.

Seasonal variation in prevalence, mean intensity, and abundance has been reported for *Amidostomum* spp. and *Epomidiostomum* spp., and could be attributed to a wide range of factors such as seasonal changes in diet or differences in exposure rates among breeding, migrating, and wintering birds. For example, prevalence of *A. acutum* in wild Mallards was lowest in January (5%) and highest in June (43%), whereas prevalence of *E. uncinatum* was lowest in September (19%) and highest in May (58%) (Birová et al. 1990). Abundance of *E. uncinatum* was lower in Blue-winged Teal collected in the fall than in those collected during spring (Wallace and Pence 1986). Prevalence and intensity of *A. anseris* and *E. crami* were similar between the incubation and brood-rearing periods in adult female Snow Geese at La Pérouse Bay, Manitoba, whereas prevalence and intensity of *A. spatulatum* were higher during the incubation period and declined during the brood-rearing period (Clinchy and Barker 1994). Findings in Snow Geese suggest a geographic and seasonal component to infections where transmission of *E. crami* was occurring throughout summer on the breeding grounds and *A. spatulatum* was being acquired primarily on the wintering grounds (Clinchy and Barker 1994).

CLINICAL SIGNS

Mild infections are usually accompanied by no clinical signs. Signs of severe infections in wild birds are nonspecific, and include unthriftiness, loss of appetite, anemia, emaciation, and general weakness (Herman and Wehr 1954; MacNeill 1970; Tuggle and Friend 1999). Trumpeter Swans (*Cygnus buccinator*) infected with *A. anseris* exhibit disorientation, staggered gait, and impaction of the esophagus (MacNeill 1970). In Canada Geese, the intensity of infection with *A. anseris* was negatively correlated with body condition (Herman and Wehr 1954).

PATHOGENESIS

Tissue injury by *Amidostomum* spp. is caused primarily by mechanical disruption of the gizzard and to a lesser extent by the host inflammatory response. The pathogenesis of infections with species of *Epomidiostomum* is probably similar but has not been reported. Third-stage larvae of *Amidostomum* spp. penetrate the soft area of the mucosa near the junction of the proventriculus or intestine with the gizzard. They develop to fourth-stage larvae with little migration within or beneath the mucosa (Leiby and Olsen 1965). Mature worms migrate and feed on blood in the gizzard mucosa causing hemorrhage, leakage of plasma proteins, and potential ischemia resulting in erosions and ulcers (Bunyea and Creech 1926; Enigk et al. 1969). Parasite activity and hemorrhage in the mucosa beneath the koilin lining and within the koilin disrupt the attachment between the glandular mucosa and koilin, leading to cracks and loss of portions of the gizzard pads. In severe infections, the result may be impaired gizzard function and nutritional deprivation (Bunyea and Creech 1926). In times of nutritional stress, severe infections with *Amidostomum* spp. may substantially contribute to fatal debilitation (Herman and Wehr 1954; Borgsteede 2005). It has been proposed that the parasite releases toxins that contribute to localized formation of ulcers; to generalized signs of depression, emaciation, dyspnea, and dysphagia; and to inflammatory cell reactions in organs remote from the parasite (Bunyea and Creech 1926; Vetési et al. 1976), but this has not been verified by experimental studies.

PATHOLOGY

Birds with severe ventricular nematodiasis may be thin to emaciated. Gross lesions associated with ventricular nematodiasis are present in the gizzard, and, even in mild infections, red to brown discoloration may be found at the margins of the koilin lining, between the koilin pads, and in the soft mucosa close to the

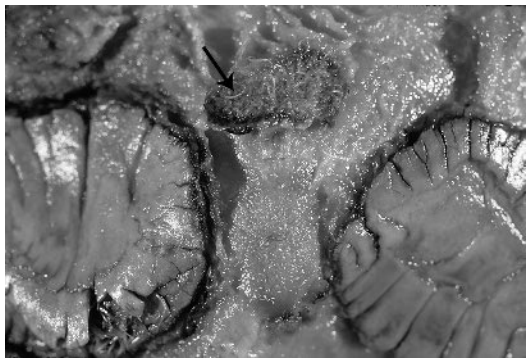


Figure 20.1. Infection with *Amidostomum* sp. in the gizzard of a Canada Goose (*Branta canadensis*). Hemorrhage is present at the margins of the koilin pads, which are cracking and separating from the mucosa. Thin nematodes lie atop focal mucosal hemorrhage between the pads (arrow). (Photograph by J. Runningen, US Geological Survey, National Wildlife Health Center files.)

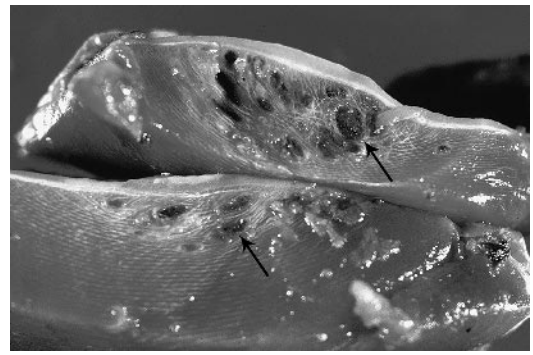


Figure 20.2. Species of *Epomidiostomum* may invade the gizzard muscle beneath the koilin pads and form granulomatous tracts (arrows), as in this Snow Goose (*Chen caerulescens*). (Photograph by J. Runningen, US Geological Survey, National Wildlife Health Center files.)

duodenal orifice (Tuggle and Crites 1984). The small threadlike nematodes are buried in the discolored mucosa and/or beneath the koilin.

Multiple genera and species of gizzard worms can occur within a single individual bird, and several species may be found occupying different locations in the gizzard (Tuggle and Crites 1984). In Snow Geese, *A. anseris* was found close to the duodenal orifice and at the periphery of the koilin; *A. spatulatum* was found in similar locations but also beneath the koilin; and *E. crami* was found beneath the koilin, partially within the mucosa, and in the gizzard muscle (Tuggle and Crites 1984).

In severe gizzard worm infections, the koilin lining is undermined by parasite activity and hemorrhage so that it develops cracked margins, becomes friable, erodes, and separates from the glandular mucosa (Figure 20.1). In severe infections with *Epomidiostomum*, tracts containing the parasite and its eggs can also be found in the underlying muscle (Figure 20.2) (Tuggle and Crites 1984). Severe lesions were found in Canada Geese with more than 150 *A. anseris*, in Snow Geese with more than 30 *A. anseris* and/or *A. spatulatum*, and in Snow Geese with more than 25 *E. crami* (Herman and Wehr 1954; Tuggle and Crites 1984). Gross lesions are similar in various species of waterfowl, but in Tundra Swans (*Cygnus columbianus*) and Trumpeter Swans, severe infections with *Amidostomum* spp. may be associated with esophageal impaction (McKelvey and MacNeill 1981). Lesions in other avian species have not been described. Reports of *Amidostomum* and *Epomidiostomum* in avian families other than Anatidae generally have focused on morphological descriptions of the parasites rather than the effects on the hosts.

In microscopic sections of gizzard, *Amidostomum* spp. are commonly visible between the mucosa and the koilin lining, within the deep layers of the koilin, and in the lumina of mucosal glands (Bunyea and Creech 1926; Vetési et al. 1976). With time, parasites may spread throughout the koilin layer and become surrounded by eggs (including embryonated eggs). Sections of the parasites are found less frequently in the mucosal propria (although the parasites must feed there), suggesting that the majority of the parasite body resides in more superficial layers. Mucosal glands containing parasites may be dilated and the epithelial cells flattened. The koilin often is separated from the mucosa and fragmented, and mucosal erosions may be present. Hemorrhage fills clefts between the mucosa and koilin lining, within the koilin, and the mucosal propria. Early inflammation consists of diffuse eosinophils in the propria, and later these are mixed with lymphocytes and macrophages (Vetési et al. 1976). Lymphoid hyperplasia progresses with time and consists of perivascular infiltrates of lymphocytes and histiocytes as well as lymphoid follicles in the deep mucosal propria; plasma cells are not a feature (Vetési et al. 1976). Fibroblasts and fibrosis also increase with time. Similar inflammatory lesions may occur in the proventriculus where larvae may have initially invaded. In infections with *Epomidiostomum* spp., granulomatous inflammation and granulomas form around the parasites in the ventricular muscularis in addition to presence of parasites and inflammation in the mucosa (Tuggle and Crites

1984). The parasites in muscle are surrounded by reactive macrophages, hemorrhage, multinucleated giant cells, fibroblasts, and fibrosis; fibrosis may be severe in gizzard muscle (Tuggle and Crites 1984).

DIAGNOSIS

The diagnosis of ventricular nematodiasis is made by observation of worms in the koilin lining and gizzard muscle or eggs in the feces. Eggs of both genera have similar features and may require trained personnel for identification to genus (Tuggle and Friend 1999). Identification to species requires examination of adult male and female worms and comparison of morphological characteristics found in established keys and species descriptions. A diagnosis of morbidity or mortality should be based on the severity of lesions and complicating factors, as well as elimination of other causes. For example, thickening and irregularity of gizzard pads as well as upper gastrointestinal impaction can be present in lead-poisoned waterfowl.

IMMUNITY

In studies of *A. anseris* in domestic geese, precipitating antibodies were not detected in infected goslings (Vetési et al. 1976 citing Bausov 1969), and goslings as well as older geese were susceptible to reinfection after initial infection with intact or irradiated larvae (Phuc and Varga 1973; Stradowski 1977 citing Georgiev 1963). In the absence of reinfection, there is evidence that *A. anseris* has a limited life span in the host, although the mechanism responsible for loss of worms and involvement of host immunity in the process is unknown (Stradowski 1977).

Age-related susceptibility has been documented experimentally in domestic geese (Phuc and Varga 1973; Stradowski 1977) and observed in wild geese (Herman and Wehr 1954; Nowicki et al. 1995). The mechanism is uncertain but may be linked to innate or acquired immunity. After experimental inoculations in domestic geese, the development of *A. anseris* was correlated with the age of the bird, in which the parasites matured more rapidly and had a longer life span in younger geese (Phuc and Varga 1973; Stradowski 1977). The greatest intensity of infection with *A. anseris* in wild Canada Geese in North Carolina, USA, occurred in younger geese, and young geese were predominantly involved in winter mortality to which *Amidostomum* was considered to be a contributor (Herman and Wehr 1954). In a later study in Illinois, USA, immature Canada Geese were found to have greater intensity of gizzard worm infections, greater prevalence of *A. anseris* infection, and a greater proportion of mature *A. anseris* than adult geese (Nowicki et al. 1995).

There is evidence that *A. anseris* infection may interfere with development of an immune response to other pathogens. The mechanism is unknown but could be related to stress, nutritional compromise, or protein loss. Antibody production in response to vaccination was compared in domestic geese with and without subclinical gastrointestinal parasite infections (specifically a combination of *A. anseris*, *Capillaria anatis*, and *Heterakis gallinarum*) in which *A. anseris* was most prevalent (Ziomko et al. 1998). Antibody production was lower and significantly delayed in parasitized geese (Ziomko et al. 1998).

PUBLIC HEALTH CONCERNS

There is no evidence that species of *Amidostomum* and *Epomidiostomum* present human health concerns. For human consumption, however, it is recommended that gizzards are thoroughly cooked or discarded if nematode-damaged tissues are apparent because of possible secondary bacterial infections (Tuggle and Friend 1999).

DOMESTIC ANIMAL HEALTH CONCERNS

Ventricular nematodiasis in domestic anatids has long been recognized throughout the world (Cram 1925; Gower 1939; Levine 1968; McDonald 1969). Instances of heavy losses and severe pathology have been reported among geese infected with *A. anseris* (Ruff 1984). This species is a concern in commercially raised ducks, geese, and pigeons (Ruff 1984; Permin and Hansen 1998). Chickens experimentally inoculated with *A. anseris* experienced some hemorrhage and destruction of the koilin, demonstrated acute or subacute inflammation of the mucosa, and had lymphoid hyperplasia, but the worms were subsequently eliminated by this host within 15 days postinoculation (Vetési et al. 1976). Additionally, *Amidostomum skrjabini* (=*A. acutum*) has produced pathology in young ducks (Ruff 1984). Other species of *Amidostomum* and species of *Epomidiostomum* are not reported or emphasized as species of concern for poultry (Ruff 1984; Permin and Hansen 1998; Merck Veterinary Manual 2006).

There is potential for transmission of gizzard worms between domestic and wild birds within the Anatidae. Species reported in domestic ducks and geese include *A. acutum*, *A. anseris*, and *E. uncinatum* (McDonald 1969). Consequently, unenclosed zoological gardens, open-air commercial farms, and backyard flocks may be at risk from wild anatids infected with these species of gizzard nematodes.

WILDLIFE POPULATION IMPACTS AND MANAGEMENT IMPLICATIONS

Although numerous studies have demonstrated pathology in individual birds resulting from ventricular nematodiasis (Jerstad 1937; Herman and Wehr 1954; MacNeill 1970; Crichton and Welch 1972; Turner and Threlfall 1975; Harradine 1982; Tuggle and Crites 1984), there presently is no evidence that robust wild populations of anatids are negatively impacted. However, mortality in Canada Goose goslings has been associated with ventricular nematodiasis (Wickware 1941; Herman and Wehr 1954). This could affect recruitment and possibly have a negative population effect if direct or indirect mortalities resulting from infections are additive. Additionally, this may be a problem if Canada Geese are being managed at the subspecies level, particularly if they are differentially impacted by gizzard worms because of host geographic-isolating mechanisms that permit differences in exposure probabilities among host subspecies.

Concerns about introducing diseases and parasites have been raised in regard to releasing captive-raised infected birds to supplement or reestablish wild populations of threatened or endangered species (Bailey and Black 1995). However, few specific examples are available regarding negative effects on small host populations caused by species of *Amidostomum* and *Epomidiostomum*, and further study seems warranted.

Infections are relatively uncommon in host families not associated with wetland habitats. However, the report of Eurasian Collared-Doves (*Streptopelia decaocto*) becoming infected with *A. anseris* at a goose farm (Ryšavý et al. 1955) suggests potential routes of entry into wild bird populations in situations where they are exposed to high concentrations of infective larvae originating from domestic anatids. Additional research is needed to determine whether local population impacts occur in these alternate hosts.

TREATMENT AND CONTROL

No methods have been developed for treatment and control of species of *Amidostomum* and *Epomidiostomum* in wild host populations, presumably because there is little evidence that ventricular nematodiasis is an important cause of epizootic die-offs.

Treatment and control of ventricular nematodiasis are appropriate for captive flocks of wild species, threatened or endangered species, and where wild and domesticated waterfowl intermingle in commercial farming operations. Where applicable, providing larger feeding areas for captive herbivorous grazing species such as the Hawaiian Goose (*Branta sandvicensis*) may decrease infections of density-dependent disease agents (Bailey and Black 1995), which would be applicable to gizzard worms. Other strategies used for domestic fowl may be applicable to captive wild anatids, including raising young and old birds separately to prevent exposure of uninfected young individuals to infected older individuals and maintaining good sanitation practices (Levine 1968). Where possible, efforts should be made to prevent access of wild anatids to captive or domesticated flocks, particularly if anthelmintics are being used, because reinfection can rapidly occur following treatment.

Anthelmintics have been used effectively on infected captive wild anatids and commercial flocks, but in these instances, treatments were focused on eliminating *A. anseris* because of its economic importance to commercial enterprises. Specific anthelmintics used to kill *A. anseris* include flubendazole (Vanparijs 1984), mebendazole (Ruff 1984 citing Enigk et al. 1973; Bailey et al. 1990), cintrin (Merck Veterinary Manual 2006), cambendazole (Ruff 1984 citing Enigk and Dey-Hazra 1971), pyrantel (Ruff 1984), and a combination of neguvon, atropine sulfate, and piperazine sulfate (Georgiev 1968).

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21
Tetrameridosis

John M. Kinsella and Donald J. Forrester

INTRODUCTION

The term tetrameridosis includes diseases caused by species of nematodes belonging to three genera of the family Tetrameridae: *Tetrameres*, *Microtetrameres*, and *Geopetitia*. Females of *Tetrameres* and *Microtetrameres* are typically found embedded in the gastric glands of the proventriculus. One or more of the much smaller males may be either associated with the females in the glands or free in the lumen of the proventriculus. A few species of *Tetrameres* (e.g., *Tetrameres strigiphila*) are found encysted in a fibrous capsule in the tunica muscularis of the proventriculus, with the female tail extending through an opening into the lumen of the proventriculus for the deposition of eggs. Species of *Geopetitia* occupy similar cysts in the wall of the esophagus, proventriculus, or the gizzard, but often have multiple females per cyst rather than one (Anderson 2000).

Infections of high intensity with species of the genus *Tetrameres* have led to emaciation, anemia, and death in domestic ducks and chickens. There is some evidence (Ellis 1970) that the damage done to the avian proventriculus by *Microtetrameres* spp. is similar to that of *Tetrameres*. One species of *Geopetitia* has caused widespread morbidity and mortality in zoo birds, but no similar reports are available for wild populations.

HISTORY

The first species of *Tetrameres* was described by Diesing (1835) from raptors in Brazil. The extreme sexual dimorphism led to much early confusion and, in one case, the globular female was thought to be a trematode, not a nematode. It was some time before the males of these species were recognized and associated with the females. Even today it is not always possible to pair males and females with assurance, since mixed infections of different species are not uncommon. This confusion has led to some problems with taxonomy, many of which are still not resolved.

The first species of *Microtetrameres* was described by Travassos (1914) from a Yellow-fronted Woodpecker (*Melanerpes flavifrons*) in Brazil, and the first species of *Geopetitia* from a Coal Tit (*Periparus ater*) in France (Chabaud 1951).

The earliest description of lesions caused by a species of *Tetrameres* appears to be that of Rust (1908) in Germany. Ellis (1970) published the first account of the pathogenicity of a species of *Microtetrameres*. It was not until comparatively recently that the first reports on disease due to *Geopetitia* have appeared (Bartlett et al. 1984; Tscherner et al. 1997).

HOST RANGE AND DISTRIBUTION

The distribution of species of *Tetrameres* is cosmopolitan. The majority of the species parasitize aquatic birds, especially Anseriformes, Ardeiformes, Gruiformes, and Charadriiformes, although some species are found in land birds such as Passeriformes and occasionally Galliformes.

In contrast to species of *Tetrameres*, species of *Microtetrameres* usually parasitize terrestrial birds, with the vast majority of species found in Passeriformes, Accipitriformes, and Strigiformes. The exceptions prove the rule in this case, since two of the species found in Ardeiformes, *Microtetrameres spiralis* and *Microtetrameres egretes*, parasitize the Cattle Egret (*Bubulcus ibis*), which feeds principally on land. The distribution of species of *Microtetrameres* is also cosmopolitan.

Eight species of *Geopetitia* are known; seven occur in wild Falconiformes, Cuculiformes, Coraciiformes, Piciformes, or Passeriformes in France, the former Soviet Union, Taiwan, Australia, India, Madagascar, Ghana, Congo, and Cuba. The natural host of the eighth species, *Geopetitia aspiculata*, remains unknown. It was originally described from the "purple sugarbird" (*Coerulea coerulea)* at the National Zoological Park, Washington, DC, USA. (Webster 1971). Unfortunately, purple sugarbird is not a recognized common

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name, and *Coerulea* is not listed among world bird species either as a genus or as a species (Clements 2000), so the type host of *G. aspiculata* remains a mystery. A good possibility is the Purple Honeycreeper (*Cyanerpes caeruleus*), since honeycreepers have sometimes been called sugarbirds. It has since been recorded from a variety of Passeriformes, Coraciiformes, and Charadriiformes at the Assiniboine Park Zoo, Winnipeg, Canada (Bartlett et al. 1984), the Lincoln Park Zoological Gardens, Chicago, Illinois, USA (French et al. 1994), and four different zoos in Austria and Germany (Tscherner et al. 1997; Juncker-Voss et al. 2001).

ETIOLOGY

Tetramerid nematodes are characterized by marked sexual dimorphism. Males have a typical nematode filiform appearance, but females are swollen by the greatly distended uterus and may be globular (*Tetrameres* spp.) (Figure 21.1a), spirally coiled (*Microtetrameres* spp.) (Figure 21.1b), or have the posterior extremity more slender than the remainder of the body, with the distal end inflated (*Geopetitia* spp.) (Figure 21.1c) (Yamaguti 1961).

The taxonomy of the genus *Tetrameres* remains in a state of confusion, and it is difficult to estimate the number of valid species described since Yamaguti (1961) listed a total of 46. In an unpublished doctoral dissertation, Mollhagen (1976) provided an extensive host list and proposed 5 new species and a number of synonymies. However, much of his dissertation remains unpublished in peer-reviewed journals and, as a result, his conclusions are not recognized as valid under the rules of the International Code of Zoological Nomenclature. Nevertheless, his dissertation is an invaluable resource for identification of species of *Tetrameres*, and the extensive bibliography covering the years 1835–1985 has been published separately (Mollhagen 1991).

Although some authors (e.g., Chabaud 1975) have considered *Microtetrameres* to be a subgenus of *Tetrameres*, Anderson (2000) believed that differences in larval and adult morphology justified its retention as a separate genus. Ellis (1971) listed host and geographical distribution for 35 species of *Microtetrameres* and gave measurements for those species described from the Western Hemisphere. Mawson (1977) described 15 new species of *Microtetrameres* from Australian birds and presented a key to the males of 52 species. Seven additional species have been described since 1977, bringing the total known species to 59.

There is currently no review of the taxonomy of the genus *Geopetitia* available. Webster (1971) listed eight species, but considered three of these doubtful because they had been described from females only. In light of the notable lack of host specificity of *G. aspiculata*, the validity of existing species needs to be reexamined.

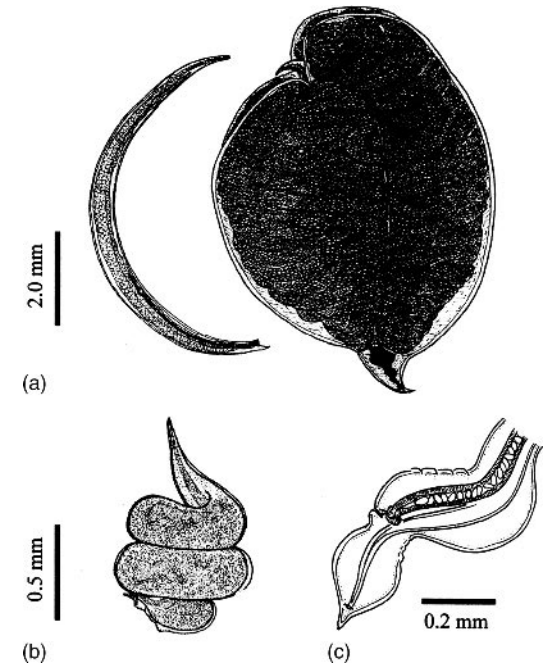


Figure 21.1. Line drawings of representatives of the three genera of Tetrameridae. (a) Adult male (left) and female (right) of *Tetrameres strigiphila* from a Barred Owl (*Strix varia*). Courtesy of D. B. Pence and the *Journal of Parasitology*. (b) Adult female of *Microtetrameres aquila* from a golden eagle (*Aquila chrysaetos*). Courtesy of S. C. Schell and *Transactions of the American Microscopical Society*. (c) Posterior extremity of an adult female *Geopetitia aspiculata* from a Purple Sugarbird (*Coerulea coerulea*). Courtesy of W. A. Webster and the *Proceedings of the Helminthological Society of Washington*.

EPIZOOTIOLOGY

Life History

Information on life cycles of seven species of *Tetrameres* was summarized by Anderson (2000). The majority of species of this genus parasitize aquatic

birds such as anseriforms and ciconiiforms and intermediate hosts are usually crustaceans. A smaller number of species parasitize terrestrial birds, especially passeriforms and galliforms, and intermediate hosts are normally insects, most commonly orthopterans and coleopterans. Kovalenko (1960) implicated fish as paratenic hosts of *Tetrameres fissispina*, but the role of carrier hosts in the genus remains largely undefined. The following are examples of typical aquatic and terrestrial life cycles.

Larvae of *T. fissispina*, a common parasite in anseriforms, reach the third or infective stage in the intermediate host, *Gammarus lacustris*, in 8–18 days (Garkavi 1949). In ducklings, males and females occur together in glands at 10 days postinfection, but by 18 days only females are found, indicating that insemination of the females occurs early in infections, followed by death of the males (Cvetaeva 1960).

The intermediate host of *Tetrameres cardinalis*, a parasite of the Northern Cardinal (*Cardinalis cardinalis*), was determined to be an orthopteran, *Locusta migratoria*. Infective third-stage larvae are encapsulated in the fat body at 11 days (Quentin and Barre 1976), and when fed to a cardinal developed to the fourth stage by 11 days postinfection.

The life history of *Microtetrameres corax*, a parasite of the Black-billed Magpie (*Pica hudsonia*) in Colorado, was described by Bethel (1973). Eggs were fed to grasshoppers (*Melanopus* spp.), and third-stage larvae were found in the thoracic region of the hemocoel and among the fat bodies of the abdominal cavity as early as 27 days postinfection. Grasshoppers were fed to laboratory-born magpies and adult males and females of *M. corax* were recovered 48 days later.

Quentin et al. (1986) infected *Tylotropidus patagiatus* and *Locusta migratoria* (Orthoptera: Acrididae) with eggs of *Microtetrameres inermis* from Orange Weavers (*Ploceus aurantius*). Third-stage larvae were found free in the hemocoel 18 days later.

The life cycle of *G. aspiculata* has been studied only in zoos and the natural definitive host remains unknown. Bartlett et al. (1984) placed gravid female worms on apple peels and fed them to crickets (*Acheta domestica).* Infective third-stage larvae were found by 48 days, and when administered orally to a Cut-throat (*Amadina fasciata*), two adults were found in a cyst on the proventricular serosa 35 days later. French et al. (1994) infected crickets (*Acheta domestica*) and cockroaches (*Blattella germanica* and *Supella supellectilium*) with eggs from *G. aspiculata*, and obtained infective larvae at 35 days. Larvae were found at the junction of the proventriculus and the ventriculus 24–48 h postinfection in Zebra Finches (*Taeniopygia guttata*), and raised nipplelike nodules on the serosal surface of the proventriculus were observed at 2 weeks. A mass of mature worms was found in a proventricular nodule at 14 weeks.

Prevalence and Intensity

As with many nematode infections, prevalences and intensities of *Tetrameres* spp. tend to be higher in confined domestic birds than in wild populations. For example, Czaplinski (1962) reported a 46% prevalence of *T. fissispina* in domestic Mallards (*Anas platyrhynchos*) in Poland, but only a 27% prevalence in wild Mallards. In East Slovakia, *T. fissispina* occurred at higher prevalences in wild mallards, but higher intensities in domestic Mallards (Birova et al. 1990). Kinsella (1973) found 66% of American Coots (*Fulica americana*) infected with *Tetrameres globosa* in Florida, while only 25% of Purple Gallinules (*Porphyrio martinica*) and 6% of Common Moorhens (*Gallinula chloropus*) from the same area were infected with the same species (Kinsella et al. 1973), presumably reflecting differences in feeding preferences for intermediate hosts. Mollhagen (1976) states that intensities of infections of *Tetrameres* spp. in wild birds usually average fewer than ten worms per bird, although intensities in the hundreds have been reported occasionally.

Only a few studies of seasonal and long-term trends in *Tetrameres* have been done in wild birds. In a study of Northern Bobwhites (*Colinus virginianus*) in 1968–1969 in northern Florida, Davidson et al. (1980) found more than 70% of juveniles and adult birds infected with *Tetrameres pattersoni*, with intensities as high as 65 per bird. A summer peak of relative abundance was found, coinciding with an abundance of arthropod intermediate hosts. But in a 1983–1984 survey of the same population, Moore et al. (1986) found only 5% of adults and no juveniles infected with *T. pattersoni*, possibly due to the decline of these intermediate hosts. In a 5-year study of Northern Bobwhites in southern Florida, Forrester et al. (1984) reported *T. pattersoni* to be totally absent in 1 year.

From 1976 to 1983, 25 birds of 14 species (Passeriformes: 12, Coraciiformes: 1, Charadriiformes: 1) that died at the Assiniboine Park Zoo in Winnipeg were found infected with *G. aspiculata*. Intensities could not be exactly determined but were estimated to range from 6 to 50 (Bartlett et al. 1984). At the Lincoln Park Zoological Gardens in Chicago, 96 birds of 25 species (Passeriformes: 19, Piciformes: 5, Ciconiiformes: 1), and at three zoos in Germany, 77 birds of 41 species (Passeriformes: 32, Piciformes: 5, Columbiformes: 2, Coraciiformes: 1, Charadriiformes: 1) were infected with *G. aspiculata* (French et al. 1994; Tscherner et al. 1997) but prevalences and intensities were not recorded. Between 1993 and 1998, *G. aspiculata* was reported as the cause of death of

44 birds at the Viennese Zoo aviary (Juncker-Voss et al. 2001).

CLINICAL SIGNS

Clinical signs of infections with *Tetrameres* spp. in pigeons, ducks, and chickens include weakness, loss of appetite, diarrhea, and emaciation with atrophy of the thoracic musculature (Wehr 1971; Endo and Inoue 1989).

Fink et al. (2004) infected three groups of chickens with doses of 25, 100, and 400 larvae of *Tetrameres americana*, respectively, and found that infected chickens exhibited no difference in weight gain compared to controls. Higher doses influenced the establishment rate and intensity of infection, but not worm size, or host mortality.

There is virtually no information on clinical signs of disease in wild birds due to species of the genus *Microtetrameres*. Bethel (1973) infected two laboratory-born Black-billed Magpies with 110 and 74 females of *M. corax* and found experimental birds less aggressive and less active than controls at 35 days postinfection.

Many zoo birds infected with *G. aspiculata* showed no specific signs other than sudden death. Others had a history of weight loss and abdominal distension (Bartlett et al. 1984; French et al. 1994).

PATHOLOGY

Clinical Pathology

Hematological changes were studied in 12 chicks experimentally infected with *Tetrameres mohtedai* by Ramaswamy and Sundaram (1983). A marked eosinophilia was found during the early stages of infection when juveniles migrated through the wall of the proventriculus, followed by heterophilia and lymphocytopenia when the nematodes developed in the proventricular glands. A normocytic and normochromic anemia in early stages of development progressed to a macrocytic, hypochromic anemia as the worms matured.

We know of no information on clinical pathology of birds infected with species of *Microtetrameres* or *Geopetitia*.

Gross Pathology

Infections of high intensity with *Tetrameres* spp. are characterized by enlargement of the proventriculus, with thickening of the wall. The females within the glands can be seen through the wall as dark red, slightly raised foci 2–3 mm in diameter, resembling hematomas. The infected proventricular glands may be covered with a foamy, necrotic material. The small intestinal mucosa may be swollen and covered with mucus and the contents greenish due to the presence of bile, or granular and golden-brown due to the presence of red blood cells (Popova 1954).

No information is available on gross pathology due to *Microtetrameres* spp.

Cysts of *G. aspiculata* range in size from small plaques on the serosal surface of the esophagus, proventriculus, or gizzard to large cysts containing up to 50 worms and filling the whole abdominal cavity. Adhesions may form between the cysts and the serosa of the liver or spleen causing inflammation and fibrosis (Bartlett et al. 1984; French et al. 1994). Younger cysts are delicate and membranous and the worms can be seen through the wall while older cysts become hard, friable masses.

Histopathology

Bergan et al. (1994) described lesions caused by *Tetrameres striata* in Mallards. In most cases, the gravid females caused pressure atrophy and necrosis of the proventricular gland mucosa, with complete loss of acini, but little or no inflammatory response around the parasites or in the compacted mucosa or submucosa (Figure 21.2). Occasional lesions were noted in the submucosa surrounded by a thin layer of fibrous material forming a cyst, with or without adjacent inflammatory cells. A similar pattern of pressure atrophy and loss of acini was reported by Mascarenhas and Ghosh (1992) in proventricular lesions caused by *T. mohtedai* in fowl, but gravid females were also found deep within the muscular layer of the gizzard. These females were found to cause atrophy of the adjacent muscle fibers and severe cellular reaction characterized by infiltration of lymphocytes, plasma cells, eosinophils, and macrophages, and proliferation of fibrovascular granulation tissue.

Ellis (1970) conducted an in situ study of sections of adult female *Microtetrameres centuri* in Eastern and Western Meadowlarks (*Sturnella magna* and *Sturnella neglecta*) and demonstrated nucleated red blood cells within the lumen of the intestine, indicating this species to be hematophagous. Mechanical growth of the females within the proventricular glands caused dilatation of the gland lobule with mild pressure atrophy of the epithelium. Involved gland lobules were nonfunctional and mild hyperemia of the lamina propria was noted. There was no marked inflammatory response, but increased mucus secretion and a mild proventriculitis were observed. Bethel (1973) noted a similar pattern in natural infections of *M. corax* in Black-billed Magpies, with pressure atrophy of the glandular tissue around the worms but no connective tissue or cellular response. Loss of secretory function in infected glands

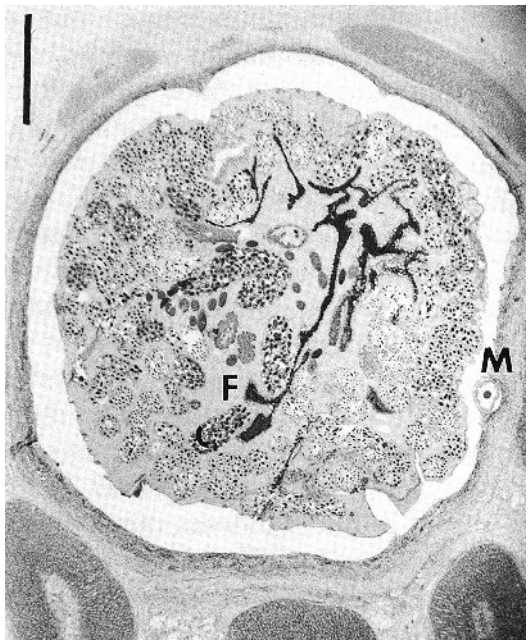


Figure 21.2. Section of a proventriculus of a Mallard (*Anas platyrhynchos*) infected with an adult male (M) and female (F) *Tetrameres striata*. Note the lack of an inflammatory response. Bar = 500 μm. Courtesy of D. B. Pence and the *Journal of Wildlife Diseases*.

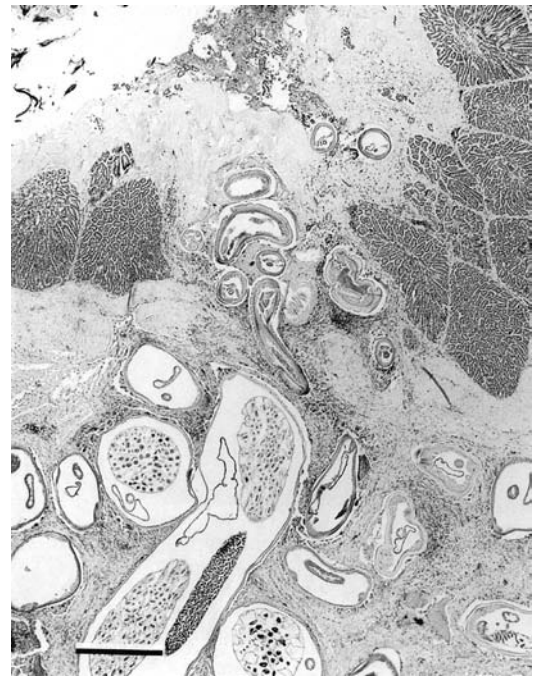


Figure 21.3. Section of the proventriculus of a White-lined Tanager (*Tachyphonus rufus*) showing a specimen of *Geopetitia aspiculata* protruding from a cyst through the proventricular wall. Bar = 500 μm. Courtesy of C. M. Bartlett and the *Journal of Wildlife Diseases*.

was also reported in *Microtetrameres nestoris* infections of the New Zealand Kaka (*Nestor meridionalis*) (Clark et al. 1979).

Within the cyst, adults of *G. aspiculata* are surrounded by fibrinous exudate or granulation tissue containing scattered fibroblasts, giant cells, heterophils, and lymphocytes. Both live and dead worms may be surrounded by erythrocytes, degranulated heterophils, and giant cells, and thick-shelled, larvated nematode eggs may be seen both free in the cysts and within female worms (Bartlett et al. 1984). In areas penetrated by the posterior end of the worms (Figure 21.3), the wall of the proventriculus may lose its structural integrity, and fibrinous exudate, scattered heterophils, and sloughed proventricular gland cells may be present in the proventricular lumen. Mucosal damage may render the host susceptible to secondary bacterial and fungal invasion (French et al. 1994).

DIAGNOSIS

Diagnosis of tetramerid infections in a living bird by examination of fecal samples is difficult and usually not recommended. Eggs are oval, thick-shelled, larvated, and, in some species, polar tufts of filaments are present. It would be difficult to distinguish them from other common spirurid eggs found in bird feces such as *Dispharynx nasuta*. Tscherner et al. (1997) used endoscopic examination of some birds to confirm the characteristic cysts of *Geopetitia* on the wall of the proventriculus.

At necropsy, lesions of *Tetrameres* and *Microtetrameres* can often be seen as dark red, slightly raised foci (which contain the female worms, swollen with ingested blood) on the serosal wall of the unopened proventriculus. Compression of the fundic glands without dissection will often express the females intact. Care should be taken to search for the smaller, colorless male worms since identification of species is primarily based on the males. Females of *Tetrameres* are globular and divided into four sectors by longitudinal grooves while females of *Microtetrameres* are twisted into two or three tight coils. Males of *Tetrameres* often have two to four rows of spines on the body, although

one subgenus, *Gynaecophila*, is spineless. Males of *Microtetrameres* are always spineless.

Both sexes of the adults of *Geopetitia* may be coiled as in *Microtetrameres* spp., but the tail of female *Geopetitia* spp. are drawn out into a posterior inflation separated by a constriction.

IMMUNITY

No experimental studies have been conducted on immunity to infections with this family of nematodes. Neither Davidson et al. (1980) nor Forrester et al. (1984) found any significant differences in prevalences or intensities of infection of *T. pattersoni* between juvenile and adult Northern Bobwhites. However, in a study of free-ranging chickens in Tanzania, Fink et al. (2005) found that prevalences of *T. americana* were significantly higher in chicks than in growers or adults, although the mean intensity did not differ significantly. Whether this difference was related to age-specific immunity remains to be proven.

PUBLIC HEALTH CONCERNS

There is no evidence that these nematodes infect humans.

DOMESTIC ANIMAL HEALTH CONCERNS

Many species of *Tetrameres* are shared between wild and domestic birds, including *T. fissispina* and *Tetrameres crami* in wild and domestic Mallards (Czaplinski 1962; McDonald 1969; Birova et al. 1990), and *Tetrameres zakharowi* in wild and domestic geese (McDonald 1969; Mollhagen 1976). Both wild and domestic birds may then act as sources for the infection of intermediate hosts. For example, Hudina and Pavlovic (1989) reported "a great number of deaths" in flocks of carrier pigeons (Rock Pigeons, *Columba livia*) attributed to infections of *T. fissispina* acquired en route during a competition, presumably from feeding on intermediate hosts in the wild.

WILDLIFE POPULATION IMPACTS

Data are lacking on the impact of most members of the Tetrameridae on wild populations of birds. An exception might be *T. fissispina*, which has been listed as a cause of morbidity and mortality in waterfowl (McDonald 1969), although this needs further study. As with other parasitic infections, factors such as crowding and change in availability of food supplies can increase stress on the host and potentiate the effect of infections. Crowding can also increase transmission and lead to infections of higher intensities.

TREATMENT AND CONTROL

Several treatment regimes have been used to treat tetramerid infections in captive birds. These include a single oral dose of Levamisole to control *T. americana* in Rock Pigeons (Panigrahy et al. 1982). A subcutaneous dose of ivermectin or a combination of Panacur and ivermectin given intraperitoneally has been used to control infections of *G. aspiculata* in zoo birds (Tscherner et al. 1997).

To prevent transmission of *G. aspiculata* at the Assiniboine Park Zoo, feeding of farm-raised crickets was stopped and efforts were made to reduce the numbers of feral insects, resulting in a decrease from nine birds infected per year in 1981 to three in 1983 (Bartlett et al. 1984). Similarly, control of cockroaches at the Viennese Zoo aviary significantly reduced mortalities due to *G. aspiculata* (Juncker-Voss et al. 2001).

We are not aware of any studies on treatment of infections of species of *Microtetrameres*, but the abovementioned drugs might be effective.

MANAGEMENT IMPLICATIONS

Although there are no reports of deaths in birds in the wild attributed to species of *Geopetitia*, the demonstrated epizootic and pathogenic potential of *G. aspiculata* in zoo environments warrants further study. The natural hosts of *G. aspiculata* are assumed to be tropical birds of the order Passeriformes, since they were the host group most often infected in zoos. Because of the well-documented lack of host specificity of *G. aspiculata*, there is legitimate concern that it could spread to the local avifauna in both Europe and North America if infected birds are kept in outdoor flight cages (Bartlett et al. 1984).

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22
Avioserpensosis

John M. Kinsella

INTRODUCTION

The term avioserpensosis is applied to infections and diseases caused by nematodes of the genus *Avioserpens* from the family Dracunculidae. This family contains only two genera—*Dracunculus*, parasitic in the body cavities and subcutaneous tissues of reptiles and mammals; and *Avioserpens*, parasitic in the subcutaneous tissues of waterbirds (Chabaud 1975). *Avioserpens* spp. produce tumorlike swellings in the submandibular region and legs, and sometimes on the shoulders and shanks of ducks and other waterbirds. These swellings can lead to interference with swallowing, asphyxiation, and death. At least three species of *Avioserpens* have caused epizootics in domestic birds in Eurasia, but similar outbreaks have not been reported thus far in wild birds.

HISTORY

Sugimoto (1919) described *Filaria taiwana* from the Mallard (*Anas platyrhynchos*) in Formosa. Wehr and Chitwood (1934) first proposed the genus *Avioserpens* for a new species, *Avioserpens denticulophasma*, from a "white heron" and the Anhinga (*Anhinga anhinga*) from North America. These two species of *Avioserpens* are now considered to be synonyms (Supryaga 1971a). The first detailed report of disease due to *Avioserpens taiwana* was by Truong-Tan-Ngog (1937) in domestic ducks in Indochina. More recently, there have been a series of reports on outbreaks of disease due to *Avioserpens* spp. in domestic ducks, geese, and chickens in Russia (Garkavi et al. 1972; Garkavi and Golub 1974) and China (Li et al. 1981, 1983, 1990; Wang et al. 1983). Similar outbreaks have not been reported in Europe or North America.

ETIOLOGY

The genus *Avioserpens* was reviewed by Supryaga (1971a), who recognized only three valid species: *Avioserpens taiwana* (Sugimoto 1919), *Avioserpens galliardi* Chabaud and Campana, 1949, and *Avioserpens mosgovoyi* Supryaga, 1965. A fourth species, *Avioserpens sichuanensis*, was described by Li (1983) from domestic ducks in China. Adults are ovoviviparous and are characterized by extreme sexual dimorphism. The females reach lengths of up to 64 cm while the males only range from 6 to 14 mm.

HOST RANGE AND DISTRIBUTION

Gibson (1973) summarized host and distribution records for the three species of *Avioserpens* recognized by Supryaga (1971a). *Avioserpens taiwana* is a parasite of species of Anseriformes in southeast Asia and the US, and has also been reported from the Anhinga and a "white heron" (either *Ardea alba* or *Egretta thula*) in the US. *A. galliardi* appears to be primarily a parasite of species of Ciconiiformes in France, Spain, Russia, Canada, and the US, but has also been reported from species of Anseriformes and Gaviiformes. *Avioserpens mosgovoyi* is a parasite of species of Gruiformes, Podicipediformes, Anseriformes, and Gaviiformes in Spain and Russia, and has recently been found for the first time in a Least Sandpiper (*Calidris minutilla*) in the US (J. Kinsella, R. Brannian, and C. Roderick, unpublished data). *Avioserpens sichuanensis* has so far been recorded only from domestic ducks.

EPIZOOTIOLOGY

Transmission and Life History

The females of *A. taiwana* are found in tumorlike swellings in subcutaneous tissues, mainly in the submandibular areas. The anterior ends of gravid females pierce the end of the swelling, producing an opening through which the first-stage larvae escape into the water. Here, they are ingested by copepods, including *Cyclops sternuus*, *Eucyclops serratulus*, *Mesocyclops leuckarti*, and *Thermocyclops hyalinus* (Wang et al. 1983). The larvae then penetrate through the gut wall of

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the copepods into the hemocoel, molting to the second stage after 3–4 days at 28–30°C, and the third stage at 7 days. In experimental studies with ducklings, males were found in the mesenteries 18 days postinfection and females in the subcutaneous tissue at 20 days. The life cycle of *A. mosgovoyi* is quite similar, with the molt to the second stage in copepods taking place at 6–8 days postinfection and the third stage at 10–11 days (Supryaga 1969, 1971b). Paratenic or transfer hosts include fish (roach, gobies, and sticklebacks), frogs, and dragonfly larvae. Paratenic hosts can ingest infective copepods and infective, encysted larvae will persist in their tissues for up to 2.5 months. Thus, definitive hosts can become infected by ingestion of either infected copepods or infected paratenic hosts. After ingestion by Eurasian Coots (*Fulica atra*), the third molt takes place in the body cavity, and the fourth-stage larvae migrate via the air sacs to the subcutaneous tissues, where they molt to adults on days 12–14 postinfection. Adult females start to discharge larvae at 4 weeks postinfection. Males persist in tissues much longer than do females, up to 260 days.

The life history of *A. sichuanensis* from Sichuan province, China, is similar (Li et al. 1988). Intermediate hosts include the copepods, *Mesocyclops leuckarti*, *Thermocyclops hyalinus*, *Thermocyclops taihokuensis*, *Eucyclops serratulus*, *Macrocyclops fuscus*, and *Tropocyclops prasinus*. In experimentally infected domestic ducklings, females begin producing larvae at 29 days postinfection, and then gradually shrivel and die over a period of 3 days. The entire life cycle, including development in the copepods, takes about 36–40 days.

Prevalence and Intensity

Although there are considerable data on prevalence and intensity of *Avioserpens* spp. in domestic birds, many of the records in wild birds are based on reports from a single infected host. In a study of 6 species of herons and egrets in Spain, Nogueserola et al. (2002) reported 4 of 65 hosts infected with *A. galliardi*, each with a single nematode. Similar infections of low prevalence and intensity have been reported for an *Avioserpens* spp. in Common Mergansers (*Mergus merganser*) from Utah (1/8 individuals, 13%) (McDonald 1974), and for *A. galliardi* in Great Egrets (*Ardea alba*) from Florida (4/63 individuals, 6%) (Sepulveda et al. 1999).

CLINICAL SIGNS

Swellings in the throat may lead to difficulty in swallowing, anorexia, retarded growth, sluggishness, and eventually even asphyxiation. Swellings of the shank may interfere with the ability to swim (Truong-Tan-Ngog 1937).

PATHOLOGY

In some cases, swellings containing worms are small. After adult worms die, they are slowly resorbed and replaced by fibrous tissues. In other cases, swellings are very large and occasionally as large as the head of the bird. In early stages of the infection, the swelling is painless and soft, but as the infection progresses, it becomes hard, voluminous, and painful. An open lesion is usually present through which the larvae are released. This opening may eventually close without complication by formation of scar tissue or may lead to secondary bacterial infection, abscesses, and fatal infections (Wehr 1971).

DIAGNOSIS

Since adult *Avioserpens* spp. are ovoviviparous and the larvae are released directly into the external environment, diagnosis must be based on characteristic swellings in the submandibular areas that may also occur occasionally on the shoulders or shanks. The extremely long females are often entangled and may be difficult to remove intact.

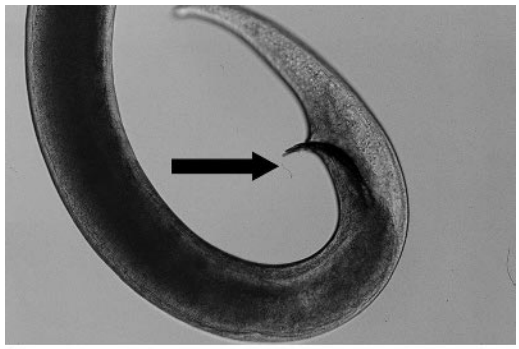
Identification of species depends on morphological features of the much smaller males, which have darkly chitinized and permanently extruded spicules (Figure 22.1a). Males are not usually present within the swelling, but may be found separately in the mesenteries or subcutaneous tissues. Both males and females have a characteristic inflation of the esophagus, which is diagnostic for members of the Dracunculidae (Figure 22.1b).

IMMUNITY

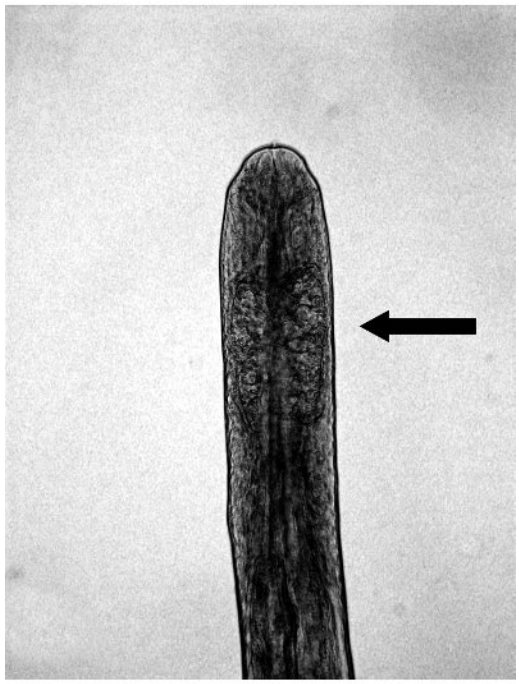
Although no experimental studies on immunity have been done, a number of authors have remarked that this is primarily a disease of young birds. Garkavi and Golub (1974) found that adult domestic ducks were resistant to infection by *A. mosgovoyi*, and Garkavi et al. (1972) found only goslings infected with an *Avioserpens* species. The most severe losses in duck flocks in China due to *A. sichuanensis* infections were reported in ducklings 1–2 months of age (Chen et al. 1984).

PUBLIC HEATH CONCERNS

There are no reports of infections of *Avioserpens* spp. in humans and infected birds pose no risk to public health.



(a)



(b)

Figure 22.1. Fixed and cleared specimens of *Avioserpens mosgovoyi*. (a) Posterior end of male showing darkly chitinized, permanently extruded spicules. (b) Anterior end of female showing characteristic inflation of the esophagus. Courtesy of C. Roderick and R. Brannian, U.S. Geological Survey, National Wildlife Health Center, Madison, WI.

DOMESTIC ANIMAL HEALTH CONCERNS

Although significant outbreaks of disease due to *Avioserpens* spp. have been reported in domestic ducks and chickens in Russia and China (Garkavi and Golub 1974; Li et al. 1981, 1990; Chen et al. 1984), there is no evidence of these infections spreading to wild birds or vice versa. It is probable that crowded conditions among domestic fowl lead to very intense infections in copepod intermediate hosts which in turn lead to high prevalence and intensity of infection in susceptible avian hosts. In 14 flocks consisting of 8,173 ducklings in Sichuan province, mortality due to *A. sichuanensis* ranged from 50 to 62% (Li et al. 1983).

WILDLIFE POPULATION IMPACTS

Reports of *Avioserpens* spp. infections in wild birds are sporadic and isolated. Epizootics in domestic birds demonstrate their potential for causing significant morbidity and mortality, especially in young birds, but no epizootics have been reported in wild populations, possibly because characteristic lesions have not been recognized. Biologists should pay particular attention to occurrence of swellings on the head, neck, sternum, and legs of wild birds.

TREATMENT AND CONTROL

Treatment of *Avioserpens* infections by systemic anthelmintics has not been reported. Some cures in China have been achieved by direct injection of anthelmintics into the parasitic swellings, although this is not common practice in western veterinary medicine (Li et al. 1981; Chen et al. 1984).

Prevention of infections in ducklings can be achieved by providing uncontaminated food and water and preventing contact with paratenic hosts. For example, an outbreak of *A. mosgovoyi* in domestic ducklings in the former USSR was attributed to ingestion of fish that had been collected from a river inhabited by infected "bald coots" (most likely Eurasian Coot, *Fulica atra*). Withdrawal of fish from the diet eradicated the infection (Garkavi and Golub 1974).

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23
Heterakis and *Ascaridia*

Alan M. Fedynich

INTRODUCTION

The genera *Heterakis* and *Ascaridia* are members of the nematode order Ascaridida, a relatively large group of parasitic nematodes that have direct life cycles and occur primarily in the gastrointestinal tracts of their hosts. Both genera occur worldwide, and one or more species have been reported from most of the major taxonomic orders of birds. Pathogenicity varies widely within both genera and is dependent on host group and history of host–parasite association. More species of *Ascaridia* appear to negatively affect birds than species of *Heterakis*. However, this may reflect disproportionate attention on those parasites that have economic impacts. Species of *Heterakis* and *Ascaridia* have caused disease and/or reduced fitness in economically important domesticated birds such as chickens, pheasants, and turkeys (Schmidt and Kuntz 1970; Norton et al. 1999; Menezes et al. 2003), wild-caught captive birds in zoological gardens (Griner et al. 1977; Callinan 1987; Balaguer et al. 1992), pen-raised game birds (Woodburn 1994; Millán et al. 2004a), and wild birds (Wehr and Shalkop 1963; Ayeni et al. 1983; Rizzoli et al. 1999; Draycott et al. 2000; Tompkins et al. 2002; Daehlen 2003).

SYNONYMS

Heterakiasis, heterakosis, ascariasis, ascarosis.

HISTORY

The heterakid and ascarid nematodes of birds have been recognized since the late 1700s. Many of the first descriptions of these parasitic nematodes were provided by early investigators, including Bloch, Dujardin, Fröhlich, Gmelin, Linstow, Railliet, Rudolphi, Schneider, and Schrank (Skrjabin et al. 1951; Yamaguti 1961). By the early 1900s, new species were described and diseases noted from necropsies performed primarily on captive wild hosts from zoological gardens (Baylis and Daubney 1922; Chandler 1926; Canavan 1929, 1931). Information from these early studies provided important insights into variation in pathogenicity of these worms among different host species. For example, reports of nodular typhlitis associated with severe infections of *Heterakis isolonche* in several species of captive pheasants helped to establish that some hosts are highly susceptible to these parasites (Schwartz 1924; Griner et al. 1977). Subsequent studies established that *Heterakis gallinarum* and *Ascaridia galli* are particularly pathogenic to Helmeted Guineafowl (*Numida meleagris*) (Ayeni et al. 1983), but not to Northern Bobwhite (*Colinus virginianus*) (Cram et al. 1931; Davidson et al. 1982).

Growing interest in the early twentieth century both in the potential exchange of parasites between wild and domestic populations and in the importance of parasites as causes of mortality in game birds led to a series of parasitological surveys of game species in North America and Europe, including Northern Bobwhite (Cram et al. 1931), Ruffed Grouse (*Bonasa umbellus*) (Morgan and Hamerstrom 1941; Erickson et al. 1949), Wild Turkey (*Meleagris gallopavo*) (Maxfield et al. 1963), Ring-necked Pheasant (*Phasianus colchicus*) (Gilbertson and Hugghins 1964), and European species of grouse, partridges, and pheasants (Madsen 1941, 1952). Consequently, early studies focusing on wild game birds contributed significantly to our understanding about the occurrence and geographic distribution of species of *Heterakis* and *Ascaridia*.

DISTRIBUTION AND HOST RANGE

Species of *Heterakis* and *Ascaridia* are widely distributed geographically, and at least one species from each genus has been found on all continents except Antarctica (Tables 23.1 and 23.2). Heterakid nematodes have been found in at least 107 wild and captive bird species (Table 23.1), and ascarid nematodes have been reported from at least 139 host species (Table 23.2). Several species are cosmopolitan (e.g.,

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Table 23.1. Nonexhaustive list of avian hosts infected with *Heterakis* spp., geographic location where infection was reported, and reporting authors.

Host	*Heterakis* sp.	Location	Reference
STRUTHIONIFORMES			
Rheidae			
Greater Rhea (*Rhea americana*)	*Heterakis gallinarum*	France (zoo)	Cram (1927)
TINAMIFORMES			
Tinamidae			
Gray Tinamou (*Tinamus tao*)	*Heterakis nattereri**	Brazil	Vicente et al. (1993)
Red-winged Tinamou (*Rhynchotus rufescens*)	*Heterakis gallinarum*	Brazil	Vicente et al. (1993)
Small-billed Tinamou (*Crypturellus parvirostris*)	*Heterakis inglisi*	Brazil	Vicente et al. (1993)
Solitary Tinamou (*Tinamus solitarius*)	*Heterakis nattereri**	Brazil	Vicente et al. (1993)
Spotted Nothura (*Nothura maculosa*)	*Heterakis gallinarum*	Brazil	Vicente et al. (1993)
	Heterakis spiculata	South America	Kaseta (1973)
Tataupa Tinamou (*Crypturellus tataupa*)	*Heterakis spiculata*	Brazil	Pinto et al. (2006)
Undulated Tinamou (*Crypturellus undulates*)	*Heterakis spiculata*	Brazil	Vicente et al. (1993)
Variegated Tinamou (*Crypturellus variegates*)	*Heterakis inglisi*	Brazil	Vicente et al. (1993)
	Heterakis spiculata	Brazil	Pinto et al. (2006)
Yellow-legged Tinamou (*Crypturellus noctivagus*)	*Heterakis spiculata*	Brazil	Vicente et al. (1993)
CICONIIFORMES			
Ardeidae			
Black-crowned Night-Heron (*Nycticorax nycticorax*)	*Heterakis pavonis*	Japan	Madsen (1950)
Ciconiidae			
Maguari Stork (*Ciconia maguari*)	*Heterakis valdemucronata**	Brazil	Cram (1927)
Marabou Stork (*Leptoptilos crumeniferus*)	*Heterakis gallinarum*	Uganda	Bwangamoi et al. (2003)
ANSERIFORMES			
Anatidae			
Ashy-headed Goose (*Chloephaga poliocephala*)	*Heterakis dispar*	England (zoo)	Madsen (1950)
Barnacle Goose (*Branta leucopsis*)	*Heterakis dispar*	NR	Cram (1927)
Black Swan (*Cygnus atratus*)	*Heterakis dispar*	Germany (zoo)	Cram (1927)
	Heterakis gallinarum	Germany (zoo)	Johnston (1912)
Canada Goose (*Branta canadensis*)	*Heterakis dispar*	USA	Cram (1927)
Cape Barren Goose (*Cereopsis novaehollandiae*)	*Heterakis dispar*	India	Maplestone (1932)†
Common Shelduck (*Tadorna tadorna*)	*Heterakis dispar*	NR	Cram (1927)
	Heterakis gallinarum	NR	Cram (1927)
Eurasian Wigeon (*Anas penelope*)	*Heterakis dispar*	NR	McDonald (1969)
Gadwall (*Anas strepera*)	*Heterakis dispar*	NR	McDonald (1969)
Greater White-fronted Goose (*Anser albifrons*)	*Heterakis dispar*	Texas, USA	Purvis et al. (1997)
Greylag Goose (*Anser anser*)	*Heterakis dispar*	NR	McDonald (1969)
Hawaiian Goose (*Branta sandvicensis*)	*Heterakis dispar*	NR	Cram (1927)
Lesser White-fronted Goose (*Anser erythropus*)	*Heterakis dispar*	NR	McDonald (1969)
Mallard (*Anas platyrhynchos*)	*Heterakis dispar*	NR	McDonald (1969)

(*continues*)

Table 23.1. (*Continued*)

Host	*Heterakis* sp.	Location	Reference
Maned Duck (*Chenonetta jubata*)	*Heterakis dispar*	Australia (zoo)	Johnston (1912)
Muscovy Duck (*Cairina moschata*)	*Heterakis dispar*	NR	Cram (1927)
Ross' Goose (*Chen rossii*)	*Heterakis dispar*	Texas, USA	Fedynich et al. (2005)
Snow Goose (*Chen caerulescens*)	*Heterakis dispar*	North America	Swinyard (1931) and Forbes et al. (1999)
Speckled Teal (*Anas flavirostris*)	*Heterakis dispar*	NR	McDonald (1969)
Swan Goose (*Anser cygnoides*)	*Heterakis dispar*	NR	McDonald (1969)
Taiga Bean-Goose (*Anser fabalis*)	*Heterakis dispar*	NR	Cram (1927)
Upland Goose (*Chloephaga picta*)	*Heterakis dispar*	Chile	González et al. (2005)
		Falkland Islands	Harradine (1982)
West Indian Whistling-Duck (*Dendrocygna arborea*)	*Heterakis dispar*	NR	McDonald (1969)
Whooper Swan (*Cygnus cygnus*)	*Heterakis dispar*	NR	McDonald (1969)
Wood Duck (*Aix sponsa*)	*Heterakis dispar*	Germany (zoo)	Cram (1927)
FALCONIFORMES			
Cathartidae			
King Vulture (*Sarcoramphus papa*)	*Heterakis gallinarum*	NR	Madsen (1950)
GALLIFORMES			
Cracidae			
Red-billed Curassow (*Crax blumenbachii*)	*Heterakis nattereri**	Brazil	Cram (1927)
	Heterakis oscari	Brazil	Yamaguti (1961)
Megapodiidae			
Australian Brush-turkey (*Alectura lathami*)	*Heterakis gallinarum*	NR	Madsen (1950)
Maleo (*Macrocephalon maleo*)	*Heterakis gallinarum*	NR	Cram (1927)
Odontophoridae			
California Quail (*Callipepla californica*)	*Heterakis isolonche*	Oregon, USA	Moore et al. (1989)
Northern Bobwhite (*Colinus virginianus*)	*Heterakis gallinarum*	North America	Venard (1933) and Davidson et al. (1982)
	Heterakis isolonche	North America	Cram et al. (1931) and Venard (1933)
Phasianidae			
Barbary Partridge (*Alectoris barbara*)	*Heterakis gallinarum*	Canary Islands	Foronda et al. (2005)
	Heterakis tenuicauda	Algeria	Madsen (1950)
Black Grouse (*Tetrao tetrix*)	*Heterakis gallinarum*	Europe	Madsen (1952) and Bezubik (1960)
Blood Pheasant (*Ithaginis cruentus*)	*Heterakis isolonche*	India (zoo)	Baylis and Daubney (1922)
Dusky Grouse (*Dendragapus obscurus*)	*Heterakis gallinarum*	North America	Buss et al. (1958)
Blue Eared-Pheasant (*Crossoptilon auritum*)	*Heterakis isolonche*	Canada	Webster (1982)
		USA (zoo, origin China)	Canavan (1929)
Brown Eared-Pheasant (*Crossoptilon mantchuricum*)	*Heterakis gallinarum*	NR	Madsen (1950)
	Heterakis isolonche	USA (zoo)	Griner et al. (1977)
Caspian Snowcock (*Tetraogallus caspius*)	*Heterakis macroura*	Turkestan	Yamaguti (1961)
Ceylon Junglefowl (*Gallus lafayetii*)	*Heterakis pusilla**	Africa	Cram (1927)
Cheer Pheasant (*Catreus wallichi*)	*Heterakis gallinarum*	NR	Madsen (1950)
Chinese Francolin (*Francolinus pintadeanus*)	*Heterakis gallinarum*	NR	Madsen (1950)

Table 23.1. (*Continued*)

Host	*Heterakis* sp.	Location	Reference
Chukar (*Alectoris chukar*)	*Heterakis gallinarum*	India	Maplestone (1932)†
Common Quail (*Coturnix coturnix*)	*Heterakis gallinarum*	Russia	Wetherbee (1961)
		Turkey	Kurtpinar (1957)
Copper Pheasant (*Syrmaticus soemmerringii*)	*Heterakis gallinarum*	NR	Madsen (1950)
	Heterakis pavonis	Japan	Madsen (1950)
Crested Fireback (*Lophura ignita*)	*Heterakis gallinarum*	India	Maplestone (1932)†
	Heterakis isoloncheo	India (zoo)	Chandler (1926)
Crested Partridge (*Rollulus rouloul*)	*Heterakis gallinarum*	Canada	Webster (1982)
Double-spurred Francolin (*Francolinus bicalcaratus*)	*Heterakis brevispiculum*	Africa	Cram (1927)
Eurasian Capercaillie (*Tetrao urogallus*)	*Heterakis gallinarum*	Europe	Bezubik (1960) and Baruš et al. (1984)
Golden Pheasant (*Chrysolophus pictus*)	*Heterakis gallinarum*	USA (zoo, origin China)	Canavan (1929, 1931)
	Heterakis isolonche	USA (zoo)	Cram (1927) and Griner et al. (1977)
Gray Junglefowl (*Gallus sonneratii*)	*Heterakis beramporia*	Asia	Levine (1968)
		India (zoo)	Madsen (1950)
	Heterakis isolonche	India	Maplestone (1932)†
Gray Partridge (*Perdix perdix*)	*Heterakis gallinarum*	Europe	Clapham (1935), Madsen (1941, 1952)
		North America	Yocom (1943)
Gray Peacock-Pheasant (*Polyplectron bicalcaratum*)	*Heterakis brevispiculum*	Africa, Brazil, Puerto Rico	Levine (1968)
		India (zoo)	Chandler (1926)
	Heterakis isolonche	India (zoo)	Maplestone (1932)†
Greater Prairie-Chicken (*Tympanuchus cupido*)	*Heterakis gallinarum*	North America	Morgan and Hamerstrom (1941) and Harper et al. (1967)
Greater Sage-Grouse (*Centrocercus urophasianus*)	*Heterakis gallinarum*	North America	Simon (1940) and Wehr (1940a)
Green Peafowl (*Pavo muticus*)	*Heterakis hamulus**	Germany (zoo)	Cram (1927)
Green Pheasant (*Phasianus versicolor*)	*Heterakis gallinarum*	NR	Cram (1927)
	Heterakis pavonis	Japan	Madsen (1950)
Hazel Grouse (*Bonasa bonasia*)	*Heterakis gallinarum*	NR	Skrjabin et al. (1951)
	Heterakis silindae	China	Madsen (1950)
Helmeted Guineafowl (*Numida meleagris*)	*Heterakis brevispiculum*	Africa	Madsen (1950)
	Heterakis gallinarum	Africa	Ayeni et al. (1983)
Hill Partridge (*Arborophila torqueola*)	*Heterakis gallinarum*	India (zoo)	Baylis and Daubney (1922)
	Heterakis vulvolabiata	India (zoo)	Chandler (1926)
Himalayan Monal (*Lophophorus impejanus*)	*Heterakis gallinarum*	India (zoo)	Baylis and Daubney (1922)
	Heterakis isolonche	India (zoo)	Baylis and Daubney (1922)

(*continues*)

Table 23.1. (*Continued*)

Host	*Heterakis* sp.	Location	Reference
Himalayan Snowcock (*Tetraogallus himalayensis*)	*Heterakis macroura*	Turkestan	Madsen (1950)
Indian Peafowl (*Pavo cristatus*)	*Heterakis gallinarum*	NR	Cram (1927)
	*Heterakis hamulus**	Germany (zoo)	Cram (1927)
Kalij Pheasant (*Lophura leucomelanos*)	*Heterakis gallinarum*	India	Maplestone (1932)†
	Heterakis isoloncheo	India	Maplestone (1932)†
Lady Amherst's Pheasant (*Chrysolophus amherstiae*)	*Heterakis gallinarum*	NR	Madsen (1950)
	Heterakis isolonche	Germany	Yamaguti (1961)
		USA (zoo, origin China)	Canavan (1931)
Lesser Prairie-Chicken (*Tympanuchus pallidicinctus*)	*Heterakis isolonche*	Texas, USA	Pence and Sell (1979)
Long-billed Partridge (*Rhizothera longirostris*)	*Heterakis interlabiata*	Africa	Yamaguti (1961)
		Asia	Cram (1927)
		England (zoo)	Madsen (1950)
Ocellated Turkey (*Meleagris ocellata*)	*Heterakis isolonche*	USA (zoo)	Griner et al. (1977)
Red Junglefowl (*Gallus gallus*)	*Heterakis beramporia*	Asia	Cram (1927)
	Heterakis brevispiculum	South America	Yamaguti (1961)
	Heterakis gallinarum	Asia	Schmidt and Kuntz (1970) and Arya (1990)
	Heterakis indica	Asia	Schmidt and Kuntz (1970)
	Heterakis kumaoni	India	Arya (1990)
	Heterakis nainitalensis	India	Arya (1990)
	Heterakis parva	Asia	Schmidt and Kuntz (1970)
	*Heterakis pusilla**	Africa	Cram (1927)
Red Spurfowl (*Galloperdix spadicea*)	*Heterakis gallinarum*	India (zoo)	Baylis and Daubney (1922)
Red-legged Partridge (*Alectoris rufa*)	*Heterakis gallinarum*	Europe	Clapham (1935) and Millán et al. (2004b)
Red-necked Francolin (*Francolinus afer*)	*Heterakis silindae*	Africa	Yamaguti (1961)
Reeves' Pheasant (*Syrmaticus reevesii*)	*Heterakis gallinarum*	NR	Madsen (1950)
Ring-necked Pheasant (*Phasianus colchicus*)	*Heterakis gallinarum*	Europe	Clapham (1935) and Madsen (1952)
		North America	Gilbertson and Hugghins (1964)
	Heterakis isolonche	North America, Russia	Yamaguti (1961)
	Heterakis pavonis	Asia	Levine (1968)
Rock Partridge (*Alectoris graeca*)	*Heterakis dispar*	Turkey	Köroğlu and Tasan (1996)
	Heterakis gallinarum	Italy	Rizzoli et al. (1999)
		Turkey	Köroğlu and Tasan (1996)
	Heterakis tenuicauda	Italy	Rizzoli et al. (1999)
		Turkestan	Madsen (1950)

Table 23.1. (*Continued*)

Host	*Heterakis* sp.	Location	Reference
Rock Ptarmigan (*Lagopus muta*)	*Heterakis gallinarum*	Europe	Madsen (1952) (citing Galli-Valerio 1931 and Redi 1708)
		North America	Braun and Willers (1967) (see references therein)
Ruffed Grouse (*Bonasa umbellus*)	*Heterakis gallinarum*	North America	Wehr (1940a) and Erickson et al. (1949)
	Heterakis isolonche	North America	Wehr (1940a) and Kalla et al. (1997)
Satyr Tragopan (*Tragopan satyra*)	*Heterakis bosia*	India (zoo)	Baylis and Daubney (1922)
	Heterakis gallinarum	India (zoo)	Baylis and Daubney (1922)
	Heterakis isolonche	India (zoo)	Baylis and Daubney (1922)
	Heterakis tragopanis	India (zoo)	Lal (1942)
See-see Partridge (*Ammoperdix griseogularis*)	*Heterakis tenuicauda*	Turkestan	Madsen (1950)
Sharp-tailed Grouse (*Tympanuchus phasianellus*)	*Heterakis gallinarum*	North America	Morgan and Hamerstrom (1941)
	Heterakis pedioecetes	Canada	Mawson (1956)
Silver Pheasant (*Lophura nycthemera*)	*Heterakis beramporia*	Asia	Levine (1968)
	Heterakis dispar	India	Maplestone (1932)†
	Heterakis gallinarum	India	Maplestone (1932)†
	Heterakis isolonche	India	Maplestone (1932)†
	Heterakis parva	India (zoo)	Maplestone (1932)†
	Heterakis pavonis	Asia	Levine (1968)
		India (zoo)	Maplestone (1932)†
Sooty Grouse (*Dendragapus fuliginosus*)	*Heterakis gallinarum*	North America	Beer (1944)
Swamp Francolin (*Francolinus gularis*)	*Heterakis gallinarum*	India (zoo)	Baylis and Daubney (1922)
Vulturine Guineafowl (*Acryllium vulturinum*)	*Heterakis gallinarum*	India	Maplestone (1932)†
	Heterakis tenuicauda	Africa	Yamaguti (1961)
Wild Turkey (*Meleagris gallopavo*)	*Heterakis gallinarum*	North America	Maxfield et al. (1963)
	Heterakis meleagris	China	Yamaguti (1961)
Willow Ptarmigan (*Lagopus lagopus*)	*Heterakis gallinarum*	Europe	Madsen (1950)
		North America	Cram (1927)
GRUIFORMES			
Otididae			
Great Bustard (*Otis tarda*)	*Heterakis gallinarum*	Asia, Germany (zoo)	Cram (1927)
Houbara Bustard (*Chlamydotis undulate*)	*Heterakis gallinarum*	India	Madsen (1950)
Little Bustard (*Tetrax tetrax*)	*Heterakis gallinarum*	Asia, Germany (zoo)	Cram (1927)
Psophiidae			
Dark-winged Trumpeter (*Psophia viridis*)	*Heterakis psophiae*	Brazil	Cram (1927)
	*Heterakis skrjabini**	Brazil	Cram (1927)

(*continues*)

Table 23.1. (*Continued*)

Host	*Heterakis* sp.	Location	Reference
CHARADRIIFORMES			
Alcidae			
Crested Auklet (*Aethia cristatella*)	*Heterakis kurilensis*	Russia	Yamaguti (1961) and Muzaffar and Jones (2004) (citing Baruš et al. 1978)
Whiskered Auklet (*Aethia pygmaea*)	*Heterakis kurilensis*	Russia	Yamaguti (1961)
COLUMBIFORMES			
Columbidae			
Eared Dove (*Zenaida auriculata*)	*Heterakis gallinarum*	Chile	González et al. (2004)
Eurasian Collared-Dove (*Streptopelia decaocto*)	*Heterakis gallinarum*	Czechoslovakia	Baruš (1966a)
PSITTACIFORMES			
Cacatuidae			
Pink Cockatoo (*Cacatua leadbeateri*)	*Heterakis gallinarum*	NR	Madsen (1950)
STRIGIFORMES			
Strigidae			
Eurasian Pygmy-Owl (*Glaucidium passerinum*)	*Heterakis dispar*	NR	Cram (1927)

Note: Some host–parasite reports found only in the review literature did not include geographic location and often included hosts from wild, domestic, and private and public zoological collections; where possible, hosts from zoos and host origin of capture are noted. NR, not reported.

*Reported as species inquirenda by Madsen (1950).

†Maplestone (1932) included hosts pooled together from zoo and/or domestic birds.

Heterakis dispar, *H. gallinarum*, *Ascaridia columbae*, *A. galli*), which reflects the worldwide distribution of their hosts. Other species tend to occur in specific geographic regions (e.g., *Heterakis pavonis* in Japan, *Ascaridia geei* in China) or tend to occur in particular host families (e.g., *Heterakis kurilensis* in Alcidae, *Ascaridia aegyptiaca* in Caprimulgidae, and *Ascaridia hermaphroditia* and *Ascaridia platyceri* in Psittacidae).

ETIOLOGY

Heterakis and *Ascaridia* are classified in the phylum Nemata (Nematoda), class Secernentea, order Ascaridida, and superfamily Heterakoidea. Members of this superfamily have monoxenous (direct) life cycles, develop within the gastrointestinal tract of the definitive host, produce eggs that embryonate outside the host, and have a prominent preanal sucker encompassed by a cuticularized ring (Anderson 2000). The superfamily Heterakoidea includes the families Heterakidae and Ascaridiidae. The subfamily Heterakinae is placed within the Heterakidae and contains the genera *Africana*, *Ganguleterakis*, *Heraldakis*, *Heterakis*, *Odontoterakis*, *Paraspidodera*, *Spinicauda*, and *Strongyluris* (Skrjabin et al. 1951; Inglis 1991). Depending on taxonomic authority, there are about 29 species of *Heterakis* (Table 23.1). Some of these are incompletely described and will likely be synonymized after additional study (Madsen 1950).

The family Ascaridiidae includes the subfamily Ascaridiinae with the single genus *Ascaridia* (Skrjabin et al. 1951). At least 41 species of *Ascaridia* have been reported from birds (Table 23.2).

Species of *Heterakis* are small (5.5–31 mm; males slightly smaller than females), white-to-tan colored nematodes (Figure 23.1) that typically occur in the ceca of the definitive host (Cram 1927; Maplestone 1932; Madsen 1950). Like other members of their subfamily, heterakid nematodes have a transversely striated cuticle; three lips that encircle the mouth, with each lip having two papillae; lateral flanges or alae (may be absent); a three-part esophagus composed of a pharynx that is narrow anteriorly, broadens posteriorly, and ends with a well-developed posterior bulb; and a valvular apparatus within the bulb of the esophagus (Figure 23.2) (Skrjabin et al. 1951; Yamaguti 1961; Yorke and Maplestone 1962).

Table 23.2. Nonexhaustive list of avian hosts infected with *Ascaridia* spp., geographic location where infection was reported, and reporting authors.

Host	*Ascaridia* sp.	Location	Reference
STRUTHIONIFORMES			
Rheidae			
Greater Rhea (*Rhea americana*)	*Ascaridia orthocerca*	Brazil, Italy (zoo)	Cram (1927)
Struthionidae			
Ostrich (*Struthio camelus*)	*Ascaridia struthionis*	Italy (zoo)	Skrjabin et al. (1951)
TINAMIFORMES			
Tinamidae			
Red-winged Tinamou (*Rhynchotus rufescens*)	*Ascaridia pintoi*	Brazil	Skrjabin et al. (1951)
Spotted Nothura (*Nothura maculosa*)	*Ascaridia brasiliana*	Brazil	Yamaguti (1961)
CICONIIFORMES			
Ardeidae			
Little Egret (*Egretta garzetta*)	*Ascaridia aegyptiaca*	Egypt	Cram (1927)
Ciconiidae			
Marabou Stork (*Leptoptilos crumeniferus*)	*Ascaridia galli*	Africa	Bwangamoi et al. (2003)
ANSERIFORMES			
Anatidae			
Black Scoter (*Melanitta nigra*)	*Ascaridia galli*	NR	McDonald (1969)
Common Pochard (*Aythya ferina*)	*Ascaridia galli*	NR	McDonald (1969)
Greylag Goose (*Anser anser*)	*Ascaridia galli*	NR	Skrjabin et al. (1951)
Mallard (*Anas platyrhynchos*)	*Ascaridia galli*	NR	Gower (1939)
	Ascaridia styphlocerca	Africa	Gower (1939)
Muscovy Duck (*Cairina moschata*)	*Ascaridia galli*	NR	Gower (1939)
Northern Pintail (*Anas acuta*)	*Ascaridia galli*	NR	Cram (1927)
FALCONIFORMES			
Accipitridae			
Crested Goshawk (*Accipiter trivirgatus*)	*Ascaridia galli*	Taiwan	Su and Fei (2004)
Eastern Marsh-Harrier (*Circus spilonotus*)	*Ascaridia dolichocerca*	New Guinea	Cram (1927)
Sharp-shinned Hawk (*Accipiter striatus*)	*Ascaridia galli*	Massachusetts, USA	Rankin (1946)
GALLIFORMES			
Cracidae			
Black-fronted Piping-Guan (*Pipile jacutinga*)	*Ascaridia sergiomeirai*	Brazil	Yamaguti (1961)
	Ascaridia serrata	Brazil	Skrjabin et al. (1951)
Megapodiidae			
Australian Brush-turkey (*Alectura lathami*)	*Ascaridia catheturina*	Australia	Skrjabin et al. (1951)
Odontophoridae			
Northern Bobwhite (*Colinus virginianus*)	*Ascaridia compar*	NR	Baylis and Daubney (1922)
	Ascaridia galli	North America	Cram et al. (1931)
Scaled Quail (*Callipepla squamata*)	*Ascaridia cordata*	Mexico	Cram (1927)
Phasianidae			
Ahanta Francolin (*Francolinus ahantensis*)	*Ascaridia francolina*	Africa	Skrjabin et al. (1951)

(*continues*)

Table 23.2. (*Continued*)

Host	*Ascaridia* sp.	Location	Reference
Barbary Partridge (*Alectoris barbara*)	*Ascaridia galli*	Canary Islands	Foronda et al. (2005)
Black Grouse (*Tetrao tetrix*)	*Ascaridia compar*	Europe	Bezubik (1960)
	Ascaridia galli	Denmark	Madsen (1952)
	Ascaridia magnipapilla	Europe	Cram (1927) and Baruš (1966b)
Blood Pheasant (*Ithaginis cruentus*)	*Ascaridia galli*	India (zoo)	Baylis and Daubney (1922)
Dusky Grouse (*Dendragapus obscurus*)	*Ascaridia bonasae*	Canada	Bendell (1955)
Chukar (*Alectoris chukar*)	*Ascaridia compar*	India (zoo)	Baylis and Daubney (1922)
		USA (zoo, origin Asia)	Canavan (1929)
	Ascaridia galli	Nevada, USA	Tibbitts and Babero (1969)
	Ascaridia numidae	USA (zoo, origin Asia)	Canavan (1929)
Common Quail (*Coturnix coturnix*)	*Ascaridia compar*	Russia	Wetherbee (1961)
Crested Guineafowl (*Guttera pucherani*)	*Ascaridia numidae*	Africa	Skrjabin et al. (1951)
Double-spurred Francolin (*Francolinus bicalcaratus*)	*Ascaridia francolina*	Africa	Cram (1927)
Eurasian Capercaillie (*Tetrao urogallus*)	*Ascaridia compar*	Europe	Lund (1946) and Baruš et al. (1984)
	Ascaridia galli	Europe	Lund (1946) and Baruš (1966b)
	Ascaridia magnipapilla	Europe	Baruš (1966b)
Gray Partridge (*Perdix perdix*)	*Ascaridia compar*	Czechoslovakia	Baruš (1966b)
		Denmark	Madsen (1941)
	Ascaridia galli	Denmark	Madsen (1952)
Greater Prairie-Chicken (*Tympanuchus cupido*)	*Ascaridia galli*	North America	Morgan and Hamerstrom (1941)
Hazel Grouse (*Bonasa bonasia*)	*Ascaridia compar*	Europe	Skrjabin et al. (1951)
	Ascaridia galli	NR	Skrjabin et al. (1951)
Helmeted Guineafowl (*Numida meleagris*)	*Ascaridia compar*	NR	Baylis and Daubney (1922)
	Ascaridia galli	Nigeria	Ayeni et al. (1983)
	Ascaridia numidae	Africa	Cram (1927)
Himalayan Snowcock (*Tetraogallus himalayensis*)	*Ascaridia skrjabini*	Russia	Yamaguti (1961)
Indian Peafowl (*Pavo cristatus*)	*Ascaridia columbae*	NR	Skrjabin et al. (1951)
	Ascaridia galli	NR	Yamaguti (1961)
Ocellated Turkey (*Meleagris ocellata*)	*Ascaridia galli*	NR	Skrjabin et al. (1951)
Red Junglefowl (*Gallus gallus*)	*Ascaridia compar*	NR	Baylis and Daubney (1922)
	Ascaridia galli	Asia	Schmidt and Kuntz (1970)
		Brazil	Cram (1927)
	Ascaridia styphlocerca	Africa	Yamaguti (1961)
Ring-necked Pheasant (*Phasianus colchicus*)	*Ascaridia compar*	Czechoslovakia	Baruš (1966b)
	Ascaridia galli	Europe	Madsen (1952)
		North America	Gilbertson and Hugghins (1964)
Rock Partridge (*Alectoris graeca*)	*Ascaridia compar*	Italy	Rizzoli et al. (1999)
	Ascaridia numidae	Africa	Skrjabin et al. (1951)

Table 23.2. (*Continued*)

Host	*Ascaridia* sp.	Location	Reference
Rock Ptarmigan (*Lagopus muta*)	*Ascaridia borealis*	Europe	Skrjabin et al. (1951)
	Ascaridia compar	Alaska, USA	Babero (1953)
		Norway	Madsen (1952)
	Ascaridia galli	Alaska, USA	Braun and Willers (1967) (citing DeLeonardis 1952)
Ruffed Grouse (*Bonasa umbellus*)	*Ascaridia bonasae*	North America	Rankin (1946) and Mawson (1956)
	Ascaridia galli	North America	Connell and Doremus (1937) and Wehr (1940a, b)
Sharp-tailed Grouse (*Tympanuchus phasianellus*)	*Ascaridia galli*	North America	Morgan and Hamerstrom (1941) and Wehr (1940a)
Vulturine Guineafowl (*Acryllium vulturinum*)	*Ascaridia numidae*	USA (zoo, origin Africa)	Canavan (1929)
White-tailed Ptarmigan (*Lagopus leucura*)	*Ascaridia compar*	Alaska, USA	Babero (1953)
	Ascaridia galli	Alaska, USA	Braun and Willers (1967) (citing DeLeonardis 1952)
Wild Turkey (*Meleagris gallopavo*)	*Ascaridia dissimilis*	Europe	Levine (1968)
		North America	Maxfield et al. (1963)
	Ascaridia galli	North America	Maxfield et al. (1963)
Willow Ptarmigan (*Lagopus lagopus*)	*Ascaridia borealis*	Europe	Skrjabin et al. (1951)
	Ascaridia compar	Europe	Madsen (1952)
		North America	Babero (1953)
	Ascaridia galli	Alaska, USA	Braun and Willers (1967) (citing DeLeonardis 1952)
GRUIFORMES			
Cariamidae			
Red-legged Seriema (*Cariama cristata*)	*Ascaridia pterophora*	Brazil	Cram (1927)
Gruidae			
Black Crowned-Crane (*Balearica pavonina*)	*Ascaridia cristata*	India (zoo)	Baylis and Daubney (1922)
Blue Crane (*Anthropoides paradiseus*)	*Ascaridia stroma*	USA (zoo, origin Africa)	Canavan (1929)
Common Crane (*Grus grus*)	*Ascaridia stroma*	India (zoo)	Baylis and Daubney (1922)
Demoiselle Crane (*Anthropoides virgo*)	*Ascaridia stroma*	USA (zoo, origin Asia)	Canavan (1931)
Gray Crowned-Crane (*Balearica regulorum*)	*Ascaridia cristata*	NR	Baylis and Daubney (1922)
Sandhill Crane (*Grus canadensis*)	*Ascaridia pterophora*	Florida, USA	Spalding et al. (1996)
Sarus Crane (*Grus antigone*)	*Ascaridia cristata*	India (zoo)	Baylis and Daubney (1922)
	Ascaridia stroma	NR	Cram (1927)
Whooping Crane (*Grus americana*)	*Ascaridia pterophora*	Florida, USA	Spalding et al. (1996)
COLUMBIFORMES			
Columbidae			
African Green-Pigeon (*Treron calvus*)	*Ascaridia fasciata*	Africa	Skrjabin et al. (1951)

(*continues*)

Table 23.2. (*Continued*)

Host	*Ascaridia* sp.	Location	Reference
Band-tailed Pigeon (*Patagioenas fasciata*)	*Ascaridia columbae*	Colorado, USA	Olsen and Braun (1980)
Black-billed Cuckoo-Dove (*Macropygia nigrirostris*)	*Ascaridia australis*	Australia	Cram (1927)
Common Wood-Pigeon (*Columba palumbus*)	*Ascaridia columbae*	Czechoslovakia	Baruš (1966b) (citing Vojtechovská-Mayerová 1952)
Eurasian Collared-Dove (*Streptopelia decaocto*)	*Ascaridia columbae*	Czechoslovakia	Baruš (1966a)
		Florida, USA	Bean et al. (2005)
Eurasian Turtle-Dove (*Streptopelia turtur*)	*Ascaridia columbae*	Czechoslovakia	Baruš (1966a)
Luzon Bleeding-heart (*Gallicolumba luzonica*)	*Ascaridia columbae*	Canada	Webster (1982)
		India (zoo)	Baylis and Daubney (1922)
Madagascar Green-Pigeon (*Treron australis*)	*Ascaridia longecirrata*	Belgian Congo	Yamaguti (1961)
Mourning Dove (*Zenaida macroura*)	*Ascaridia columbae*	North America	Lee et al. (2004)
Oriental Turtle-Dove (*Streptopelia orientalis*)	*Ascaridia columbae*	NR	Skrjabin et al. (1951)
	Ascaridia geei	China	Skrjabin et al. (1951)
Picui Ground-Dove (*Columbina picui*)	*Ascaridia columbae*	NR	Cram (1927)
Rameron Pigeon (*Columba arquatrix*)	*Ascaridia columbae*	NR	Cram (1927)
	Ascaridia maculosa	Africa	Skrjabin et al. (1951)
Rock Pigeon (*Columba livia*)	*Ascaridia columbae*	Chile	Toro et al. (1999)
		Czechoslovakia	Baruš (1966a)
		India	Wajihullali et al. (1982)
		North America	Wehr and Hwang (1964)
	Ascaridia maculosa	Germany	Yamaguti (1961)
	Ascaridia razia	India	Skrjabin et al. (1951)
Ruddy Ground-Dove (*Columbina talpacoti*)	*Ascaridia columbae*	NR	Cram (1927)
	Ascaridia magalhãesi	Brazil	Yamaguti (1961)
Ruddy Quail-Dove (*Geotrygon montana*)	*Ascaridia magalhãesi*	Brazil	Cram (1927)
Scaled Pigeon (*Patagioenas speciosa*)	*Ascaridia columbae*	NR	Cram (1927)
Spotted Dove (*Streptopelia chinensis*)	*Ascaridia columbae*	NR	Skrjabin et al. (1951)
White-winged Dove (*Zenaida asiatica*)	*Ascaridia columbae*	North America	Conti and Forrester (1981)
Yellow-footed Pigeon (*Treron phoenicopterus*)	*Ascaridia columbae*	India (zoo)	Baylis and Daubney (1922)
PSITTACIFORMES			
Cacatuidae			
Cockatiel (*Nymphicus hollandicus*)	*Ascaridia platyceri*	Canada	Webster (1982)
		Germany	Hartwich and Tscherner (1979)
Ducorps' Cockatoo (*Cacatua ducorpsii*)	*Ascaridia platyceri*	Czech Republic	Kajerova et al. (2004b)
Yellow-crested Cockatoo (*Cacatua sulphurea*)	*Ascaridia hermaphrodita*	South America	Cram (1927)
	Ascaridia platyceri	Czech Republic	Kajerova et al. (2004b)
Psittacidae			
Alexandra's Parrot (*Polytelis alexandrae*)	*Ascaridia columbae*	Australia, Brazil	Kajerova et al. (2004a) (citing Johnston and Mawson 1941 and Ferrola et al. 1976)
	Ascaridia platyceri	Australia	Mines (1979)

Table 23.2. (*Continued*)

Host	*Ascaridia* sp.	Location	Reference
Austral Parakeet (*Enicognathus ferrugineus*)	*Ascaridia platyceri*	Germany	Hartwich and Tscherner (1979)
Australian King-Parrot (*Alisterus scapularis*)	*Ascaridia columbae*	Australia	Kajerova et al. (2004a)
	Ascaridia platyceri	Czech Republic	Kajerova et al. (2004b)
Black-billed Parrot (*Amazona agilis*)	*Ascaridia hermaphrodita*	South America	Cram (1927)
Black-winged Lovebird (*Agapornis taranta*)	*Ascaridia platyceri*	Germany	Hartwich and Tscherner (1979)
Blue-and-yellow Macaw (*Ara ararauna*)	*Ascaridia hermaphrodita*	Brazil	Skrjabin et al. (1951)
Bluebonnet (*Northiella haematogaster*)	*Ascaridia platyceri*	Australia	Mines (1979)
Blue-crowned Parakeet (*Aratinga acuticaudata*)	*Ascaridia hermaphrodita*	South America	Kajerova et al. (2004a)
Blue-fronted Parrot (*Amazona aestiva*)	*Ascaridia hermaphrodita*	South America	Cram (1927)
Blue-headed Parrot (*Pionus menstruus*)	*Ascaridia hermaphrodita*	South America	Kajerova et al. (2004a)
Bourke's Parrot (*Neophema bourkii*)	*Ascaridia columbae*	Australia	Kajerova et al. (2004a)
	Ascaridia platyceri	Australia	Mines (1979)
		Germany	Hartwich and Tscherner (1979)
Brown-throated Parakeet (*Aratinga pertinax*)	*Ascaridia hermaphrodita*	South America	Kajerova et al. (2004a)
Budgerigar (*Melopsittacus undulatus*)	*Ascaridia columbae*	Australia, Brazil	Kajerova et al. (2004a)
	Ascaridia platyceri	Canada	Webster (1982)
		Czech Republic	Kajerova et al. (2004b)
Caatinga Parakeet (*Aratinga cactorum*)	*Ascaridia hermaphrodita*	Brazil	Skrjabin et al. (1951)
Crimson Rosella (*Platycercus elegans*)	*Ascaridia platyceri*	Czech Republic	Kajerova et al. (2004b)
		Germany	Hartwich and Tscherner (1979)
Cuban Parrot (*Amazona leucocephala*)	*Ascaridia hermaphrodita*	South America	Cram (1927)
	Ascaridia platyceri	Czech Republic	Kajerova et al. (2004b)
Dusky Parrot (*Pionus fuscus*)	*Ascaridia hermaphrodita*	Brazil	Skrjabin et al. (1951)
Eastern Rosella (*Platycercus eximius*)	*Ascaridia galli*	United Kingdom	Peirce and Bevan (1973)
	Ascaridia platyceri	Czech Republic	Kajerova et al. (2004b)
		Germany	Hartwich and Tscherner (1979)
Elegant Parrot (*Neophema elegans*)	*Ascaridia galli*	England	Peirce and Bevan (1973)
	Ascaridia platyceri	Australia	Mines (1979)
Festive Parrot (*Amazona festiva*)	*Ascaridia hermaphrodita*	South America	Cram (1927)
Fischer's Lovebird (*Agapornis fischeri*)	*Ascaridia platyceri*	Czech Republic (origin Africa)	Kajerova et al. (2004a, b)
Golden-winged Parakeet (*Brotogeris chrysoptera*)	*Ascaridia sergiomeirai*	Brazil	Kajerova et al. (2004a)
Green-cheeked Parakeet (*Pyrrhura molinae*)	*Ascaridia hermaphrodita*	Brazil	Skrjabin et al. (1951)
Green-rumped Parrotlet (*Forpus passerinus*)	*Ascaridia sergiomeirai*	Brazil	Yamaguti (1961)
Hispaniolan Parakeet (*Aratinga chloroptera*)	*Ascaridia hermaphrodita*	Brazil	Skrjabin et al. (1951)

(*continues*)

Table 23.2. (*Continued*)

Host	*Ascaridia* sp.	Location	Reference
Hyacinth Macaw (*Anodorhynchus hyacinthinus*)	*Ascaridia hermaphrodita*	Germany (zoo)	Hartwich and Tscherner (1979)
Jandaya Parakeet (*Aratinga jandaya*)	*Ascaridia platyceri*	Czech Republic	Kajerova et al. (2004b)
Long-tailed Parakeet (*Psittacula longicauda*)	*Ascaridia nicobarensis*	Great Nicobar Island (India)	Soota et al. (1971)
Mallee Ringneck (*Barnardius barnardi*)	*Ascaridia platyceri*	Australia	Mines (1979)
Mealy Parrot (*Amazona farinose*)	*Ascaridia hermaphrodita*	Brazil	Skrjabin et al. (1951)
Orange-winged Parrot (*Amazona amazonica*)	*Ascaridia hermaphrodita*	USA (zoo)	Canavan (1931)
	Ascaridia ornata	Brazil	Kajerova et al. (2004a)
Port Lincoln Parrot (*Barnardius zonarius*)	*Ascaridia platyceri*	Czech Republic	Kajerova et al. (2004b)
Puerto Rican Parrot (*Amazona vittata*)	*Ascaridia hermaphrodita*	South America	Kajerova et al. (2004a)
Red-and-green Macaw (*Ara chloropterus*)	*Ascaridia hermaphrodita*	Argentina	Martínez et al. (2003)
Red-lored Parrot (*Amazona autumnalis*)	*Ascaridia hermaphrodita*	Nicaragua	Schmidt and Neiland (1973)
Red-necked Parrot (*Amazona arausiaca*)	*Ascaridia hermaphrodita*	South America	Kajerova et al. (2004a)
Red-rumped Parrot (*Psephotus haematonotus*)	*Ascaridia platyceri*	Australia	Mines (1979)
		Germany	Hartwich and Tscherner (1979)
		Czech Republic	Kajerova et al. (2004b)
Red-winged Parrot (*Aprosmictus erythropterus*)	*Ascaridia platyceri*	Australia	Mines (1979)
Regent Parrot (*Polytelis anthopeplus*)	*Ascaridia platyceri*	Australia	Mines (1979)
		Germany	Hartwich and Tscherner (1979)
Rose-ringed Parakeet (*Psittacula krameri*)	*Ascaridia hermaphrodita*	USA (zoo, origin Brazil)	Canavan (1931)
	Ascaridia platyceri	Czech Republic (origin Africa)	Kajerova et al. (2004a, b)
Rosy-faced Lovebird (*Agapornis roseicollis*)	*Ascaridia platyceri*	Czech Republic (origin Africa)	Kajerova et al. (2004a, b)
Sapphire-rumped Parrotlet (*Touit purpuratus*)	*Ascaridia hermaphrodita*	Russia	Kajerova et al. (2004a)
Scaly-headed Parrot (*Pionus maximiliani*)	*Ascaridia hermaphrodita*	Brazil	Skrjabin et al. (1951)
	Ascaridia sergiomeirai	Brazil	Pinto et al. (1993)
Scarlet Macaw (*Ara macao*)	*Ascaridia hermaphrodita*	Brazil	Skrjabin et al. (1951)
Scarlet-chested Parrot (*Neophema splendida*)	*Ascaridia platyceri*	Australia	Mines (1979)
		Czech Republic	Kajerova et al. (2004b)
Sun Parakeet (*Aratinga solstitialis*)	*Ascaridia hermaphrodita*	South America	Cram (1927)
Tui Parakeet (*Brotogeris sanctithomae*)	*Ascaridia sergiomeirai*	Brazil	Skrjabin et al. (1951)
Turquoise Parrot (*Neophema pulchella*)	*Ascaridia platyceri*	Australia	Mines (1979)
Vinaceous Parrot (*Amazona vinacea*)	*Ascaridia hermaphrodita*	South America	Cram (1927)
White-eared Parakeet (*Pyrrhura leucotis*)	*Ascaridia hermaphrodita*	South America	Kajerova et al. (2004a)

(*continues*)

Table 23.2. (*Continued*)

Host	*Ascaridia* sp.	Location	Reference
White-eyed Parakeet (*Aratinga leucophthalma*)	*Ascaridia hermaphrodita*	Brazil	Skrjabin et al. (1951)
	Ascaridia sergiomeirai	Brazil	Pinto et al. (1993)
Yellow-collared Lovebird (*Agapornis personatus*)	*Ascaridia platyceri*	Czech Republic	Kajerova et al. (2004b)
		New Zealand	Weeks (1981)
Yellow-crowned Parrot (*Amazona ochrocephala*)	*Ascaridia hermaphrodita*	South America	Cram (1927)
Yellow-fronted Parakeet (*Cyanoramphus auriceps*)	*Ascaridia platyceri*	Germany	Hartwich and Tscherner (1979)
CUCULIFORMES			
Cuculidae			
Common Cuckoo (*Cuculus canorus*)	*Ascaridia cuculina*	Russia	Skrjabin et al. (1951)
Greater Coucal (*Centropus sinensis*)	*Ascaridia circularis*	Thailand	Skrjabin et al. (1951)
	Ascaridia trilabium	Asia	Cram (1927)
STRIGIFORMES			
Strigidae			
Eurasian Eagle-Owl (*Bubo bubo*)	*Ascaridia galli*	NR	McDonald (1969)
New Britain Hawk-Owl (*Ninox odiosa*)	*Ascaridia australis*	Australia	Skrjabin et al. (1951)
CAPRIMULGIFORMES			
Caprimulgidae			
Nacunda Nighthawk (*Podager nacunda*)	*Ascaridia amblimoria*	Brazil	Skrjabin et al. (1951)
PASSERIFORMES			
Emberizidae			
Yellowhammer (*Emberiza citronella*)	*Ascaridia galli*	NR	McDonald (1969)
Passeridae			
House Sparrow (*Passer domesticus*)	*Ascaridia galli*	NR	McDonald (1969)
Turdidae			
Mistle Thrush (*Turdus viscivorus*)	*Ascaridia galli*	NR	Yamaguti (1961)

Note: Some host–parasite reports found only in the review literature did not include geographic location and often included hosts from wild, domestic, and private and public zoological collections; where possible, hosts from zoos and host origin of capture are noted. NR, not reported.

Ascaridia are large (16–120 mm; males typically smaller than females), opaque white-colored worms (Figure 23.1) found in the intestinal tract (Cram 1927; Ruff 1984). Like other members of their subfamily, ascarid nematodes have lips that lack an interlabia, a long club-shaped esophagus without a posterior bulb, cuticular lateral flanges (may be absent), and lack a ventriculus and ceca (Skrjabin et al. 1951; Yamaguti 1961; Yorke and Maplestone 1962).

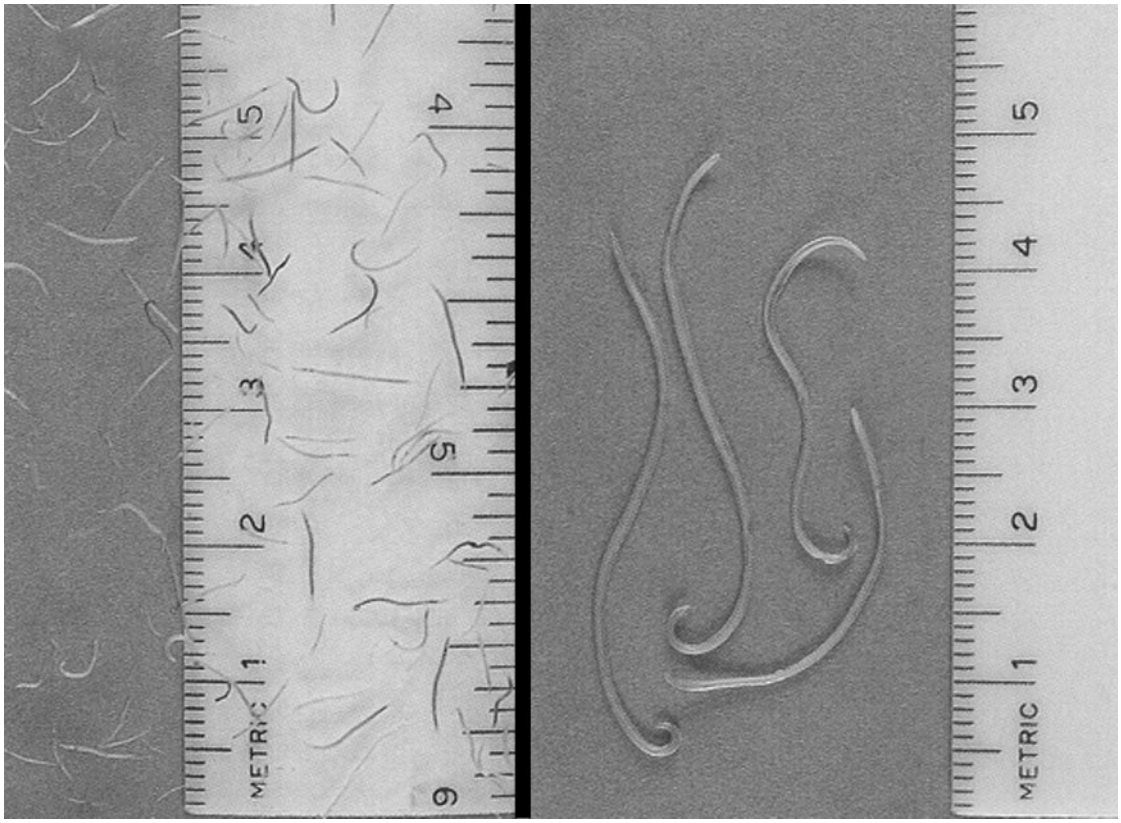
EPIZOOTIOLOGY

Life History

Most life history information about heterakid and ascarid nematodes is based on studies of species that occur in economically important host species such as pigeons and poultry. These include *A. columbae*, *Ascaridia dissimilis*, *A. galli*, *Ascaridia numidae*, *H. gallinarum*, and *H. isolonche*. In general, gravid females release unembryonated eggs into the intestinal tract of the host and the eggs are subsequently voided with the feces (Ruff 1984; Anderson 2000). Embryonation occurs after eggs exit the host, and transmission is complete when a susceptible host ingests the embryonated egg (Anderson 2000).

Embryonation is dependent on environmental conditions. Optimal development of the eggs of *H. gallinarum* occurs at 18–33°C and eggs take from 7 to 17 days to become infective (Anderson 2000). By contrast, eggs of *A. galli* embryonate in about 5–14 days at 22–34°C (Anderson 2000).

At least two species, *H. gallinarum* and *A. galli*, use earthworms as transport hosts. Larvae of both nematode species have been found in *Lumbricus terrestris*, *Allolobophora caliginosa*, and *Eisenia foetida*,



(a) (b)

Figure 23.1. (a) *Heterakis* and *Ascaridia* (b). Courtesy of R. Davidson, Southeastern Cooperative Wildlife Disease Study; originally published in *Field Manual of Wildlife Diseases in the Southeastern United States*, 2nd ed. Southeastern Cooperative Wildlife Disease Study, Athens, GA.

but only larvae of *H. gallinarum* appear to accumulate (Lund et al. 1966; Augustine and Lund 1974). Eggs hatch after they are ingested by the earthworm and transmission occurs when the earthworm is itself eaten by the definitive host (Lund et al. 1966; Augustine and Lund 1974). Eggs and larvae of *A. galli* are retained briefly by earthworms before being expelled into the soil (Augustine and Lund 1974). Other transport hosts have been documented for *H. gallinarum*, including sow bugs (*Porcellio scaber*) (Spindler 1967) and grasshoppers (*Bruneria brunnea*, *Camnula pellucida*, and *Melanoplus* spp.) (Frank 1953). Flies (*Musca domestica* and *Lucilia* sp.) can also serve as mechanical carriers of the eggs of *H. gallinarum* (Frank 1953).

In chickens, eggs of *H. gallinarum* hatch in the upper intestinal tract and larvae migrate to the ceca within 24 h (Ruff 1984). Here the larvae occur in close association with the cecal mucosa and may invade the crypts of the mucosa (Vatne and Hansen 1965). Peak association with cecal tissue is about 3 days and by day 12, most larvae have migrated to the distal third of the cecal lumen (Vatne and Hansen 1965) where they develop into adults. Females mature in about 24–36 days (Levine 1968). For *H. isolonche* and possibly other species, larvae penetrate the cecal mucosa where they develop into adults (Schwartz 1924). They are often found encapsulated on the cecal wall (Schwartz 1924; Callinan 1987; Balaguer et al. 1992).

The eggs of *A. galli* hatch in the proventriculus and upper intestinal tract and larvae live in the lumen of the duodenum and jejunum for about 9 days (Ackert 1931; Ruff 1984). They subsequently invade the spaces between the intestinal villi and penetrate the mucosa (Ackert 1931). Larvae live in the intestinal mucosa for

about 17 days before they move into the intestinal lumen to mature (Ackert 1931). Maturation of *A. columbae* takes about 35–40 days in Rock Pigeons (*Columba livia*) (Wehr and Hwang 1964). By contrast, maturation times for *A. galli* and *A. dissimilis* range from 28 to 30 days (Wehr 1942; Ruff 1984).

Ectopic migration has been reported for several species of ascarid nematodes including *A. columbae* in Rock Pigeons (Wehr and Shalkop 1963; Wajihullali et al. 1982), *A. dissimilis* in domestic turkeys (Norton et al. 1999), and *Ascaridia perspicillum* (=*Ascaridia galli*) in Indian Peafowl (*Pavo cristatus*) (Rao et al. 1981). Larvae of *A. columbae* have been found in the liver, intestinal mesenteries, lungs, and gizzard lining (Wehr and Hwang 1964; Wajihullali et al. 1982), whereas larvae of *A. dissimilis* have been found in the liver, bile duct, and portal veins (Norton et al. 1999). Larval migration may be the result of overcrowding in the intestines (Wajihullali et al. 1982). Larvae of *A. columbae* and *A. dissimilis* fail to develop in these tissues, suggesting this is not a normal pathway for larval development (Wehr and Hwang 1964; Wajihullali et al. 1982; Norton et al. 1999). However, several adult *A. perspicillum (=Ascaridia galli*) have been found in the bile duct of an Indian Peafowl chick (Rao et al. 1981), which suggests they either migrated as adults from the intestinal tract or reached the duct as larvae and subsequently matured.

Eggs subjected to normal environmental conditions can remain viable for extended periods. Farr (1956, 1961) reported that the eggs of *H. gallinarum* remained infective in soil at a Maryland, USA, outdoor test facility for up to 229 weeks (4.4 years) while eggs of *Ascaridia* spp. (likely *A. galli* and/or *A. dissimilis*) remained viable for up to 136 weeks (2.6 years). A temperature of 57°C was lethal to eggs of *H. gallinarum*, although eggs of *A. galli* survived slightly higher temperatures (Christenson et al. 1942). Eggs of *Ascaridia lineata* (=*Ascaridia galli*) were killed when exposed to a temperature of 43°C for 12 h or a temperature of about −12°C for 22 h (Ackert 1931).

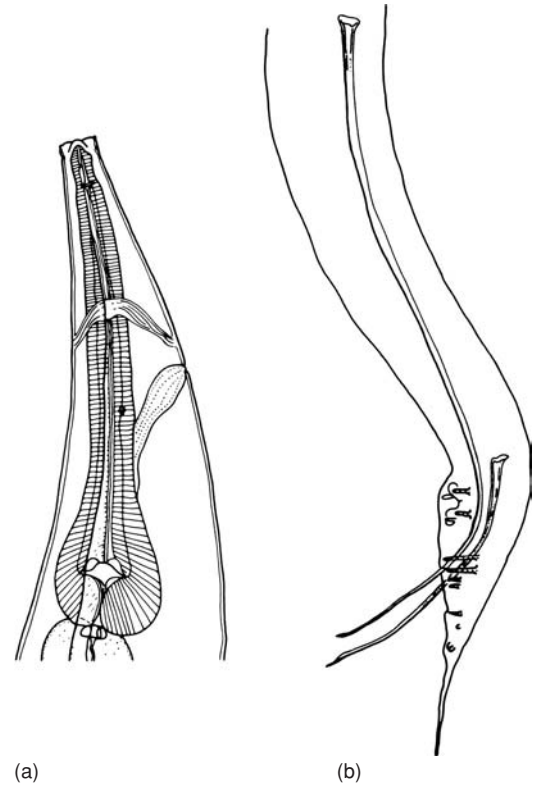


Figure 23.2. *Heterakis gallinarum* head (a) and tail (b). Modified from Ruff (1984) *Diseases of Poultry,* 8th ed., after Skrjabin and Shikhobalova (1949) and Lane (1917), respectively, and reproduced with permission from Blackwell Publishing.

Prevalence and Intensity

The prevalence and intensity of infections with *Heterakis* and *Ascaridia* vary widely, ranging from 0 to 100% prevalence and intensities of up to thousands of worms within a single host. Differences in intrinsic factors such as host resistance, immunity, co-occurring susceptible host species, behavior, diet, age, and sex and extrinsic environmental factors such as season, precipitation, and geographic location may explain this variability. For example, prevalence and intensity of infection of *H. gallinarum* in Ring-necked Pheasants have ranged from 78 to 100% and from 1 to 2,116 worms, respectively, at a hunting estate in Great Britain (Draycott et al. 2000). By contrast, prevalence and intensity in Ring-necked Pheasants in the upper Midwest US have been much lower (Gilbertson and Hugghins 1964; Greiner 1972).

Some studies have shown a host age effect. For example, higher prevalence of *H. gallinarum* was found in juvenile Greater Prairie-Chickens (*Tympanuchus cupido*) than in adults (Harper et al. 1967). Prevalence of infection in other host species may be higher in adult birds. Examples include *H. gallinarum* in Ring-necked Pheasants (Gilbertson and Hugghins 1964), *Heterakis bonasae* (=*Heterakis isolonche*) in Northern Bobwhites (Blakeney and Dimmick 1971), *A. columbae* in White-winged Doves (*Zenaida asiatica*) (Glass et al. 2002), and *Ascaridia compar* in Willow Ptarmigan (*Lagopus lagopus*) (Wissler and Halvorsen 1977).

Prevalence and intensity of infections may vary between host sexes. For example, prevalence of *H. gallinarum* was higher in male Greater Prairie-Chickens (Harper et al. 1967), whereas prevalence of *H. bonasae* (=*Heterakis isolonche*) was similar between male and female Ruffed Grouse, but mean intensity was higher in females (Kalla et al. 1997). Others have reported no relationship between host sex and infections of *H. isolonche* in Northern Bobwhites (Moore et al. 1987) or of *A. columbae* in White-winged Doves (Glass et al. 2002).

The effect of season has been examined in some detail. Prevalence and intensity of *H. gallinarum* was higher in spring than fall in Wild Turkeys (McJunkin et al. 2003). Prevalence was similar among seasons for *H. bonsae* (=*Heterakis isolonche*) in Ruffed Grouse, but mean intensity was highest in February and lowest in October–December (Kalla et al. 1997). Prevalence of *A. compar* in Willow Ptarmigan and Eurasian Capercaillie (*Tetrao urogallus*) was highest in summer and fall and lowest in winter (Lund 1946, reporting data from Hülphers 1930; Wissler and Halvorsen 1977).

CLINICAL SIGNS

Signs of heterakiasis and ascariasis in wild birds have not been adequately described, probably because moribund individuals are more susceptible to predation and thus are rarely found. Nonspecific signs of heterakiasis in captive pheasants include emaciation, weakness, diarrhea, dyspnea, and terminal gasping (Helmboldt and Wyand 1972; Balaguer et al. 1992). Nonspecific clinical signs described as unthriftiness have also been observed in Rock Pigeons that had heavy infections of *A. columbae* (Wehr and Shalkop 1963).

PATHOLOGY

Little information is available regarding pathological responses of wild birds to heterakid and ascarid nematodes. Much of our understanding about pathological responses comes mainly from poultry and captive wild birds, particularly pheasants, which seem highly susceptible to several species of *Heterakis*. The ceca of Ring-necked Pheasants infected with *H. gallinarum* may exhibit congestion, thickening and petechiation of the mucosa, intussusception, and cecal abscesses (Menezes et al. 2003).

Nodular typhlitis has been reported in pheasants infected with *H. gallinarum* (Menezes et al. 2003) and *H. isolonche* (Schwartz 1924; Griner et al. 1977; Callinan 1987; Balaguer et al. 1992). Nodules appear pale white or pink to dark brown and are approximately 1–8 mm in size (Griner et al. 1977; Menezes et al. 2003). A host inflammatory response is evident during early nodule development when heterakid nematodes become surrounded by lymphocytes, macrophages, and fibroblasts (Callinan 1987; Balaguer et al. 1992). Older nodules containing degenerated heterakids have a preponderance of epithelioid cells, plasma cells, and giant cells (Balaguer et al. 1992).

A pathological response to ascarid nematodes has been reported for chickens (Ackert 1923) and Rock Pigeons (Wajihullali et al. 1982) and is likely similar in susceptible wild hosts. Ascarid nematodes irritate the mucosal lining of the intestine and can produce edema, hyperemia, hemorrhage and destruction of villi, dilation of the crypts of Lieberkühn, and infiltration by eosinophils and lymphoid cells (Ackert 1923; see reviews in Mozgovoi 1953). High intensities of adult *A. numidae* in the lumen are associated with localized excess mucus formation indicative of mild catarrhal enteritis (Matta and Ahluwalia 1979). Acute catarrhal enteritis caused by infections with *A. perspicillum* (=*Aseradia galli*) has been reported in Indian Peafowl (Rao et al. 1981).

Intestinal obstruction was reported in a Chukar (*Alectoris chukar*), caused by over 260 worms of *A. galli* (Tibbitts and Babero 1969). Intestinal dilation and obstruction have been observed in heavy infections of *A. columbae* in Rock Pigeons (Wajihullali et al. 1982).

Several species of ascarids have been known to cause nodulation in susceptible hosts. Small pinhead sized nodules have been found on the intestinal wall surrounding *A. columbae* in Rock Pigeons (Wajihullali et al. 1982). Larvae of *A. numidae* in Helmeted Guineafowl are surrounded by macrophages, some eosinophils and giant cells, and are contained within collagen fiber capsules (Matta and Ahluwalia 1979).

The host response at sites of ectopic migration varies widely. No pathological effects are associated with larvae of *A. columbae* within the gizzard lining of Rock Pigeons, while intensive cellular infiltration of leukocytes may occur around ascarid larvae in tissues of the intestine, mesenteries, liver, and lung (Wajihullali et al. 1982). Development of granulomatous lesions in the liver of Rock Pigeons infected with *A. columbae* initially involves infiltration of host lymphoid cells and some eosinophils around degenerating larvae, followed by an increase in eosinophils, and finally engulfment of the larval debris by giant cells (Wehr and Shalkop 1963). Ascarid-induced hepatic lesions and purulent cholangitis and thickening of bile duct walls resulting from infiltration of lymphocytes, plasma cells, and heterophils have been reported in Indian Peafowl (Rao et al. 1981). Severe catarrhal inflammation accompanied with hyperplasia of mucosal epithelial cells can occur at affected sites in the proventriculus (Rao et al. 1981).

DIAGNOSIS

Definitive diagnosis is by direct observation and identification of adult worms. Identification of species of *Heterakis* usually requires examination of male features because females in this genus are often indistinguishable (Maplestone 1932; Madsen 1950). Identification of species of *Ascaridia* usually requires examination of male spicules, determination of number of caudal papilla, and other distinct morphological characteristics that are described in taxonomic keys or original species descriptions. Eggs of *H. gallinarum* and *A. galli* can be distinguished from one another on the basis of size and shape (Christenson et al. 1942).

IMMUNITY

Little is known about immunity to *Heterakis* and *Ascaridia* in wild birds. While some species can cause morbidity and mortality in some hosts, few or no pathological responses are seen in others. For example, *H. isolonche* is pathogenic in pheasants, but is relatively benign in Northern Bobwhites. Similarly, while infections with *H. gallinarum* can be acquired by sympatric Ring-necked Pheasants and Gray Partridges (*Perdix perdix*), substantially higher worm intensities occur in pheasants (Madsen 1941). Whether these findings in pheasants and partridges are the result of innate or acquired immunity or simply host species differences in exposure probabilities to infective parasite stages has yet to be determined.

There is speculation that higher prevalence and intensity of infection by certain species of helminths in juvenile birds may be the result, in part, of the bird's immunological naivety (Buscher 1965; Fedynich et al. 2005). However, this may not be the case for heterakids and ascarids because numerous studies have shown higher prevalence and/or intensity of infection in adults (see Prevalence and Intensity section). Unfortunately, data from wild birds regarding immunological responses caused by heterakids and ascarids are lacking. Study of this topic is particularly problematic because immunological responses can vary between wild and domestic individuals of the same species and birds can only be reliably grouped into two (juvenile and adult) or possibly three age classes (juvenile, subadult, and adult).

PUBLIC AND DOMESTIC ANIMAL HEALTH CONCERNS

Human exposure to avian heterakid or ascarid nematodes does not appear to pose a health risk. However, there is potential for transmission between domestic and wild birds. Wild birds may serve as sources of infection for poultry flocks, and domestic birds may aide in maintaining cycles of transmission in wild birds or possibly introduce new species to wild populations (Wehr 1940a, 1942; Maxfield et al. 1963). Ascarid nematodes that negatively affect domesticated birds raised for commercial purposes include *A. dissimilis* in turkeys (Wehr 1942; Norton et al. 1992), *A. galli* in chickens (Ruff 1984) and Helmeted Guineafowl (Ayeni et al. 1983), and *A. numidae* in Helmeted Guineafowl (Matta and Ahluwalia 1979). Of these, *A. dissimilis* causes significant concern in North America because hepatic lesions caused by this ascarid worm can lead to condemnation of affected turkeys (Norton et al. 1999) and domestic flocks can experience high mortality (Norton et al. 1992).

Heterakis gallinarum is also a potential threat to domestic and wild Galliformes because it can transmit *Histomonas meleagridis*, which is the protozoan that causes histomoniasis in domestic and Wild Turkeys (Cole and Friend 1999; Forrester and Spalding 2003; Chapter 7).

WILDLIFE POPULATION IMPACTS

Our early understanding of *Heterakis* and *Ascaridia* infections was derived primarily from observations and experimental studies of individual hosts. Population-level impacts have been more difficult to assess, particularly regarding the sublethal effects of these nematodes on predation rates, host competition, and host fitness. Our knowledge of these factors is improving. Recent studies have found negative relationships between *A. compar* and body weight in juvenile Willow Ptarmigan (Daehlen 2003) and indications that parasite assemblages (including *A. compar*, *Heterakis altacia*, *H. gallinarum*, and *Heterakis tencicauda*) may influence population cycles of the Rock Partridge (*Alectoris graeca*) (Rizzoli et al. 1999).

Significant negative impacts on wild bird populations can occur when parasitized pen-raised birds are released into the wild. The introduction of new parasite species into native populations is a concern. This may have occurred when 27 Whooping Cranes (*Grus americana*) and a Sandhill Crane (*Grus canadensis*) apparently infected with *Ascaridia pterophora* were released in Florida (Spalding et al. 1996). The only previous record of this ascarid worm was from the Red-legged Seriema (*Cariama cristata*) in Brazil (Cram 1927). Introduced birds can also help maintain and perpetuate heterakid or ascarid nematodes already present in wild populations. Negative effects of infection with *H. gallinarum* at the population level have been observed

in released stocks of pen-reared Ring-necked Pheasants in Britain, including lower host body condition (Robertson and Hillgarth 1993) and lower reproduction and survival than wild pheasants (Woodburn 1994). In Spain, there is a concern that populations of the Red-legged Partridge (*Alectoris rufa*) may be negatively affected by infected pen-reared stock released into the wild to support recreational hunting activities (Millán et al. 2004a; Villanúa et al. 2007).

Parasites may play important roles in facilitating or inhibiting host species invasions and may have significant negative effects on native host species when unintentional introductions of exotic parasites occur (Prenter et al. 2004). Several studies conducted in the UK suggest that maintenance of *H. gallinarum* in populations of the introduced Ring-necked Pheasant has negatively impacted the native Gray Partridge (Tompkins et al. 2000, 2001, 2002). Heterakid and ascarid nematodes can also be spread to new geographic regions via the pet trade (Webster 1982), via acquisition of exotic birds for zoological gardens (Tables 23.1 and 23.2), via human introductions of exotic birds into native ecosystems, and from natural range expansions of infected hosts. The magnitude of this problem is evident in the current worldwide distribution of psittacine birds and their ascarid parasites as well as the number of infected bird species that occur outside their natural geographic ranges (Tables 23.1 and 23.2).

TREATMENT AND CONTROL

Wildlife management agencies have not actively pursued preventative treatment and control activities for heterakid and ascarid nematodes in wild bird populations, likely because of practicality and cost. It is unclear whether broad-scale treatment and control activities have any long-term benefits to wild bird populations. However, for captive breeding programs involving game birds and threatened or endangered species, treatment and control programs are essential, and they typically follow procedures used for poultry. Treatment and control measures are designed to disrupt the transmission cycle of the heterakids and ascarids. Recommendations for captive birds include routinely disinfecting feeders and watering troughs, and raising birds on hardware cloth to prevent birds from contacting contaminated soil and feces (Mozgovoi 1953; Levine 1968).

Prevention of cross infection between domestic and wild Galliformes is an important consideration in captive situations. For example, Greater Prairie-Chickens and Sharp-tailed Grouse (*Tympanuchus phasianellus*) should be kept from areas used by domestic poultry (Morgan and Hamerstrom 1941). Such recommendations serve to prevent situations such as that noted by Tibbitts and Babero (1969) where Chukars likely became infected by *A. galli* when they were housed in pens frequented by chickens. Additionally, plans for restocking of wild birds should consider proximity to commercial enterprises that have the same or closely related species. For example, plans for restocking Wild Turkeys should take into consideration the proximity of turkey farms to minimize the potential of cross infection (Maxfield et al. 1963).

Anthelmintics have been used extensively on captive flocks of chickens and turkeys and they should be effective for wild or captive wild species. The U.S. Food and Drug Administration approves specific anthelmintics that are used in birds intended for human consumption (i.e., eggs or meat) and currently authorizes fenbendazole, phenothiazine, and hygromycin B for poultry (Kahn 2006). Other drugs include tetramisole and levamisole for controlling *H. gallinarum* and *A. galli* (Kahn 2006) and piperazine dihydrochloride for controlling *A. galli* (Nilsson and Alderin 1988). Additionally, levamisole has been used to control *A. columbae* in captive flocks of Rock Pigeons (Panigrahy et al. 1982). Other possible anthelmintics for birds may include albendazole, oxfendazole, and ivermectin (Dawe and Hofacre 2002). Ivermectin is effective in eliminating *H. gallinarum* and *A. galli* from flocks of Helmeted Guineafowl (Okaeme 1988). However, albendazole and fenbendazole can cause toxicosis when administered to captive columbids (Howard et al. 2002). One study found that albendazole (at the dose administered) was not satisfactory in substantially reducing *H. gallinarum* in farm-reared Red-legged Partridges that were intended for release into the wild (Villanúa et al. 2007).

There has been success in targeted field applications of anthelmintics. Several studies conducted on hunting estates in Britain have used supplemental feed treated with levamisole hydrochloride (Robertson and Hillgarth 1993; Draycott et al. 2000) and flubendazole (Woodburn et al. 2002) to reduce infections of *H. gallinarum*, thereby increasing breeding success in free-ranging Ring-necked Pheasants.

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24
Ascaridoid Nematodes: *Contracaecum*, *Porrocaecum*, and *Baylisascaris*

Hans-Peter Fagerholm and Robin M. Overstreet

INTRODUCTION

The superfamily Ascaridoidea is a medically and economically important entity made up of some 50 genera covering six families (Fagerholm 1991; Nadler and Hudspeth 1998, 2000). Adult members of few ascaridoid genera infect birds (Fagerholm 1996), and we treat in this chapter only the anisakid *Contracaecum* Railliet and Henry, 1912 (Contracaecinae) and the ascaridid *Porrocaecum* Railliet and Henry, 1912 (Toxocarinae). There are a few records of species of other anisakids in birds such as *Duplicaecum* Majumdar and Chakravarty, 1963, *Heterotyphlum* Spaul, 1927, and the ascaridid *Amplicaecum* Baylis, 1920. Some may represent accidental infections or incorrect identifications (Deardorff and Overstreet 1981b). With the exception of *Anisakis* sp. (Riley 1972), none of these species are known to harm their avian hosts or constitute a public health risk (e.g., Majumdar and Chakravarty 1963). We also treat juvenile species of the ascaridid *Baylisascaris* Sprent, 1968 (Ascaridinae) because of their detrimental influence on both birds and people.

Members of these ascaridoid genera should not be confused with those of the related superfamily Heterakoidea, such as *Heterakis gallinarum* (Schrank 1788) (see Kellogg and Reid 1970; Norton et al. 1992; Menezes et al. 2003; Chapter 23). Members of the genera *Contracaecum* and *Porrocaecum* also resemble fish nematodes of the genera *Hysterothylacium* Ward and Magath, 1916 and *Raphidascaris* Railliet and Henry, 1915 (Raphidascarididae sensu Fagerholm, 1991) (Deardorff and Overstreet 1981a, b; Nadler et al. 2007).

Although the pathogenicity of ascaridoids of birds has not been studied extensively, numerous case studies show or suggest that the host nutritional and immune status may directly or indirectly affect the severity of an ascaridoid infection. As noted below, the diseases caused by ascaridoid parasites have been referred to by a number of names depending on the species discussed.

Recent molecular studies on *Contracaecum* have regularly reported the presence of sibling species within individual morpho-species. For example, there are at least five sibling species in the *Contracaecum osculatum* complex from pennipeds (Bullini et al. 1997). This similarity among species makes identification of strains and species difficult without using molecular techniques (Nadler and Hudspeth 1998; D'Amelio et al. 2007; Mattiucci et al. 2008). Molecular studies should be used to determine the correct geographic distribution and to investigate the ecology of these and other sibling forms. Many may represent new species or strains and their potential role as pathogens in avian hosts remains unknown.

CONTRACAECUM AND *PORROCAECUM*

SYNONYMS

Infections with the adult anisakids of the genus *Contracaecum* have been referred to as nematodiasis, anisakiasis, and contracaeciasis. Infections with species of *Porrocaecum* are referred to as porrocaeciasis.

ETIOLOGY

Contracaecum contains over 60 nominal species (e.g., Yamaguti 1935; Hartwich 1964, 1974; Baruš et al. 1978) that are medium sized (some 1–8 cm long), threadlike parasites of the proventriculus, gizzard, and intestines of birds, seals, and dolphins. Whereas few synonyms of *Contracaecum* have been used recently,

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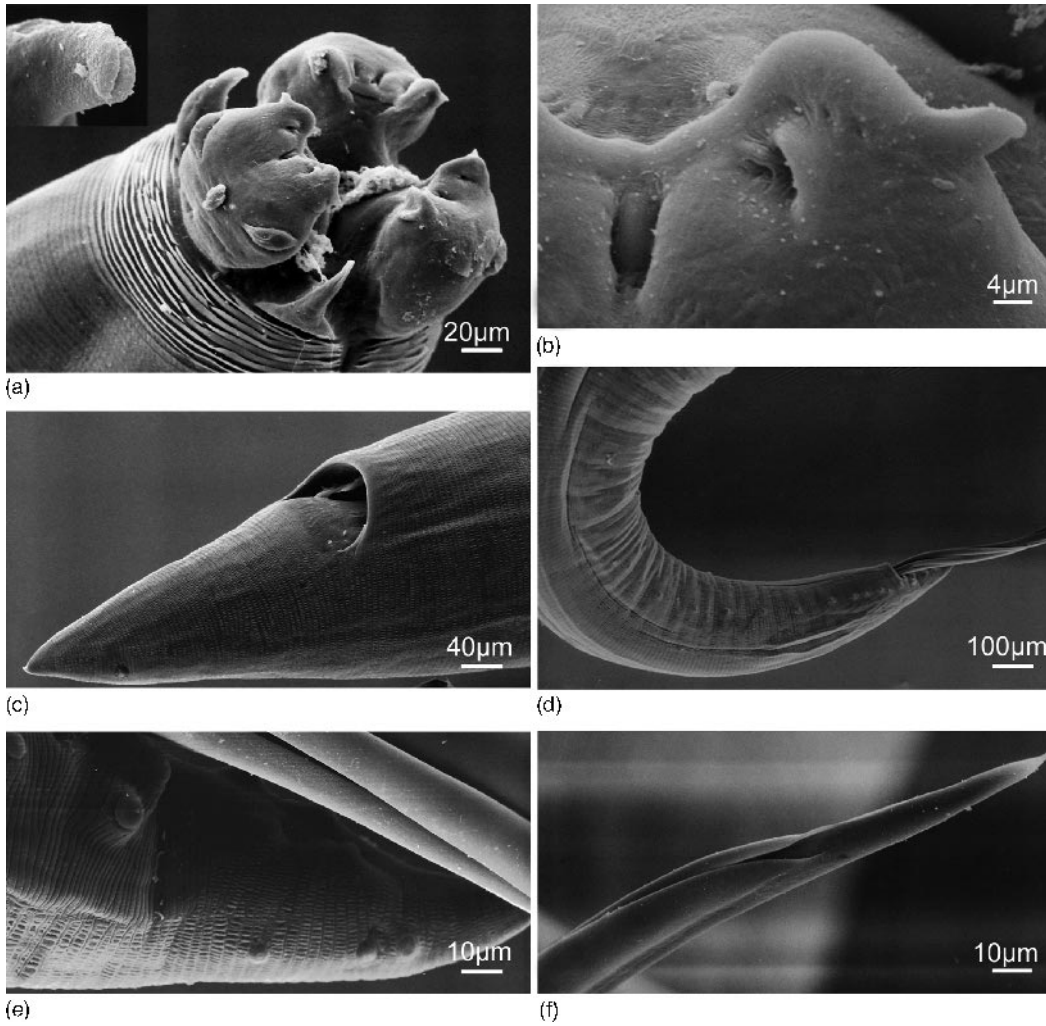


Figure 24.1. Structure of *Contracaecum variegatum* (SEM photographs). (a) Anterior end, subdorsal view, with narrow interlabia, with bifid apex (see enlarged insert). (b) Detail of labium with knoblike lateral auricle. One of the six inner labial sense organs, situated laterally on lips, is located in pit in the center of image. (c) Female tail. A subdorsal left phasmid is located near the tip of the tail. (d) Male tail with a row of subventral proximal papillae. Spicules are extruded from the cloacal orifice. (e) Enlargement of the male tail. Two separate paracloacal papillae and four distal papillae and a phasmid can be seen on the tail. The distribution of the caudel sense organs is close to that of *C. magnipapillatum* (Fig 24.5). (f) Distal end of a spicule. The extended sharp point is demarcated by the sperm channel, two winglike structures that form a tube. Modified in part from Fagesholm et al. (1996) with permission of *Helminthologia*.

some fish raphidascaridids now accepted in the genus *Hysterothylacium* (Deardorff and Overstreet 1981a) are still identified in some nontaxonomic literature as species of *Contracaecum* (Fagerholm 1978, 1982, 1988b). Concepts of the phylogeny of ascaridoid nematodes and of the systematic position of the superfamily Ascaridoidea within the order Rhabditida have been reviewed recently by De Ley and Blaxter (2002) and Nadler et al. (2007). Studies based on genetic features corroborate clades obtained on the basis of structural data (Fagerholm 1991; Nadler et al. 2000, 2005).

Species of *Contracaecum* have been defined on the basis of distribution of caudal sense organs (papillae) in the male (Fagerholm 1988a) as well as numerous

other structural features of the adult worms (Figure 24.1). No fewer than 30 fully grown adult worms of each sex should be studied by light microscopy to make identifications because of intraspecific variability in morphological features. Material for genetic analysis and scanning electron microscopy (SEM) should also be collected. While caudal features generally differentiate species from seals from those that occur in birds, morphological data suggest that host-switching might have occurred from birds to seals for some species. For example, *C. ogmorhini* Johnston and Mawson, 1941 *sensu lato* of ottarid seals has morphological features that are very close to *C. rudolphii* Hartwich, 1964 *sensu lato* from birds (Fagerholm and Gibson 1987), suggesting that this seal parasite originated from birds. By contrast, there is little evidence to suggest that species of *Contracaecum* in birds may have evolved from species that occur in seals.

The genus *Porrocaecum* contains over 30 described species that infect the intestine of birds. Good descriptions of common species are given by Baruš et al. (1978) and Anderson (2000). Many of these require revision. By definition, species of *Porrocaecum* differ from *Contracaecum* by the absence of a ventricular appendix. They have only small interlabia and they also have a delicate denticulated ridge on the lips that differs from the denticles of *Phocascaris*, a genus systematically close to *Contracaecum* (see Berland 1963; Paggi and Bullini 1994) (Figure 24.2). The spicules of male

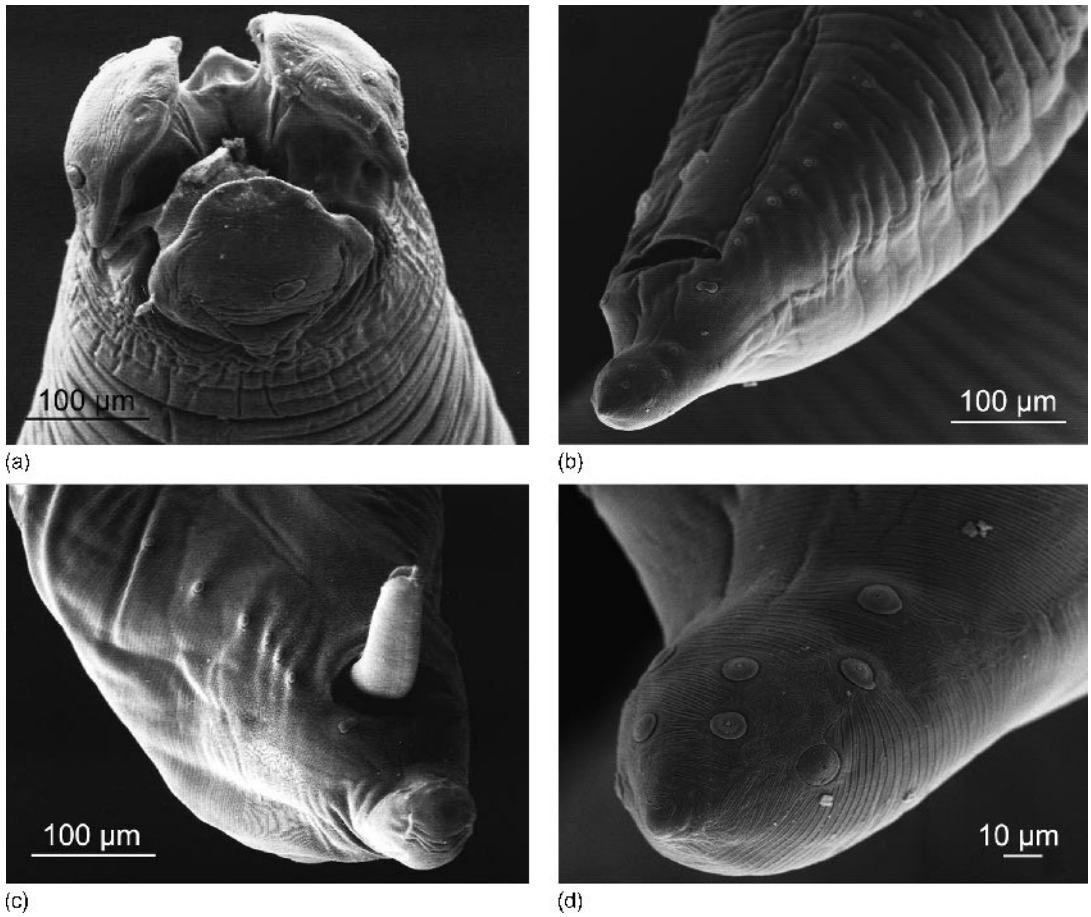


Figure 24.2. *Porrocaecum depressum* (SEM photographs) from the stomach of a Northern Goshawk (*Accipiter gentilis*). (a) Anterior lateral view. (b) Male tail with a subventral row of proximal caudal papillae. These serve as sense organs. (c) Male tail with extruded spicule. The tip of the male tail is modified into a knob. (d) Enlargement of the distal end of the male tail. Note left four distal papillae and phasmid (lowermost dimple, with minute opening).

specimens of *Porrocaecum* are usually short (1 mm), while those in male specimens of *Contracaecum* range from 2 to 12 mm in length. Species of *Porrocaecum* have been differentiated on the basis of the presence or absence of cervical alae and on the projections of the lip pulp (Osche 1955; Baruš et al. 1978; Digiani and Sutton 2001).

Host Range and Distribution

Species of *Contracaecum* are generally very common in fish-eating birds and have a wide host range and geographic distribution (Figure 24.3). In some aquatic birds, at least four different species of the genus may occur concurrently in the same individual. Common avian species are *Contracaecum magnipapillatum* Chapin, 1925 (synonym *C. magnicollare* Johnston and Mawson, 1941) from the proventriculus of migratory terns such as the Black Noddy (*Anous minutus*) that range throughout the Pacific Ocean (Fagerholm et al. 1996). *Contracaecum variegatum* (Rudolphi, 1809) from the Pacific Loon (*Gavia pacifica*) and *Contracaecum himeu* Yamaguti, 1941 from cormorants in Japan were recently redescribed (Nagasawa et al. 1999a, b). *Contracaecum microcephalum* (Rudolphi, 1809) has been described from all main continents from cormorants (*Phalacrocorax* spp.), pelicans (*Pelecanus* spp.), ducks, swans, and geese (Anatidae), and herons, egrets, and bitterns (Ardeidae) (Yamaguti 1935; Hartwich 1964). Many of these records may represent a complex of sister cryptic species similar to those described for *Contracaecum multipapillatum* (*Contracaecum multipapillatum* (Von Drasche, 1882)) *sensu lato* from the proventriculus and esophagus of pelicans, herons, cormorants, storks, and Anhinga (*Anhinga anhinga*) in North America, including Mexico and Cuba (Deardorff and Overstreet 1980b). Other common avian species include *Contracaecum pelagicum* Johnston and Mawson, 1942 from the Magellanic Penguin (*Spheniscus magellanicus*) and numerous other fish-eating birds (Fagerholm et al. 1996; Garbin et al. 2007).

Contracaecum rudolphii is considered to be the most common species of *Contracaeacum* in birds and has been reported from birds in the Neotropics, Nearctic, Palearctic, Ethiopian, and Australian regions (Johnston and Mawson 1941; Whitfield and Heeg 1977; Baruš et al. 1978; Torres et al. 1983; Amato et al. 2006). In the Palearctic Region, *C. rudolphii sensu lato* has been reported from 58 species of birds in 24 genera (Baruš et al. 1978). Also considered a senior synonym of *C. spiculigerum* by Huizinga (1971) and others (Deardorff and Overstreet 1980b), *C. rudolphii* occurs in the proventriculus of pelicans, herons, mergansers, and cormorants. Like many other species, numerous individuals are frequently found embedded in the proventriculus (Thomas 1937).

The taxonomic status of *C. rudolphii* needs to be reconsidered since it comprises a complex of at least four genetically isolated sibling forms that are morphologically similar (Bullini et al. 1986; Cianchi et al. 1992; Mattiucci et al. 1994; Li et al. 2005; D'Amelio et al. 2007; Szostakowska and Fagerholm 2007). One, *Contracaecum septentrionale* (Kreis, 1955) has been shown to be an accepted species that occurs mainly at higher latitudes (Li et al. 2005). Similarly, *Contracaecum multipapillatum* from Florida may comprise at least two genetically isolated sibling types (D'Amelio et al. 2007; H. Ma and R. M. Overstreet, unpublished observations). In Australia and Europe, additional sibling species are present (Cianchi et al. 1992; Mattiucci 2006; S. Shamsi, personal communication).

Both *Contracaecum magnipaillatum* and *C. variegatum* (Rudolphi, 1809) are structurally similar to *C. pelagicum* Johnston and Mawson, 1942 (see Fagerholm et al. 1996). Genetic studies are needed to define further these and other morphologically similar species and their host range and distribution.

Species of *Porrocaecum* are cosmopolitan in distribution, but there is a clear need to review their taxonomic status using both structural and modern genetic methods. Common species include *Porrocaecum ensicaudatum* (Zeder, 1800), a cosmopolitan parasite of passerine birds (Figure 24.4) and *Porrocaecum angusticolle* (Molin, 1860) from the intestines of raptors from throughout the world. *Porrocaecum depressum* (Zeder, 1800) has a similar cosmopolitan distribution and has been reported from approximately 47 species of birds of prey, although there is considerable morphological variation in material collected from owls (Morgan and Schiller 1950). *Porrocaecum crassum* (Deslongchamps, 1824) occurs in European waterfowl, especially ducks (Figure 24.4).

Epizootiology

Among species of ascaridoids that have been studied, third-stage (L3) larvae hatch from fully embryonated eggs that are passed in the feces of their avian definitive hosts. These are ingested by a crustacean intermediate host and penetrate into the body cavity of these invertebrates. Fish can act as paratenic hosts when they ingest infected crustaceans. The L3 larvae encyst within the intestinal wall, mesentery, liver, or other internal organs of fish, but are not known to occur within muscle tissue. When fish (or crustaceans) are ingested by a suitable avian host, the L3 larvae are released and undergo final molts to fourth-stage (L4) larvae and adults within the proventriculus of the avian host. Under some conditions, fish may serve as a

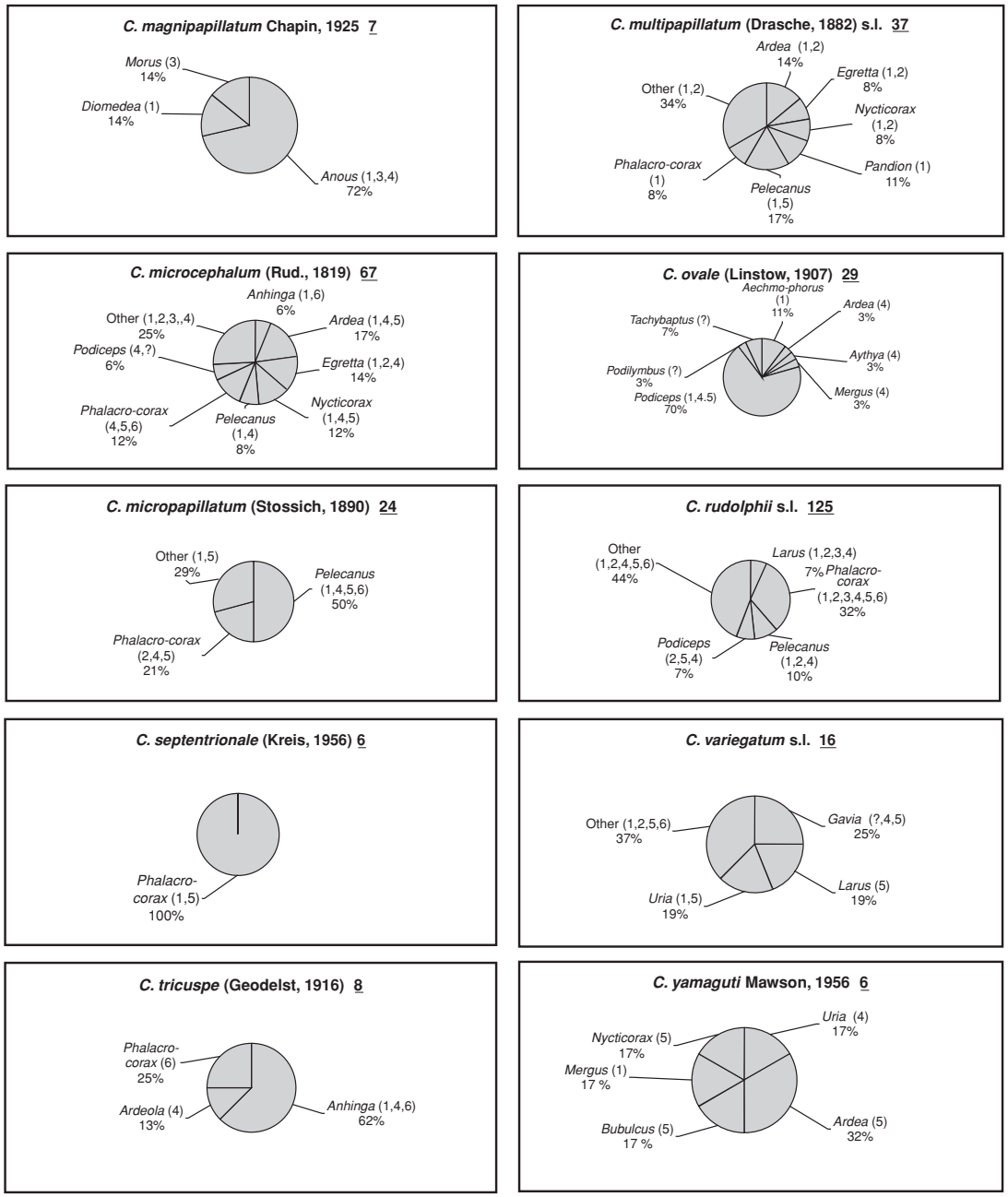


Figure 24.3. Host range of species of *Contracaecum.* Diagrams are based on published data on percentage of recorded cases of *Contracaecum* from different genera of birds. Information was retrieved from Gibson et al. (2005). The category "Other" represents records of different birds only occasionally reported to be infected. Number of cases is underlined in the heading. The origin (geographical area) of records is shown in parenthesis next to the genus name: 1, North America; 2, South America; 3, Australia and New Zealand; 4, Asia (including India and Japan); 5, Europe; 6, Africa. Species recently (re)described include *Contracaecum bioccai* Mattiucci et al., 2008 (in *Pelicanus*); *Contracaecum eudyptulae* Johnston et Mawson, 1942 (in *Eudyptula*); and *Contracaecum pelagicum* Johnston et Mawson, 1942 (in *Diomedea*). Recently defined genetically isolated strains of *Contracaecum rudolphii sensu lato* are not included.

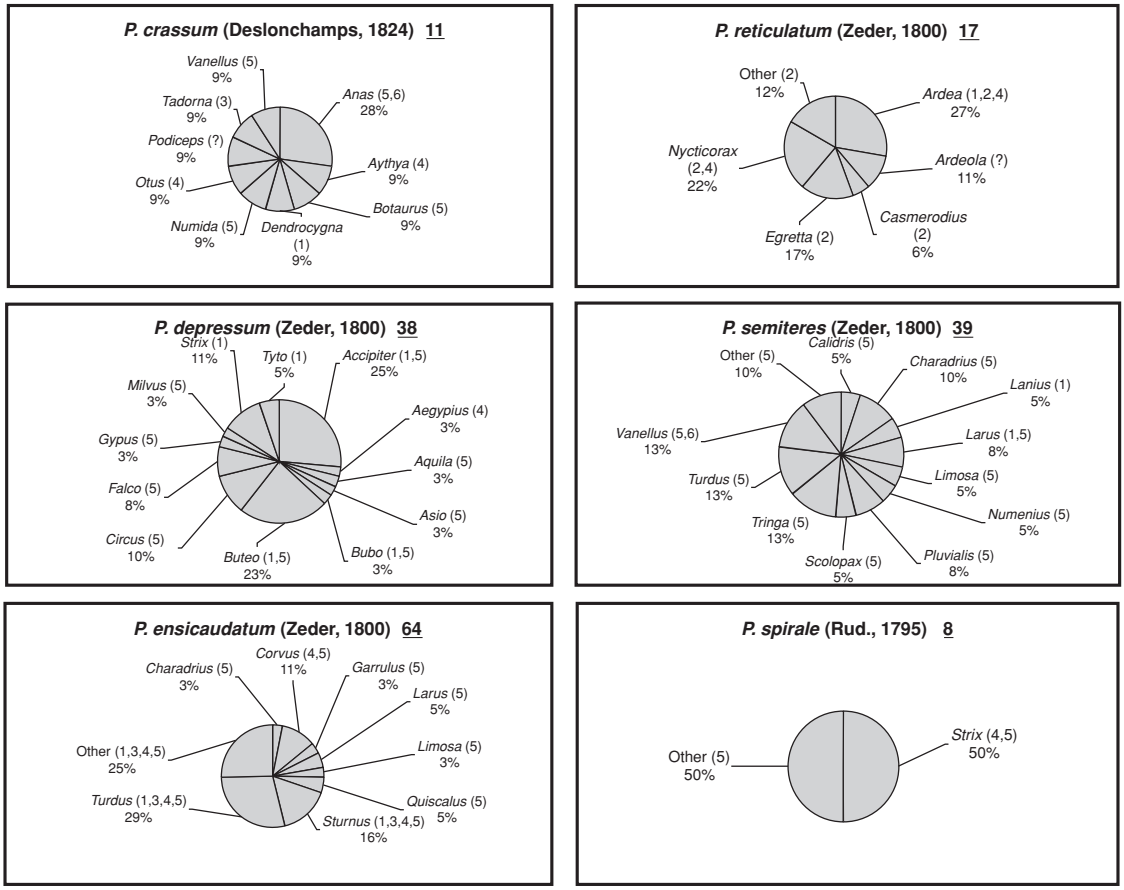


Figure 24.4. Host range of species of *Porrocaecum*. Diagrams are based on published data on percentage of recorded cases of *Porrocaecum* from different genera of birds. Information was retrieved from Gibson et al. (2005). The category "Other" represents records of different birds only occasionally reported to be infected. Number of cases is underlined in the heading. The origin (geographical area) of records is shown in parenthesis next to the genus name: 1, North America; 2, South America; 3, Australia and New Zealand; 4, Asia (including India and Japan); 5, Europe; 6, Africa. ? indicates absence of locality data.

true intermediate host and directly transmit the infection (Huizinga 1966, 1967; Køie and Fagerholm 1995; Bartlett 1996; Dziekońska-Rynko and Rokicki 2007; Szostakowska and Fagerholm 2007; R. M. Overstreet, unpublished observations). Direct "accidental" transmission of adult worms along with regurgitated food from the parent birds to the chicks is an additional important route for infection (Huizinga 1971; Fagerholm et al. 1996; Kuiken et al. 1999).

Many species of fish-eating birds serve as definitive hosts, with some species of *Contracaecum* being highly specific to their avian host and others infecting a wide range of hosts (Figure 24.3). Since some worms survive for only about 90 days (Huizinga 1971), the total number depends on continual ingestion of infected hosts. Because of the high percentage of *C. multipapillatum* in mixed infections, a primary host prey in Mississippi and Louisiana is presumably mullet (Deardorff and Overstreet 1980b).

In northern Europe, very intense infections of *C. rudolphii* have been observed in the Great Cormorant (*Phalacrocorax carbo*) (Zuchowska 2000; Kijewska et al. 2002; Szostakowska et al. 2002). Anderson (2000) and Moravec (1994) reviewed the biology of juvenile stages of *C. rudolphii sensu lato* in fishes. As many as 500 L3 larvae of *C. rudolphii* have been recorded in a single fish. When intense infections occur in fish, their market value may be reduced for esthetic reasons (Szostakowska and Fagerholm 2007).

In contrast to *Contracaecum*, the life cycle of species of *Porrocaecum* is terrestrial and includes an earthworm intermediate host. After earthworms ingest larvae from fully developed eggs, they reach the main blood vessels of the oligochaete where they remain. Some species, such as *P. depressum* and *P. angusticolle* (Molin, 1860) from birds of prey (Osche 1959), use small mammals as paratenic hosts and the larvae are found encysted in the mesentery and on the intestine and reach their avian definitive hosts when these tissues are consumed (Osche 1955; Erkinaro and Heikura 1977). Among other species, no paratenic host is involved as avian hosts may consume earthworms for food. This is the case for both *P. crassum* and *P. ensicaudatum*. The biology of the latter species has been studied by several authors. Jögis (1970) and also McNeill and Anderson (1990a, b) found that *P. ensicaudatum* is encountered only in passerine birds and that mature worms are found naturally only in the avian families Sturnidae and Turdidae (Figure 24.4). Juveniles can occur under the lining of the gizzard and in the wall of the duodenum of the final avian host, but apparently most adults occur both free and embedded in the intestine (Mawson 1956).

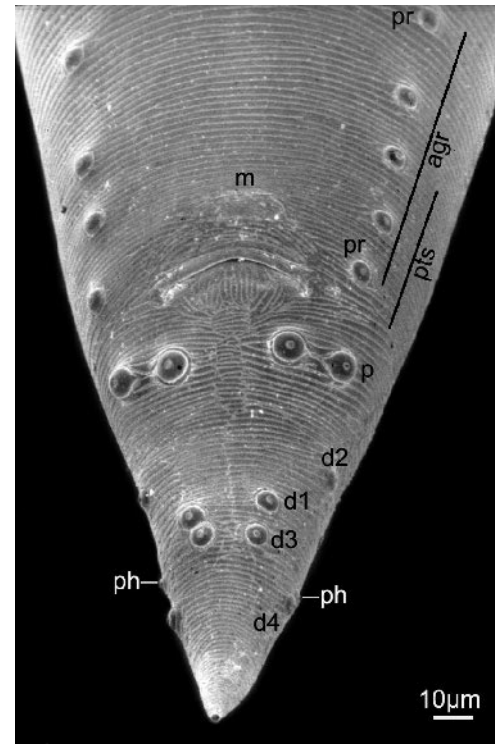


Figure 24.5. Scanning election micrograph (SEM) of the tail of a male specimen of *Contracaecum magnipapillatum* from the proventriculus of the Black Noddy (*Anous minutus*), Heron Island, the Great Barrier Reef, Australia. The distribution of the caudal papillae is an important taxonomic character. The proximal papillae (pr) are located in this species anterior to cloacal orifice as two parallel rows. The paracloacal papillae (p) are located posterior to the cloacal orifice as two large pairs that are not joined. Four pairs of distal papillae (d1, d2, d3, and d4) are located on each side of the tail. One pair of phasmids (ph) is located between the outer lateral pair of distal papillae. The left phasmid (on right in figure) is very close to the posterior most distal papilla. A precloacal median sensory area (m) is also present. The "pts zone" (precloacal tranverse striae zone) represents 25 transverse striae that start at the cloacal orifice and extend anteriorly, covering three papillae in this figure. The "aggregation zone" (agr) covers a straight row of five proximal papillae on one (or both) subventral sides of the worm. In this species, the entire region comprising five proximal papillae is evident on the left subventral side of the worm.

Diagnosis

Species of different ascaridoid genera can usually be identified to genus without difficulty. However, differentiation of species within genera may be challenging because of the often-small structural differences among species (Figure 24.5, Table 24.1). This is true for several species of the genus *Contracaecum* and is certainly the case for sibling species that cannot be distinguished by structural features alone. In these situations morphological features can be used to show that worms belong to a certain species group, but biochemical methods are needed for a correct identification. One must take into account the process of allometric growth where key morphological features such as the relative location of the vulva and spicule length continue to change until the worm has attained full length (Fagerholm 1989). The centrid, an asymmetrically oriented pair of lateral papillae in the mid-body region, may also change position as a result of allometric growth (Fagerholm et al. 1996, 1998). During growth, the length of the spicules increases from the anterior (basal) part of these structures (Fagerholm 1989), and one can argue that structural features of the point of the tip are the most conserved (Figure 24.1d). One should be careful not to rely too strongly on total spicule length if measurements are not consistent among a considerable number of fully grown worms. With recent advances in molecular methods, we have the opportunity to investigate whether

Table 24.1. Selected caudal features of diagnostic interest in adult male *Contracaecum* Railliet and Henry, 1912 from seals (a–c) and birds (d–m) as amended from Fagerholm (1988a), with new data (c.f. Figure 24.1).

	Seals			Birds									
	(a) *Contracaecum* spp. from seals, *but not*:	(b) *Contracaecum ogmorhini sensu lato*	(c) *Contracaecum radiatum*	(d) *Contracaecum bioccai*	(e) *Contracaecum magnipapillatum*	(f) *Contracaecum microcephphalum*	(g) *Contracaecum micropapillatum*	(h) *Contracaecum multipapillatum*	(i) *Contracaecum rudolphii sensu lato*	(j) *Contracaecum pelagicum*	(k) *Contracaecum septentrionale*	(l) *Contracaecum tricuspe*	(m) *Contracaecum variegatum sensu lato*
Distribution of proximal caudal papillae (numerous)													
Pts zone (region of 25 precloacal striae)													
6–17	+	−	−	−	−	−	−	−	−	−	−	−	−
2–3	−	+	+	+	+	+	+	+	+	+	+	+	+
Aggregation (region of row of 5 precloacal papillae)													
6–15	+	−	+	−	−	−	−	−	−	−	−	−	−
5	−	+	−	+	+	+	+	+	+	+	+	+	+
Number present posterior to cloaca													
0	−	+	−	+	+	+	+	−	+	+	+	+	+
1–21	+	−	+	−	−	−	−	+	−	−	−	−	−
Distribution of paracloacal papillae (2 pairs)													
Pairs joined	+	−	+	−	−	+	−	+	−	−	−	+	+
Pairs separate	−	+	−	+	+	−	+	−	+	+	+	−	−
Distribution of distal papillae (4 pairs)													
Anterior 2 pairs joined on both sides	−	−	−	−	−	−	+	−	−	−	−	−	−
Posterior 2 pairs joined on both sides	−	−	−	−	−	−	+	−	−	−	−	−	−
Size of defined caudal papillae*													
Papillae, size													
Papillae, relative size													
Length of transverse striae in cloacal region													
diminutive	−	−	+	−	−	−	−	−	−	−	−	−	−
Spicules†													
Distal end of spicules													
Points pointed	−	−	−	−	+	−	−	−	−	+	−	+?	+
Points rounded	+	+	+	+	−	+	+	+	+	−	+	−	−
Spicule length													
Spicule relative length													

Note: Selected somatic features of taxonomic interest include structures of the anterior end, e.g., labia, and the position of phasmids and centrids. Cervical, labial, and interlabial features are useful in defining some species, e.g., the elaborate structure of *C. tricuspe* (Geodelst, 1916). The position of phasmids and centrids may also be of importance (Fagerholm et al. 1998, 2004). Important generic features that are not illustrated include the position of the excretory pore just posterior to the ventral interlabium, a short ventricle and the presence of "contracaeca" (an anteriorly directed intestinal caecum and posteriorly directed ventricular appendix).

*In addition to the pattern of distribution, the size (diameter or (length + width)/2) of caudal papillae can be used as a taxonomic criterion.

†In addition to the form of the distal end of the spicules, the length of the tip can be used as a taxonomic criterion, provided that differences are statistically significant. Spicule length is often used to separate species. In this case, different growth-related factors as well as intraspecific variability need to be considered.

genetically isolated populations are also morphologically distinct. Other structural features that have been used to distinguish species or groups of species within this genus include features of the cuticle and patterns of caudal papillae (Table 24.1) (Fagerholm 1988a, 1990).

CLINICAL SIGNS

Infections with adult ascaridoids that belong to the genera *Contracaecum* and *Porrocaecum* usually produce no severe disease or clinical signs in birds. Starvation, however, with its associated signs, may be a contributory cause of death in infections of high intensity. This is frequently observed during necropsy of aquatic birds with *Contracaecum* sp. (Deardorff and Overstreet 1980b) and wild raptors (kestrels) infected with *P. depressum* (Keymer et al. 1981). Ruffled feathers and an inability to maintain balance in passerine birds have been attributed to infections with *P. ensicaudatum* (Arnall and Keymer 1975).

PATHOGENESIS AND PATHOLOGY

Infections of ascaridoids in the alimentary tract can produce a severe inflammatory response, especially when juvenile worms of some species embed and migrate within the walls of the proventriculus, esophagus, or intestine (Figure 24.6). While documented for *Contracaecum*, detailed studies of host responses to species of *Porrocaecum* in birds have not been done. Infections can cause anemia and may lead to actual disease when the bird becomes stressed. Highly pathogenic cases usually involve high-intensity infections in nestlings or young juveniles, starved individuals, birds that are stressed by environmental contamination or other causes, and birds with peritonitis or secondary microbial infections that involve other vital organs. Whereas peritonitis can result in death, the direct pathogenic effect by the worms in most cases is probably low (Deardorff and Overstreet 1980b; Greve et al. 1986).

Gross lesions caused by several species of *Contracaecum* are similar and consist of erosive, often yellow ulcers in the mucosal wall of the proventriculus and adjacent portion of the esophagus. These are associated with both petechial and larger hemorrhages. Both adult and juvenile worms attach or embed in clusters within these small to large ulcers as well as individually in isolated ulcers (Figure 24.7). They also remain free in the lumen. Worms are not always present in ulcers and experimental studies indicate that these are scars from previous worm attachments (Fagerholm 1988b). Embedded juveniles seem to be the most pathogenic stage of infection to the host. In an 18-year-old cormorant maintained for fishing in Sichuan Province, China, many additional nodules were also in the muscular layer and projected from the serosal surface (Sarashina et al. 1987).

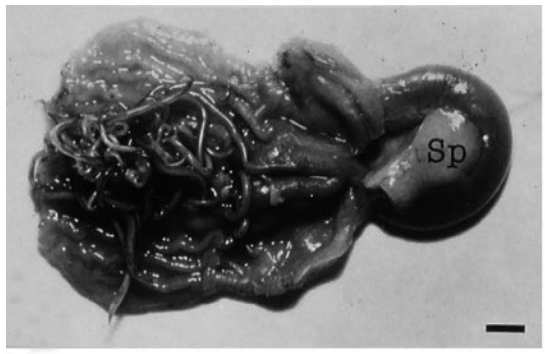
Focal mucosal ulcerations containing attached worms occur in some fish, reptiles, and marine mammals infected with other ascaridoid genera (Deardorff and Overstreet 1980a; R. M. Overstreet, personal observations). In some avian cases, especially those with only a few worms, all the individuals are free in the proventriculus or intestine and ulceration is not evident (Huizinga 1971; Deardorff and Overstreet 1980b).

Microscopic histopathological alterations caused by *Contracaecum* include compression of the glandular mucosa and ulceration with inflammatory infiltration. An eosinophilic hyaline cap often surrounds the anterior end of worms at the host–parasite interface, especially around juveniles and young adults. The tissue interface is usually a nodular area that typically includes necrosis, granulomatous inflammation, and an abundance of bacteria. Fibrosis is present and usually more abundant in chicks. The lesion can be extensive in the submucosa and muscularis mucosa. In chicks, the nodule may extend through the muscularis externa and adventitia with associated inflammatory exudates, but in older birds the nodule seldom extends farther than the muscularis interna. Recent infections may exhibit lymphocyte infiltration, and all infections include a variety of inflammatory cells, including lymphocytes, plasma cells, heterophils, macrophages, fibroblasts, and even multinucleated giant cells. The latter often occur associated with degenerating hyaline caps (Huizinga 1971; Liu and Edward 1971; Greve et al. 1986; Fagerholm et al. 1996; Kuiken et al. 1999).

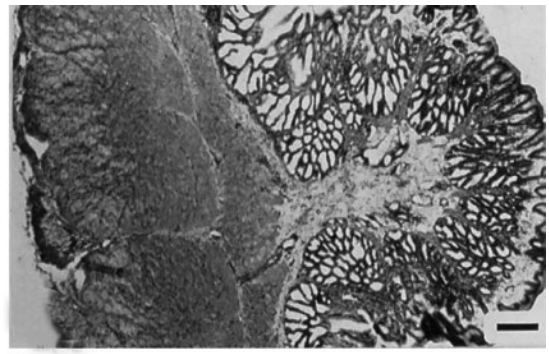
IMMUNITY

Relatively little is known about immunity to infections with *Contracaecum* and *Porrocaecum*. There is evidence that prevalence and intensity of infection with *Contracaecum* declines with age, with fewer worms in fledgling pelicans than nestling birds. This may be related to change in diet as birds are exposed to fewer worms when they are old enough to select their own prey. Age immunity may also have contributed to the decline in infections (Humphrey et al. 1978). However, Kuiken et al. (1999) recorded an increase in prevalence of *Contracaecum rudolphii sensu lato* in Double-crested Cormorants (*Phalacrocorax auritus*) to 100% of postnestling chicks and adults.

An experimental in vitro study by Raybourne et al. (1983) demonstrated that excretory–secretory (E–S) products of juvenile *Anisakis* "simplex" and *Terranova* sp. had a potent inhibitory effect on rodent lymphocyte blast transformation when compared with whole



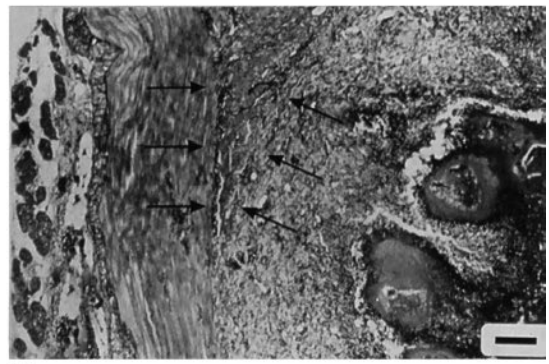
(a)



(b)



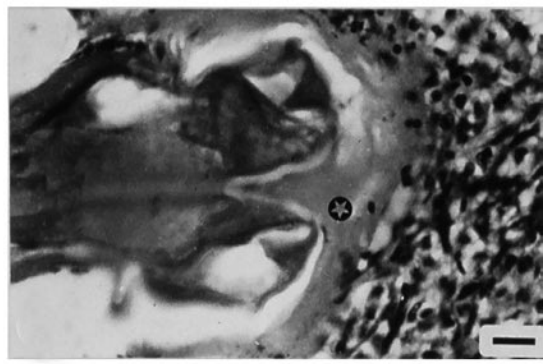
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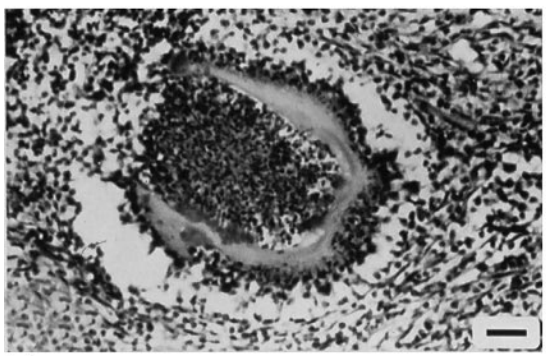
(d)



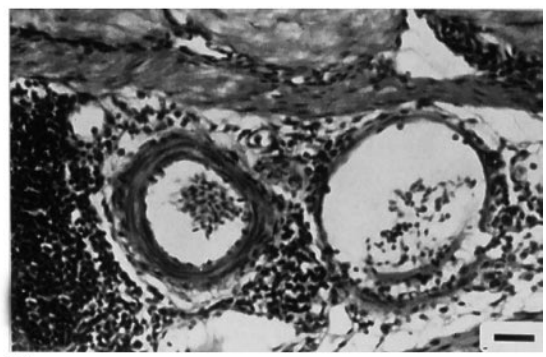
(e)



(f)



(g)



(h)

extracts of the worms and with controls. This suggests that the worms may have immunosuppressive effects in their avian hosts.

PUBLIC HEALTH CONCERNS

Avian ascaridoids are generally not thought to be a significant risk to public health. However, ascaridoids from humans are often not identified or are degenerated and cannot be identified. It has been suggested that juvenile worms may be able to acclimate to high temperature or develop to fourth-stage juveniles under some conditions and possibly infect humans. This has been reported for *C. multipapillatum* in Mayan cichlid fish from Mexico and these worms infected the intestine of an experimental kitten, developed into adults, and produced hemorrhaging ulcers (Vidal-Martinez et al. 1994; R. M. Overstreet, unpublished observations). An accidental infection by a nonfertilized species of *Contracaecum* was found embedded in the damaged brain of a striped dolphin (Martin et al. 1970). In contrast, specimens reported as *Contracaecum* sp. in a granulomatous lesion of a dog in Japan (Kitayama et al. 1967) appear to represent a species of *Hysterothylacium*, which matures in fish (Vidal-Martinez et al. 1994).

Other than attention-seeking children, most humans do not commonly eat earthworms, but infections with *Porrocaecum* spp. from earthworms, other unknown intermediate hosts, or paratenic hosts are possible. Because of the difficulty in identifying species of *Porrocaecum* in human tissue sections, similar appearing species have been misidentified as *Porrocaecum*. Examples include parasites from the genus *Terranova* Leiper and Atkinson, 1914 that mature in elasmobranchs and species of *Pseudoterranova* that mature in marine pinnipeds (Deardorff et al. 1983). Species of *Pseudoterranova* commonly infect humans, and juveniles of a *Terranova* sp. were shown to penetrate into the stomach of a rat, ultimately forming a granuloma (Deardorff et al. 1983).

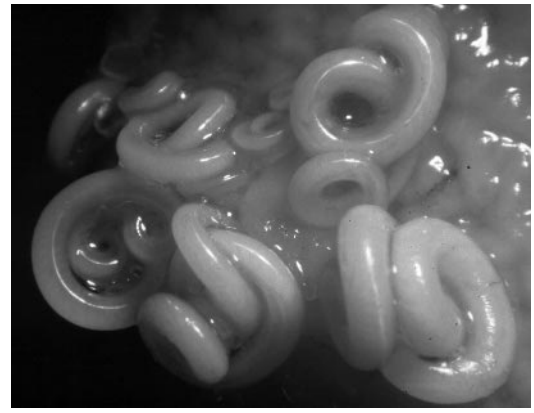


Figure 24.7. Adult *Contracaecum multipapillatum sensu lato* from a pelican. Close-up image of worms embedded in the proventricular mucosa (compare with Figure 24.6a).

DOMESTIC ANIMAL HEALTH CONCERNS

Porrocaecum spp. are a potential risk for domestic birds (Hair and Forrester 1970; Coles 1985), especially when earthworms are present or paratenic hosts have access to infected hosts. However, the apparent host specificity of adult worms may reduce the relative risk of infection to domestic hosts. Both juveniles and adults can harm the host when present in high enough numbers, and these in turn can ultimately contaminate the substratum with eggs that are capable of continuing high rates of infection. Relatively little, however, is known about the risk that wild birds pose to domesticated hosts and appropriate experimental studies are needed to help determine this.

Species of *Contracaecum* can have a major influence on aquatic birds being reared commercially, on display in zoos and other parks, or used in research or recovery. Infections can rapidly accumulate in birds that are confined in small amounts of water with fish that will serve as intermediate hosts.

←

Figure 24.6. Proventriculus of a Black Noddy (*Anous minutus*) infected with *Contracaecum magnipapillatum* (compare with Figure 24.1). (a) Numerous specimens attached to the proventricular mucosa (sp = spleen) (scale bar = 3.3 mm). (b) Normal histological appearance of proventriculus. (c) Invasion by parasite (right) with an associated inflammatory reaction (arrows indicate border of muscularis). Scale bar = 100 μm. (d) Invasion by parasite (right) with less involvement of the muscularis (cf. previous image), however, with muscularis interna obliterated (arrows). Scale bar = 100 μm. (e) Worm with hyaline cap with inflammatory exudate. Scale bar = 40 μm. (f) Hyaline cap (see star), enlarged (cf. previous image). (g) Partly resorbed hyaline cap. Scale bar = 25 μm. (h) Concentration of inflammatory cells in affected regions. Scale bar = 25 μm. Modified from Fagerholm et al. (1996) with permission of *Helminthologia*.

WILDLIFE POPULATION IMPACTS

One should be aware that most infections of ascaridoids of average intensity are not harmful and possibly even beneficial. Because adult ascaridoids often entwine among dietary items in the alimentary tracts of birds as well as of other animals, they may well aid in the digestive process and be advantageous, at least when food is abundant (e.g., Owre 1962; Deardorff and Overstreet 1980a, b; Fagerholm et al. 1996).

Even though individual birds can be heavily infected with ascaridoids, these infections probably have little influence on wildlife populations unless they are compromised by environmental conditions or stress. Intense alimentary tract ascaridoid infections can weaken avian hosts and make them more susceptible to predation, trauma from automobiles, or cause abnormal behavior. Large numbers of *Contracaecum* spp. in pelicans and other waterbirds have been reported. For example, McOrist (1989) reported peritonitis associated with *C. spiculigerum* (=*Contracaecum rudolphii sensu lato*) in 2 Rufous Night-Herons (*Nycticorax caledonicus*) that may have contributed to their starvation and death. Lesions in the birds included fibrinous hemorrhagic exudates over the intestinal serosa. There are reports of mortality in American White Pelicans (*Pelecanus erythrorhynchos*) and Brown Pelicans (*Pelecanus occidentalis*) and Double-crested Cormorants from the southeastern US that are associated with intense infections of hundreds of individuals of *C. multipapillatum* and other ascaridoids (*Contracaecum microcephalum* and *C. rudolphii sensu lato*) (Deardorff and Overstreet 1980b). Oglesby (1960) reported more than 1,100 specimens in a mixed infection from an American White Pelican, and Courtney and Forrester (1974) counted 1,192 in a Brown Pelican. Intensity of infections usually number less than 100 worms, but even birds with intense infections may appear healthy.

Fatal ascaridoid infections have also been reported from other avian hosts. Two of 92 Eurasian Kestrels (*Falco tinnunculus*) that died in the British Isles of apparent starvation had 50–70 large specimens of *P. depressum* that impacted the duodenum and upper small intestine (Keymer et al. 1981). The birds also had a concurrent infection with the coccidian *Isospora buteonis*, which may have contributed to the deaths.

TREATMENT AND CONTROL

Unmanageable infections in wild birds result from biological and environmental conditions that concentrate birds and intermediate hosts together. Little can be done about the infections, but measures can be taken to reduce compounding stress from lead shot, pesticides, and other contaminants. In domestic situations, intermediate and paratenic hosts can be eliminated or reduced. Birds can be medicated, if necessary, for treatment of breeding stock or valuable individuals. Greve et al. (1986) and Grimes et al. (1989) determined that albendazole and fenbendazole were effective against *C. multipapillatum* in the Brown Pelican and piperazine dihydrochloride, clorsulon, and Curatrem were not. Good results have been reported with a single dose of 1-tetramisole but not with arecoline hydrobromide, thiabendazole, or niclosamide (Courtney et al. 1977).

Cooking-infected products destined for dispersal or consumption by humans or other animals will kill all stages of *Contracaecum* and *Porrocaecum*, even though only the juveniles in the intermediate or paratenic hosts would be infective to humans. The US Department of Agriculture recommends freezing and storing at −20°C or below for 7 days or freezing at −35°C or below until solid and then storing at −35°C or below for 15 h or at −20°C or below for 24 h. Heating at 63°C for 17 min will also kill all nematodes as well as bacteria.

Control practices for some anisakid infections under normal conditions in the wild can be cost-prohibitive, not practical, or totally unnecessary. Mortality of nestling or young birds may occur in aquatic areas resulting from infections with *Contracaecum* spp. and the mortalities should be investigated for complicating factors. *Porrocaecum* spp. can be serious for both wild and domestic birds that have access to earthworms or other intermediate and paratenic hosts. Both juveniles and adults can harm the host when present in high enough numbers, and these in turn ultimately contaminate the substratum with eggs capable of causing infections. For *Porrocaecum* or *Contracaecum*, control strategies should first consider eliminating or reducing access to the intermediate hosts. For *Contracaecum*, hosts reared in an aquaculture setting must be prevented from feeding on infected fish and from defecating in the culture system.

BAYLISACARIS

SYNONYMS

Depending on their location in the host, infections with the juvenile L3 stage of *Baylisascaris* are referred to as baylisascariasis, larva migrans, visceral larva migrans (VLM), verminous encephalitis, cerebrospinal (or cerebellar or cerebral) nematodiasis, and nonsuppurative meningoencephalitis. Human infections are usually referred to as eosinophilic meningoencephalitis, VLM, baylisascariasis, or raccoon roundworm encephalitis.

ETIOLOGY

The genus *Baylisascaris* contains as many as nine or more species (Sprent 1968), including *Baylisascaris*

procyonis (Stefanski and Zarnowski, 1951) from the raccoon (*Procyon lotor*) and kinkajou (*Potos flavus*) in North and South America (Overstreet 1970), *Baylisascaris columnaris* (Leidy, 1856) from skunks (Mephitidae) in North America (Overstreet 1970), *Baylisascaris melis* (Gedoelst, 1920) from badgers (Mustelidae), *Baylisascaris transfuga* (Rudolphi, 1819) from bears and pandas (Ursidae and Procyonidae) throughout the world, and *Baylisascaris laevis* (Leydy, 1856) from large rodents (Rodentia). The larval stages of virtually all these species pose some risk to birds if eggs or larvae are ingested.

HOST RANGE AND DISTRIBUTION

The larval stages of species of *Baylisascaris* have been reported from a wide variety of avian hosts from North America (Figure 24.8). The absence of reports from other parts of the world likely reflects inadequate sampling; however, the infective parasites are primarily North American (Overstreet 1970). Larval stages of *Baylisascaris* are more commonly reported from ground-feeding species such as doves (*Zeinaida* spp.), where exposure to eggs is more likely.

EPIZOOTIOLOGY

Adult species of *Baylisascaris* occur in the intestines of a specific mammalian definitive host and eggs are passed in the feces. Developed eggs of *B. procyonis* and *B. laevis* may infect the final, or definitive host directly. In this case, the L3 (often reported as L2) larvae hatch in the intestine and "migrate to the intestinal mucosa" (Kazacos 2001) where they eventually develop into adults. Another route is taken when developed eggs or hatched larvae are ingested by paratenic hosts, primarily small rodents and birds. In these animals, L3 larvae hatch from the eggs and migrate via the circulatory system to the liver and the lungs and then reach different organs via the heart. The severity of the host tissue reactions depends specifically on the species of paratenic host, the site in the host, and the host immune status. Depending on the location in the body and where they cause adverse signs, larvae cause a disease referred to as visceral larva migrans (VLM), ocular larva migrans (OLM), and neural larva migrans (NLM) (Kazacos 1986; Gavin et al. 2005). When an infected paratenic host is consumed by a mammalian definitive host, L3 larvae develop directly into L4 and later into adult worms in the intestine. The life cycle of different species of *Baylisascaris* should be studied in more detail and compared with other parasites in the same subfamily, for example, *Ascaris* spp., to establish similarities in the development of the larvae and migration routes in the final host (Murrell et al. 1997; Geenen et al. 1999; Fagerholm et al. 2000).

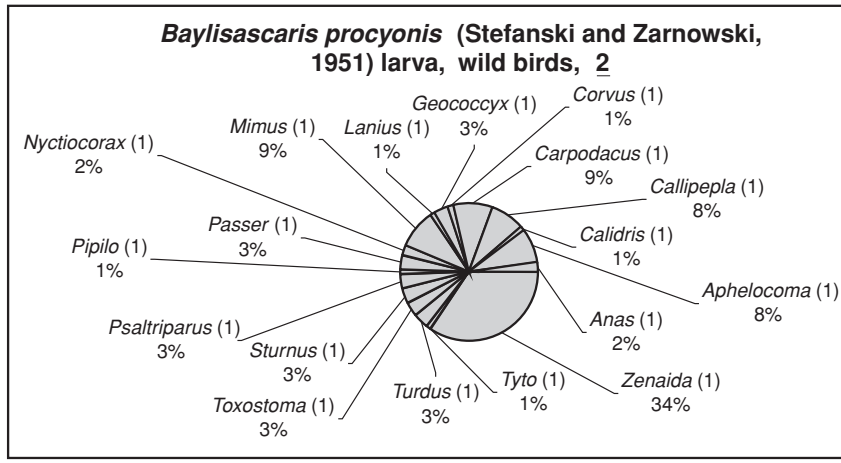
Birds become involved in the life cycle of *Baylisascaris* as paratenic hosts when they ingest eggs. Reports of infections with *Baylisascaris* in wild birds are not abundant. However, infections in birds may be more common than generally recognized, given how long infective eggs can persist in the environment. Some studies have shown that each stool from an average infected raccoon contains from 2 to 10 million eggs, that worms can produce eggs for long periods, and that the eggs of *B. procyonis* can remain infective under refrigeration for at least 12 years and probably 3–4 years in the wild where they are exposed to summer heat and winter freezing (Kazacos et al. 1982). Large outbreaks of VLM have been reported in domestic birds such as chickens, illustrating the potential of the eggs to infect birds (Richardson et al. 1980).

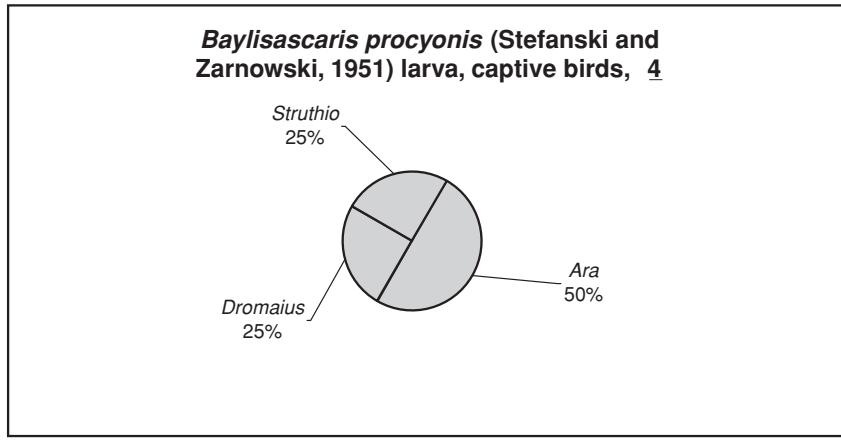
DIAGNOSIS

When found in histological sections of avian host tissues, larvae of *Baylisascaris* may be confused with L3 larvae of several other genera of nematodes that cause larva migrans. They are often not identified to species because they have few differentiating features. Diagnostic features are found on both posterior and anterior ends of larvae and in details of somatic structures that are evident in transverse sections. L3 larvae of *B. procyonis* are longer (1.27–1.56 mm) (Figure 24.9) than larvae of *Toxascaris leonina* (Linstov, 1902) Leiper, 1907 (0.65–0.75 mm). Larvae of two other genera that may be confused with *Baylisascaris*—*Lagochilascaris* sp. and *Hexametra* sp.—are longer than larvae of *Baylisascaris* and measure up to 5 mm in length for *Lagochilascaris* and up to 10 mm in length for *Hexametra* sp. (Bowman 1987). Additional distinguishing morphological features have been discussed by Bowman (1987) for *Toxocara* spp., *Porrocaecum*, *Ophidascaris*, *Travassosascaris*, and *Polydelphis*.

A transverse section of the anterior region of an L3 larva of *B. columnaris* is illustrated in Figure 24.10. High-quality, well-fixed material and considerable experience are needed to make identifications. Circumstantial evidence including geographic data and host history may also aid in the diagnosis. While it is also possible to analyze the ontogeny of the cuticula by SEM to differentiate taxonomic groups (Fagerholm et al. 2000), this is not routinely done.

With recent advances in DNA-based technologies (Gasser 2006; Nadler et al. 2007), identifications based on polymerase chain reaction amplification of ribosomal DNA sequences and internal spacers, ITS-1 and ITS-2, can be done with primers known to function in the Ascaridoidea (Nadler et al. 2000). Mitochondrial genes (mtDNA-cox2) have also been useful (Mattiucci et al. 2008) as well as the use of allozymes (Nascetti et al. 1986).





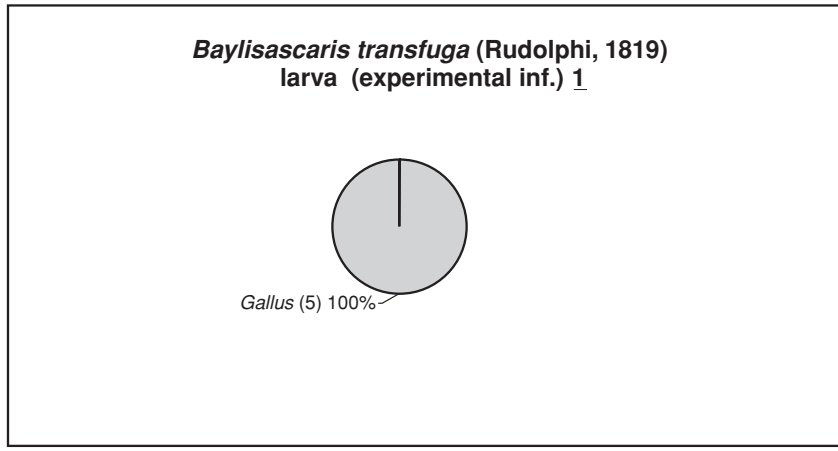


Figure 24.8. Host range of larvae (larva migrans) of species of *Baylisascaris*. Diagrams are based on published data on percentage of recorded cases of *Baylisascaris* in different genera of avian hosts. Information was retrieved from Gibson et al. (2005). The number of records is underlined in heading. The origin (geographical area) of records is shown in parenthesis after the genus name: 1, North America; 2, South America; 3, Australia and New Zealand; 4, Asia (including India and Japan); 5, Europe; 6, Africa.

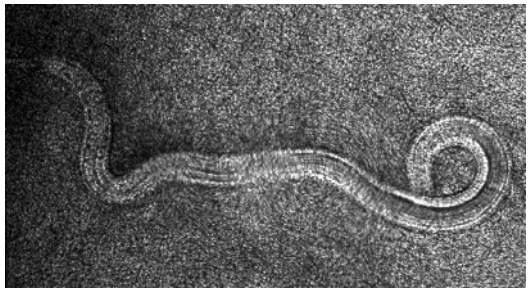


Figure 24.9. Live specimen (L3 stage) of *Baylisacaris procyonis* from the brain of paratenic mammalian vertebrate host.

CLINICAL SIGNS

Birds with central nervous system (CNS) infections of larval *B. procyonis*, *B. columnaris*, *B. melis*, and perhaps other species may first exhibit head-tilt or slight stumbling. As the infection progresses, the bird may circle, roll, or fall and eventually become recumbent and comatose before dying (Kazacos 1983). In addition to loss of coordination, torticollis, and paralysis of wings, the feathers are ruffled (Reed et al. 1981).

PATHOLOGY

There are few studies investigating tissue reactions to NLM in birds. Early work by Tiner (1953a, b) demonstrated that pathogenesis of *Baylisascaris* in the CNS depends on host species, host size, and migratory behavior of the larval worms. Experimental work with chickens has been done with *B. transfuga* (Papini et al. 1993) and *B. procyonis* (Kazacos and Wirtz 1983), but we still know few details about migratory behavior and pathogenesis of infections in avian hosts.

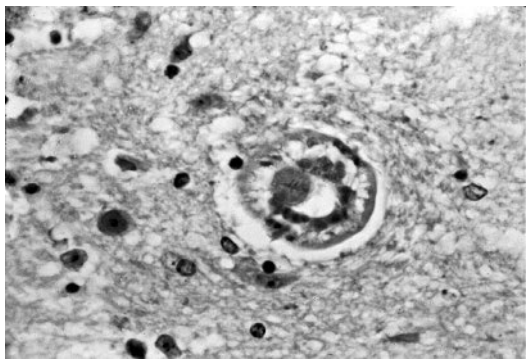


Figure 24.10. Cross section of *Baylisacaris columnaris* larva (L3) in brain of a paratenic mammalian host. Lateral alae (with expansion of hypodermis) discernable.

In mammals, host species has an effect on where juvenile L3 migrate. For example, most juvenile *B. procyonis* and *B. columnaris* are restricted to encapsulations in the thoracic musculature in rodents, but a few migrate to the brain. In mice, about 5–9% of juvenile *B. procyonis* enter the brain (R. M. Overstreet, unpublished data). Mice fed 25 eggs begin to have problems with orientation about day 14 postinfection (PI), proceed to drag their rear legs or run continuously in circles, and ultimately die at about day 21–23 PI. A single juvenile in the medulla can kill a mouse. In mice given 175 eggs, loss of coordination begins at 6–11 days PI and death occurs by 8–14 days PI. Mice infected with many more eggs show signs at day 6 PI and die at day 8–9 PI. No mouse recovered after it lost coordination.

Juvenile *Baylisascaris* appear to initially migrate within the cerebral hemispheres for a few days and then migrate to the cerebellum, medulla, and upper spinal cord, at which time disorientation becomes evident. From day 8 to day 10 PI, average length of individual worms increases from about 0.30 to 1.00 mm, causing considerable damage to the brain. The same development presumably takes place in the avian brain. Kazacos (1983) investigated infections in a variety of paratenic hosts and found that 5–15% of the juvenile worms reach the brain of most hosts. A single juvenile worm can be fatal in some avian hosts, including Emu (*Dromaius novaehollandiae*) (Kazacos et al. 1982) and macaws (*Ara arauna*, *Ara macao*, and a hybrid cross) (Armstrong et al. 1989).

Gross lesions are not evident in Northern Bobwhite (*Colinus virginianus*) and other birds killed by *B. procyonis* (Reed et al. 1981; Kazacos and Wirtz 1983), but microscopic lesions are associated with the migratory tract of the parasites and may occur throughout most areas of the brain. Microscopic lesions include sections through juvenile worms and widely disseminated nonsupportive meningoencephalitis with multifocal areas of malacia, necrosis, and intense inflammatory reaction. Longer-surviving birds exhibit focal granulomas. No alteration was observed in sites other than the brain. Chickens infected experimentally with only 200 eggs exhibited few lesions in the brain (Kazacos and Wirtz 1983).

IMMUNITY

Little is known about immunity to larval stages of *Baylisascaris* in avian hosts. On the basis of studies of larval anisakid infections in mammals, hosts are likely to have a strong antibody response, strong blood and tissue eosinophil levels, and a strong T-helper type 2 cell response. The intensity of the response is

species specific, and those in which the juveniles migrate within the brain seem to have a stronger immune response (Sheppard and Kazacos 1997).

PUBLIC HEALTH CONCERNS

The primary public health risk from *B. procyonis* is associated with contact with raccoon latrines and pica/geophagia and not interaction with infected birds or other nondefinitive hosts.

DOMESTIC ANIMAL HEALTH CONCERNS

Baylisascaris can cause fatal or severe CNS disease in pheasants, chickens, domestic quail, partridges, pigeons, exotic turkeys, Emus, and a wide variety of other species (Kazacos 2001). As is the case with human infections, these risks are associated with contact with raccoon latrines and pica/geophagia and not interaction with infected wild birds or other nondefinitive hosts (Kazacos 1983).

WILDLIFE POPULATION IMPACTS

Most infections of *Baylisascaris* in wild birds probably consist of no more than a few juveniles and do not develop into disease. However, juvenile *Baylisascaris* have been documented in the brains of numerous species of passerines as well as in a few shorebirds, quail, and ducks in studies conducted in California, USA (Evans and Tangredi 1985; Evans 2002). A Barn Owl (*Tyto alba*) was suspected to have become infected by feeding on an infected California ground squirrel (*Spermophilus beecheyi*). Abundance of passeriform birds was coincidentally low in the study during a period of time when raccoons were fed and heavily protected, presumably from NLM. Evans (2002) studied 18 species of birds and found a total of 87 birds with *B. procyonis*-associated NLM or/and VLM. In this study, four areas close to raccoon latrines were inspected for birds with abnormal behavior.

Few cases are known where wild birds have been observed with signs of larval migrans, but the actual number of clinical cases in wild birds may be underestimated because sick or moribund birds are more susceptible to predation. In east-central Kansas, a decline in Northern Bobwhite populations occurred during a period with increased raccoon and skunk populations. It was suggested that many birds had died from baylisascariasis, although this conclusion was based on only a single case (Williams et al. 1997). Page et al. (1999) found that 15 species of birds regularly visited sites of raccoon latrines in Indiana, USA, providing further evidence that birds may be exposed to infective larvae of *B. procyonis* in areas populated by raccoons.

TREATMENT AND CONTROL

In the case of *Baylisascaris* spp., especially *B. procyonis*, ingestion of only a few eggs can result in death. Management strategies for these agents primarily involve keeping raccoons and other hosts (e.g., mustelids, badgers, and ursids) from contaminating the environment with feces. Numerous compounds have been tested for treatment of adult *Baylisascaris* in the raccoon, and such treatment can help manage infections in domestic birds (e.g., Bauer and Gey 1995).

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25
Diplotriaena, Serratospiculum, and Serratospiculoides

Mauritz C. Sterner, III and Rebecca A. Cole

INTRODUCTION

Diplotriaena and *Serratospiculum* are the two most common genera of air sac parasites of birds. Nematodes that belong to the genus *Serratospiculoides* are less frequently encountered but are closely related. All three genera belong to the order Spirurida, suborder Spirurina, and like all members of this suborder, use arthropods as intermediate hosts. These three genera have historically been confused with filariid nematodes because of their long filiform bodies, sexually dimorphic features, and presence in air sacs. Males are much smaller than females and usually have a spiral tail.

Adult worms within these three genera can cause degenerative changes within the collagen and muscle layers between the epithelial and mesothelial components of the air sacs, resulting in the thickening of the air sac tissue. These changes in the lung may predispose birds to pneumonia in areas where eggs and bloody edema have blocked secondary bronchi and associated parabronchi, and lead to secondary infections of *Pseudomonas* spp., *Klebsiella* spp., and *Aspergillus* spp. These secondary bacterial infections, in turn, may result in pneumonia, air sacculitis, aspergillosis, and death of the host. Adult worms may also puncture the air sacs and gain access to the body cavity, depositing eggs within the liver and various other organs. Migration of larvae through organs and the presence of embryonated eggs can cause physical damage to tissues as well as occlusions and congestion of various structures such as bile ducts and hepatic veins.

HISTORY

The first species of *Diplotriaena* was described in Europe and later in India, Russia, Burma, Australia, China, and Africa. The first report of this genus in North America was by Walton (1927) from the Florida Scrub-Jay (*Aphelocoma coerulescens*).

Serratospiculum spp. and *Serratospiculoides* spp. are so similar that the differences can only be appreciated by microscopy. *Serratospiculum* spp. were first identified by Skrijabin (1916) from the Eurasian Kestrel (*Falco tinnunculus)* in Russia. The first species reported in the US was *Serratospiculum amaculata* (later to be renamed *Serratospiculoides amaculata*) by Wehr (1935) in a Prairie Falcon (*Falco mexicanus*) from Montana.

All three genera belong to the superfamily Filarioidea. Unlike others in Filariidae, where microfilaria are found in the blood and transmitted by biting insects (see Chapter 26), these parasites lay eggs which pass through the respiratory system and leave the body of the host via fecal material (Chabaud 1955). The eggs are subsequently eaten by foraging insects and larval development of the worm takes place within the hemocoel (Anderson 1957). Infected insects are eaten by birds to complete the life cycle (Anderson 1957, 1962, 1992; Sonin 1963).

Studies conducted by Anderson (1962) confirmed the life cycle and led to reclassification of *Diplotriaena*, *Serratospiculum*, and *Serratospiculoides* in the order Spirurida. The superfamily Filarioidea was divided into three superfamilies. Two new superfamilies were added, the Diplotriaenoidea and Aproctoidea, representing nematodes with oviparous development (Sonin 1963). The new classification placed the genus *Diplotriaena* in the family Diplotriaenidae, subfamily Diplotriaeninae and placed the two genera *Serratospiculum* and *Serratospiculoides* in the subfamily Dicheilonematinae (Anderson and Bain 1976; Anderson 1992).

HOST RANGE AND DISTRIBUTION

The distribution of species of *Diplotriaena*, *Serratospiculum*, and *Serratospiculoides* is cosmopolitan. Species of *Diplotriaena* parasitize the air sacs and

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subcutaneous tissue of birds belonging to the orders Anseriformes, Apodiformes Galliformes, Charadriiformes, Columbiformes, Piciformes, and Passeriformes. Families include Columbidae, Apodidae, Picidae, Furnariidae, Dendrocolaptidae, Tyrannidae, Vireonidae, Corvidae, Hirundinidae, Sittidae, Sylviidae, Muscicapidae, Turdidae, Mimidae, Sturnidae, Bombycillidae, Ptilogonatidae, Parulidae, Thraupidae, and Icteridae.

S*erratospiculum* spp. have been reported from North and South America, Australia, Europe, the UK, Asia, Africa, and the Middle East (Bain and Mawson 1981; Quentin et al. 1983; Gomez et al. 1993; Lierz and Remple 1997; Samour and Naldo 2001; Lloyd 2003). Species of *Serratospiculum* and *Serratospiculoides* infect the air sacs of carnivorous birds, primarily of the order Falconiformes, but have also been recorded from accipiters in North America (Sterner and Espinosa 1988; Taft et al. 1993).

ETIOLOGY

All three genera have typical characteristics of filariid nematodes in that they are long filamentous worms found usually in the subcutaneous tissues but also in the heart, lungs, air sacs, and various other organs of the host. Males are much smaller than females and posses a hooked tail (Olsen 1962). With these similarities they were originally placed in the group classified as filarids. However, further study indicated that they produced thick-shelled eggs that developed in insect hosts rather than microfilariae, which led to a reclassification of these three genera to the order Spiurida.

The type species of the genus *Diplotriaena*, *Diplotriaena ozouzi*, was described by Railliet and Henry in 1909 in Europe from a reptile, *Fuadias madagascariensis*. The number of species within the genus has been debated for some time. Skrjabin (1949) listed 65 species in this genus. Yamaguti (1961) listed a total of 76 species. Reclassification of this genus by Anderson and Bain (1976) listed over 47 species as being valid. The most recent consensus is that the genus *Diplotriaena* has 27 valid species that occur throughout the world (Anderson 1959; Levine 1980).

Skrijabin (1916) described a new genus *Serratospiculum*. Because morphological characteristics were similar to filarids, this genus was placed in the superfamily Filarioidea, family Filariidae, subfamily Setarinae (York and Maplestone 1926; Olsen 1962). Later, studies by Chabaud (1964), Anderson (1992), and Anderson and Bain (1976) showed that the species within the genus *Serratospiculum* did not develop microfilariae. Instead, eggs were eaten by insects and partial development occurred within the insect host. This life cycle resembled those of spirurid nematodes and led to the reclassification of the genus *Serratospiculum* into the order Spirurida, superfamily Diplotriaenoidea, family Diplotriaenidae, subfamily Dicheilonematinae.

The number of species within the genera *Serratospiculum* and *Serratospiculoides* has remained constant over the last several years. On the basis of length of the spicules, nine species of *Serratospiculum* are recognized from avian hosts in the order Falconiformes (Samour and Naldo 2001).

The type species for the genus *Serratospiculoides* is *Serratospiculoides alii. Serratospiculoides alii* was first described in India by Rasheed in 1960, but identified as *Hamatospiculum alii*. Chabaud et al. (1964) renamed the specimen *Serratospiculum ali*, based on differences in spicule morphology from other specimens of *Hamatospiculum* and their similarity to spicules from *Serratospiculum amaculatum*. Sonin (1968) examined these two species and determined that the morphology of spicules of *S. alii* and *S. amaculatum* were the same and intermediate in form between species of *Hamatospiculum* and *Serratospiculum*. These differences in spicule characteristics led to reclassification of this species as the new genus *Serratospiculoides* (Sonin 1968; Samour and Naldo 2001). There are only two species in this genus, *S. alii* and *S. amaculatum.*

EPIZOOTIOLOGY

The life cycles of species of *Diplotriaena*, *Serratospiculum*, and *Serratospiculoides* are indirect and use an intermediate host. Adults of the species in all three genera are found in the abdominal and thoracic air sacs of the host. The final or definitive hosts of species of *Diplotriaena* include a variety of passerine species and their intermediate hosts are grasshoppers (order Orthoptera).

Thick-shelled eggs containing fully developed first-stage (L1) larvae are passed out in the feces of the infected bird. The eggs are eaten by the nymphal stage or young adults of coprophagic (dung eating) grasshoppers (Cawthorn and Anderson 1980a; Ansari 1982). Within the grasshopper, L1 larvae are released and actively burrow out of the gut and into the hemocoel. The larvae lodge in fat bodies throughout the hemocoel and begin to develop (Olsen 1962). Within 4 days the worms have molted to become second-stage (L2) larvae. Within 8 days after invading the fat bodies, they molt again to become third-stage (L3) larvae. The L3 larvae are infective to the definitive host and remain dormant within a clear thin encapsulating membrane until the grasshopper is eaten by a suitable avian host. Encapsulated larvae can survive as long as their intermediate hosts, with most transmission during the spring and summer months (Anderson 1956, 1992; Cawthorn and Anderson 1980a; Ansari 1982).

After infected grasshoppers are eaten by a suitable avian host, larvae are digested out of the capsules and released into the intestine. The L3 larvae penetrate the intestinal lining and enter the hepatic portal system, where they develop into fourth-stage (L4) larvae, or subadults in approximately 20 days (Cawthorn and Anderson 1980a). They migrate as subadults from the hepatic portal system to the lungs via the right side of the heart and the pulmonary arteries. The subadults subsequently break out of the arterial system and into the lungs where they move to the air sac and undergo a final molt to become adult worms, approximately 20 days after entering the lungs. Adults can remain active and alive within the definitive host for several years (Cawthorn and Anderson 1980b).

Female worms mature in 4 months and begin laying eggs within the air sacs that move to the lungs through natural movement of air and mucus. Once in the lungs, the eggs are coughed up, swallowed, and pass out in the feces, completing the life cycle (Anderson 1956).

The life cycles of species of *Serratospiculum* and *Serratospiculoides* are similar. Eggs are eaten by a variety of coprophagic beetles and hatch in the gut of the intermediate hosts. The L1 larvae move to adipose tissue of the host, where they become encapsulated in a thin and transparent capsule. The L1 larvae develop and mature to the infective L3 stage within these capsules. Samour and Naldo (2001) determined that the L3 remain in the capsule until the beetle is eaten by the definitive host. After ingestion, the L3 are digested out of the capsule and penetrate the wall of the proventriculus or ventriculus. There is some histopathological evidence that the L3 go directly to the air sacs and not through the portal system (Gomez et al. 1993). Once in the air sacs, the L3 larvae undergo two molts and become immature adults (Samour and Naldo 2001). Mature adults mate and females produce a large number of thin-shelled, embryonated eggs. The eggs are coughed up into the mouth, swallowed, and passed out in the feces.

CLINICAL SIGNS

Birds infected with air sac parasites display chronic illness. Signs include lethargy ("poor doers"), labored breathing, below average body weight or size, and poor plumage or unthriftiness. Infected birds may be less likely to breed. Thin- or thick-shelled eggs can sometimes be seen by microscopy in fecal samples.

PATHOLOGY

Species of *Diplotriaena* affect many organs both as a result of the presence of the adult worms and the secondary tissue damage arising from larval migration and the presence of eggs. The lungs, liver, air sacs, and vascular system are frequently affected. Pathological changes associated with subadult, adult worms, and eggs include inflammatory lesions in the liver and lungs, bronchitis caused by lymphoid hyperplasia, edema in airways, fibroplasias within the air sacs and hepatic parenchyma, adhesions between the liver and air sacs, hemorrhage in the liver, periarteritis, endothelial swelling and vacuolation, congestion in the lungs, thrombi present in arteries, pulmonary inflammation, granulomatous pneumonia in the lungs, and mucoid hyperplasia (Cawthorn et al. 1980).

As they penetrate the intestine and enter the hepatic portal system, larvae cause petechial hemorrhages along the intestine and the liver. Traumatic liver lesions can result from the encapsulation of dead nematodes and eggs by giant cell granulomas, leading to chronic liver inflammation, including lymphoid hyperplasia (Ansari 1985; Young et al. 1998).

During migration to the lungs via arteries, the subadults cause swelling of the endothelial cells and invasion of the tunica media by macrophages, resulting in periarteritis. Once larvae reach the lungs, they cause edema, fibroplasia, hemorrhaging within the secondary bronchi, lymphoid hyperplasia, and thrombosis. Large giant cell granulomas can be found close to the secondary bronchi. As the number of larvae increase within the lungs, tissue swelling and presence of the worms can cause blockage and congestion of the air passages. Once the subadults reach the air sacs, an acute inflammatory reaction occurs around the parasites, resulting in fibrotic thickening of the air sac as well as adhesions among air sacs, the body cavity, and the internal organs (Cawthorn et al. 1980).

The majority of the pathological changes associated with species of *Serratospiculum* and *Serratospiculoides* occur in association with the adult worms in the air sacs. Air sac membranes become thickened, compromising air exchange (Kocan and Gordon 1976). Congestion, focal hemorrhages, focal necrosis, and moderate infiltration of macrophages occur within the lungs of infected birds. Serratospiculiasis is characterized by necrosis of the crop and esophagus, edema in the media of the arterioles and bronchial passages, congested hepatic veins, squamous metaplasia, hyperplasia of the mesothelium, heterophile infiltration, focal hemorrhages in the lungs, lesions within the lungs and spinal cord, air sacculitis, and pneumonia (Kocan and Gordon 1976). Other lesions include hepatitis, chronic cholangitis, pericholangitis, pericarditis, atelectasis of the air sacs, and degeneration of the collagen-muscle layer of the air sacs. Damage caused to the air sacs by species of *Serratospiculum* predispose the bird to secondary infections with species of *Aspergillus*, *Pseudomonas*, and *Klebsiella* (Samour and Naldo 2001).

DIAGNOSIS

The presence of embryonated eggs within the feces as a diagnostic tool is unreliable because other parasites besides air sac worms lay embryonated eggs. Both the location of the adult worms interwoven within the abdominal air sacs and morphological features characteristic of these genera are the only reliable means for diagnosing an infection.

PUBLIC AND DOMESTIC ANIMAL HEALTH CONCERNS

There is no evidence that these nematodes infect humans. While there have been no major die-offs or reports concerning deaths associated with this parasite in domestic fowl, free-ranging poultry, such as chickens and turkeys, can become infected by feeding on grasshoppers, although reports are rare.

WILDLIFE POPULATION IMPACTS

Although deaths of individuals have been attributed to infection with species of these genera (Bigland et al. 1964; Ward and Fairchild 1972; Sterner and Espinosa 1988; Ackerman 1992; Young et al. 1998; Hawkins et al. 2001), no widespread mortality events have been reported.

TREATMENT AND CONTROL

Since the only reliable means for determining whether these parasites are present is to physically find and identify the worms, treatment specifically for air sac nematodes is usually not undertaken. However, infections with species of *Serratospiculum* or *Serratospiculoides* have been treated with mebendazole, Panacur, and fenbendazole with some degree of success in raptors used for falconry. In the Middle East, Samour and Naldo (2001) reported that these parasites are routinely removed endoscopically 3–5 days after treatment with ivermectin. The surgery is followed with a second dose of ivermectin 1–2 weeks later (Lierz 2001; Lloyd 2003). Control of these parasites in wildlife populations is generally not undertaken.

DISCLAIMER

Any use of trade, product, or firm names in this publication is for descriptive purposes only and does not imply endorsement by the U.S. government.

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26
Filarioid Nematodes

Cheryl M. Bartlett

INTRODUCTION

Filarioids are highly specialized nematode parasites of the tissues and tissue spaces of birds, mammals, amphibians, and reptiles. About 160 species are known from birds. Infections are probably much more common than this number and that the literature suggest, primarily because adult worms can be notoriously difficult to find and thus are often not sought or are overlooked at necropsy. Also, with notable exceptions, avian filarioids are not pathogenic, and even among those that are, few draw attention by provoking clinical signs. In addition, adult worms of some species are short lived and disappear after microfilariae (MF), the specialized, first-stage larvae produced by adult female worms, are released. This ephemerality of adults (an unusual life history trait) is balanced by longevity of MF.

The life cycles of all filarioids involve a hematophagous invertebrate intermediate host (vector) that ingests MF. After release by the adult female, MF generally enter the bloodstream and become available to vectors. Blood-borne MF are relatively easy to detect; indeed, much of our awareness of avian filarioids as common parasites in birds is based on surveys of avian hosts for hematozoa by examination of stained blood smears. However, surveys of birds for helminths do not always include hematozoa. Most have never included microscopic examination of skin, yet skin rather than blood is occupied by MF of some avian filarioids and such MF are probably more widespread than currently realized. Finally, individual birds can harbor more than one filarioid species (same genus or different genera), and this possibility is not always taken into account at necropsy.

This chapter provides the first published synopsis of the taxonomy, biology, and pathogenesis of the filarioid parasites of the world's birds (see www.integrativescience.ca for exhaustive references). It also emphasizes information that can facilitate finding adult filarioids in birds at necropsy since species identifications are based on adults, and modern studies of wildlife parasites and diseases require accuracy with respect to species and pathogen identifications.

HISTORY

Modern classification (Anderson et al. 1974) places filarioids in the superfamily Filarioidea, order Spirurida, suborder Spirurina. Some avian nematodes are still initially identified incorrectly at the level of superfamily, reflecting the overly quick (and outdated) tendency to assign the labels of filarid, filariid, filarioid, or filarial worm to any nematode found outside the lumen of the gastrointestinal tract. However, taxonomic clarity was achieved with the CIH Key No. 1 (Chabaud, in Anderson et al. 1974) to nematode superfamilies in conjunction with the CIH Key No. 3 (Anderson and Bain 1976) to genera in superfamilies Diplotriaenoidea, Aproctoidea, and Filarioidea. The important taxonomic understanding is that nematodes producing microfilariae (family Onchocercidae in superfamily Filarioidea) or those that seem to be closely related (family Filariidae in superfamily Filarioidea) are now classified separately from nematodes inhabiting the air sacs of their hosts (superfamilies Diplotriaenoidea and Aproctoidea) (Chapter 25). Diplotriaenoids (which parasitize both birds and reptiles) produce thick-shelled eggs with fully differentiated first-stage larvae that pass to the outside via the respiratory system and gut of the host. They are believed to be a homogeneous group representing a line of evolution fundamentally distinct from filarioids. Similarly, species in Aproctoidea (which parasitize only birds) generally produce thick-shelled eggs containing fully developed first-stage larvae. They inhabit the air sacs and, unlike the diplotriaenoids, also the nasal cavities, orbits, and subcutaneous tissues of the neck and head. Reports of diplotriaenoids and aproctoids in organs (e.g., lungs, kidneys, liver, intestine) are likely incorrect and might result when delicate air sacs are disrupted at necropsy (Anderson 2000). Aproctoids may be more closely related to filarioids than are the

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diplotriaenoids; more information is required about their life cycles and development.

It is regrettable that confusion still occurs with respect to the superfamilies Filarioidea, Diplotriaenoidea, and Aproctoidea. Some confusion is understandable given the taxonomic challenge posed by the CIH keys for the nonexpert. Confusion is also fostered by the continued use of "filariid" and "filarioid" for collective reference to the three superfamilies (e.g., Bain et al. 1992; Chabaud and Bain 1994).

ETIOLOGY

Adult Filarioid Worms

All genera of avian filarioids are classified in the family Onchocercidae, and 16 genera are recognized. Only *Pelecitus* has species that parasitize both avian and nonavian hosts (Bartlett and Greiner 1986; Jiménez-Ruiz et al. 2004). Approximately 160 species are known, based mainly on descriptions of adult worms. Descriptions based only on MF are discouraged. There is one report of a biologically aberrant dog heartworm (*Dirofilaria immitis*) infection in a captive Humboldt Penguin (*Spheniscus humboldti*) (Sano et al. 2005), but this will not be discussed further.

Adult avian filarioids are slender, whitish nematodes with a thin cuticle. They are generally 1–5 cm in length, although shorter or longer specimens are not uncommon. Females are generally larger than males. In comparison to other nematode parasites of vertebrates, filarioids have a simple external morphology and thus they pose a taxonomic challenge for the nonexpert. Cephalic papillae are reduced and other cephalic structures (e.g., pseudolabia) are lacking. The caudal end similarly tends to be nonornate although males have caudal papillae and, in some species, caudal alae (wing-like structures) are present. Delicate or marked cuticular cross striations may occur over the length of the body. A few species have nonprominent lateral alae. Other cuticular ornamentation is lacking.

Microfilariae

Avian filarioids produce MF that are either blood-borne or skin-inhabiting. Microfilarial morphology can provide useful clues about generic identity, but is insufficient as the sole basis for identification of species which must be based on adult worms. Key features of MF are easily and best observed in wet mounts and can be obscured in dry blood smears. The latter is unfortunate as hematozoan surveys generally use dried smears; however, such surveys have contributed greatly to our general awareness of avian filarioids as common parasites of birds. Such surveys are not able to provide reliable information about the genus or species of filarioid, although reports do exist where identifications are based solely on MF. These should be viewed with caution and used in conjunction with additional information to make definitive identifications.

HOST RANGE AND DISTRIBUTION

A summary of the host distributions of avian filarioids is presented in Table 26.1 and can help guide a search for adult worms at necropsy. Here, the 150 plus families of birds (Howard and Moore 1984) are cross-referenced with the 16 genera of avian filarioids. It is clear from this information that *Pelecitus* is the most broadly distributed genus with reports from 17 avian orders, followed by *Chandlerella* in 13, and *Paronchocerca*, *Splendidofilaria*, and *Cardiofilaria* in 11. Also evident is how much work remains to be done with respect to the occurrence of these parasites in most avian families. For example, the family Phasianidae has received considerable study because of its economic importance and appears to contain the most genera (8). Similarly, avian families that are well represented in zoological collections and the pet trade have large numbers of reported genera. For example, 7 genera have been reported from the Psittacidae and 7 genera have been reported in both the Muscicapidae and Corvidae. Due to lack of data it is also premature to make conclusions about possible geographic trends in the distribution of avian filarioids, for example, whether some are more common in temperate zones versus tropics, or Nearctic versus Palearctic. However, cross-referenced information in Table 26.1 may help reveal biologically meaningful relationships, since parasite genera are ordered and grouped by subfamily in a scheme that seeks to reflect evolutionary relationships (Anderson and Bain 1976).

At the finer level of parasite genera and species, *Paronchocerca* appears unique among the genera of avian filarioids that contain numerous species. Each of the 17 species seems restricted to a single family of birds and some degree of host–parasite coevolution should be considered in attempting to understand why (Bartlett and Anderson 1986). As a genus, *Paronchocerca* occurs in a broad range of hosts but it is known predominantly from those orders generally considered "primitive" or those that occupy the "primitive" adaptive zone, that is, the aquatic and shoreline habitats. *Paronchocerca* may have become established in early Ornithurae and subsequently persisted in some of the "primitive" birds, as well as having transferred to "modern" groups, which now occupy the aquatic adaptive zone originally occupied by the earliest Ornithurae.

Table 26.1. Reports of avian filarioid genera in families of birds.

Bird orders and families	*Pelecitus*	*Struthiofilaria*	*Paronchocerca*	*Pseudlemdana*	*Striatofilaria*	*Aproctella*	*Cardiofilaria*	*Andersonfilaria*	*Chandlerella*	*Splendidofilaria*	*Dessetfilaria*	*Lemdana*	*Aproctiana*	*Sarconema*	*Eulimdana*	*Eufilaria*	Mf
Struthioniformes																	
1 Struthionidae		+	+						+								+
Rheiformes																	
2 Rheidae																	
Casuariiformes																	
3 Casuariidae																	
4 Dromaiidae									+								+
Apterygiformes																	
5 Apterygidae																	
Tinamiformes																	
6 Tinamidae			+														+
Sphenisciformes																	
7 Spheniscidae			+														+
Gaviiformes																	
8 Gaviidae										+							+
Podicipediformes																	
9 Podicipedidae	+																+
Procellariiformes																	
10 Diomedeidae																	
11 Procellariidae																	
12 Hydrobatidae																	
13 Pelecanoididae																	
Pelecaniformes																	
14 Phaethontidae																	
15 Pelecanidae									+								+
16 Sulidae																	
17 Phalacrocoracidae									+								+
18 Anhingidae	+		+														+
19 Fregatidae																	
Ciconiiformes																	
20 Ardeidae	+		+				+										+
21 Balaenicipitidae																	
22 Scopidae																	
23 Ciconiidae			+														+
24 Threskiornithidae	?																+
25 Phaenicopteridae					+												+
Anseriformes																	
26 Anhimidae																	
27 Anatidae	+									+				+			+
Falconiformes																	
28 Cathartidae																	+
29 Pandionidae	+																+
30 Accipitridae	+						+					+					+
31 Sagittariidae																	
32 Falconidae	?						+		+	+							+
Galliformes																	
33 Megapodiidae																	

(*continues*)

Table 26.1. (*Continued*)

Bird orders and families	*Pelecitus*	*Struthiofilaria*	*Paronchocerca*	*Pseudlemdana*	*Striatofilaria*	*Aproctella*	*Cardiofilaria*	*Andersonfilaria*	*Chandlerella*	*Splendidofilaria*	*Dessetfilaria*	*Lemdana*	*Aproctiana*	*Sarconema*	*Eulimdana*	*Eufilaria*	Mf
34 Cracidae	+																+
35 Phasianidae	+		+			+	+		+	+		+				+	+
36 Opisthocomidae																	+
Gruiformes																	
37 Mesitornithidae	+																+
38 Turnicidae																+	+
39 Pedionomidae																	
40 Gruidae									+								+
41 Aramidae																	
42 Psophiidae																	
43 Rallidae	+		+						+	+							+
44 Heliornithidae																	
45 Rhynochetidae																	
46 Eurypygidae																	+
47 Cariamidae																	
48 Otididae			+														+
Charadriiformes																	
49 Jacanidae																	+
50 Rostratulidae																	
51 Dromadidae																	
52 Haematopodidae																	
53 Ibidorhynchidae																	
54 Recurvirostridae																	
55 Burhinidae																	+
56 Glareolidae																	
57 Charadriidae							+								+		+
58 Scolopacidae			+			+	+								+		+
59 Thinocoridae																	
60 Chionididae																	
61 Stercorariidae																	
62 Laridae	+		?												+		+
63 Rynchopidae																	
64 Alcidae															+		+
Columbiformes																	
65 Pteroclididae						+											+
66 Columbidae	+					+	+		+	+					+		+
Psittaciformes																	
67 Loriidae																	+
68 Cacatuidae							+		+								+
69 Psittacidae	+					+	+		+	+			+		+		+
Cuculiformes																	
70 Musophagidae																	+
71 Cuculidae	+		+				+									+	+

Table 26.1. (*Continued*)

Bird orders and families	*Pelecitus*	*Struthiofilaria*	*Paronchocerca*	*Pseudlemdana*	*Striatofilaria*	*Aproctella*	*Cardiofilaria*	*Andersonfilaria*	*Chandlerella*	*Splendidofilaria*	*Dessetfilaria*	*Lemdana*	*Aproctiana*	*Sarconema*	*Eulimdana*	*Eufilaria*	Mf
Strigiformes																	
72 Tytonidae																	
73 Strigidae	+					+	+			+		+					+
Caprimulgiformes																	
74 Steatornithidae																	
75 Podargidae																	
76 Nyctibiidae																	
77 Aegothelidae																	+
78 Caprimulgidae									+								+
Apodiformes																	
79 Apodidae	+						+		+						+		+
80 Hemiprocnidae																	
81 Trochilidae																	
Coliiformes																	
82 Coliidae																	+
Trogoniformes																	
83 Trogonidae	+																+
Coraciiformes																	
84 Alcedinidae	+					+		+		+							+
85 Todidae																	
86 Momotidae																	+
87 Meropidae									+	+							+
88 Coraciidae	+																+
89 Brachypteraciidae																	
90 Leptosomatidae																	
91 Upupidae						+											+
92 Phoeniculidae																	
93 Bucerotidae												+					+
Piciformes																	
94 Galbulidae																	
95 Bucconidae																	+
96 Capitonidae																	+
97 Indicatoridae																	
98 Ramphastidae	+									+	+				+		+
99 Picidae	+		+			+	+		+								+
Passeriformes																	
100 Eurylaimidae																	+
101 Dendrocolaptidae	+																+
102 Furnariidae	+																+
103 Formicariidae	+						+										+
104 Conopophagidae																	
105 Rhinocryptidae																	
106 Cotingidae	+																+

(*continues*)

Table 26.1. (*Continued*)

Bird orders and families	*Pelecitus*	*Struthiofilaria*	*Paronchocerca*	*Pseudlemdana*	*Striatofilaria*	*Aproctella*	*Cardiofilaria*	*Andersonfilaria*	*Chandlerella*	*Splendidofilaria*	*Dessetfilaria*	*Lemdana*	*Aproctiana*	*Sarconema*	*Eulimdana*	*Eufilaria*	Mf
107 Pipridae																	
108 Tyrannidae	+					+				+							+
109 Oxyruncidae																	
110 Phytotomidae																	
111 Pittidae						+											+
112 Xenicidae																	
113 Philepittidae																	
114 Menuridae																	
115 Atrichornithidae																	
116 Alaudidae																	+
117 Hirundinidae									+	+							+
118 Motacillidae							+		+	+							+
119 Campephagidae							+									+	+
120 Pycnonotidae	+									+						+	+
121 Irenidae																	+
122 Laniidae						+	+			+							+
123 Vangidae																	
124 Bombycillidae						+											+
125 Dulidae																	
126 Cinclidae																	+
127 Troglodytidae																	
128 Mimidae										+							+
129 Prunellidae																	
130–142 Muscicapidae	+					+	+		+	+					+	+	+
143 Aegithalidae																	+
144 Remizidae																	
145 Paridae							+		+	+							+
146 Sittidae																	
147 Certhiidae									+	+							+
148 Rhabdornithidae																	
149 Climacteridae																	+
150 Dicaeidae																	+
151 Nectariniidae	+						+		+	+					+		+
152 Zosteropidae																	
153 Meliphagidae																	+
154–158 Emberizidae	+					+				+							+
159 Parulidae	+					+	+										+
160 Drepanididae																	
161 Vireonidae						+											+
162 Icteridae	+					+	+		+	+						+	+
163 Fringillidae						+				+							+
164 Estrildidae								+	+	+						+	+
165 Ploceidae	+									+						+	+
166 Sturnidae							+		+	+						+	+
167 Oriolidae							+		+								+

Table 26.1. (*Continued*)

Bird orders and families	*Pelecitus*	*Struthiofilaria*	*Paronchocerca*	*Pseudlemdana*	*Striatofilaria*	*Aproctella*	*Cardiofilaria*	*Andersonfilaria*	*Chandlerella*	*Splendidofilaria*	*Dessetfilaria*	*Lemdana*	*Aproctiana*	*Sarconema*	*Eulimdana*	*Eufilaria*	Mf
168 Dicruridae									+								+
169 Callaeidae																	
170 Grallinidae																	+
171 Artamidae																	+
172 Cracticidae																	+
173 Ptilonorhynchidae																	
174 Paradisaeidae																	+
175 Corvidae	+			+	+			+		+	+					+	+

Note: Parasite genera are arranged to group closely related taxa. Bird families follow Howard and Moore (1984); all families are included to help highlight where information is lacking. The "?" denotes an irresolvable question as to the species of avian filarioid in the genus noted (i.e., pertains to a *species inquirenda*). References available at www.integrativescience.ca.

Finally, at the level of individual avian hosts, concurrent infections with more than one species of *Chandlerella* and *Splendidofilaria* have been noted (e.g., Hibler 1963; Bartlett and Anderson 1980a). Concurrent infections with more than one species in the same filarioid genus might be restricted to parasite genera where adults of the different species tend to occupy very different locations. This diversity of anatomical sites is most commonly observed in the genera *Chandlerella* and *Splendidofilaria*.

The host distribution of avian filarioids is apt to be narrowest when vectors are host specific (e.g., most lice) and broader when vectors feed on a wide range of avian host species (e.g., blood-sucking dipterans).

EPIZOOTIOLOGY

Life Cycles and Life History Traits

The life cycles of avian filarioids follow the standard filarioid pattern that invariably involves a vertebrate definitive host and an invertebrate intermediate host, the latter referred to as the vector. Adult male and female worms in the vertebrate mate, and the females produce microfilariae that enter the host's blood or skin. Upon ingestion by a hematophagous arthropod, development proceeds to a so-called sausage stage and then to the second and finally to the infective third stage. Infective third-stage larvae migrate to the head and mouthparts of the arthropod and, while the arthropod feeds, break out of these locations and onto the vertebrate's skin. Given conditions of suitable moisture, they quickly enter the puncture wound made by the arthropod and gain entrance to the vertebrate's body. In the vertebrate, development continues to the fourth larval stage and finally the fifth or adult stage of development. An enduring mystery is how filarioids manage to find their highly specific, final locations in the body of the vertebrate host (Anderson 2000).

The first details about development of an avian filarioid in its intermediate host were provided by Anderson (1956) working with *Splendidofilaria fallisensis*, a parasite of American Black Ducks (*Anas rubripes*). Hibler (1963), working with *Splendidofilaria picacardina*, *Eufilaria longicaudata*, and *Chandlerella striatospicula* in Black-billed Magpies (*Pica hudsonia*), provided detailed information about development in the intermediate host as well as the first, and to this day only, details about morphological development of fourth-stage larvae in the avian host. In naïve magpies exposed to naturally infected vectors, third-stage larvae of *S. picacardina* migrate to the definitive site where adult worms are normally found, behind the semilunar valves in the myocardium. By 2 weeks postinfection (PI) in magpies, female fourth-stage larvae ($N = 4$) were 2.55–2.95 mm long (vs. 33–38 mm for mature adult females) and a male fourth-stage larva was 2 mm (vs. 11–20 mm for mature adult males). In laboratory-reared American Coots (*Fulica americana*) experimentally infected with *Pelecitus fulicaeatrae*, subadult fourth-stage worms were present in the definitive site of the ankles when birds were first examined 20 days PI (Bartlett and Anderson 1989). Morphologic changes continued during the fifth stage and details were noted

at 20, 30, and 55 days PI. Morphologic changes during this stage (other than those normally associated with differentiation of the reproductive tract) have been reported for filarioids in other vertebrate hosts, but are unusual among parasitic nematodes.

Among species that have blood-borne MF, the prepatent periods of *S. picacardina*, *C. striatospicula*, and *E. longicaudata* are 42–73, 37–53, 34–76 days, respectively (Hibler 1963), 21–36 days for *Cardiofilaria nilesi* (Niles et al. 1965; Niles and Kulasiri 1970), 30–36 days for *S. fallisensis* (Anderson 1956), and approximately 3 months for *Splendidofilaria californiensis* (Weinmann et al. 1979). Little is known about the prepatent period of avian filarioids that have skin-inhabiting MF. MF of *P. fulicaeatrae* appear in fluid adjacent to adult worms 210–265 days PI (Bartlett and Anderson 1989). Studies of other species of avian filarioids with skin-inhabiting MF are required to determine if they also have such apparently long prepatent periods.

Our understandings of the remarkably diverse locations of adult filarioids in birds are congruent with generalizations in Anderson (2000) who emphasized two points: (1) that filarioids have freed themselves from dependency on the food chain of their vertebrate hosts for transmission (cf. most other nematode parasites in vertebrate hosts) and thus have been able to radiate extensively throughout their hosts' bodies, and (2) that species have been reported from all organ systems and most tissues. Although adult avian filarioids are notoriously difficult to find, various insights and resources (Table 26.2) can help overcome the challenge. In addition, a general understanding of the locations occupied by adult filarioid worms in birds is extremely useful and these can be summarized by parasite subfamily and genus (Table 26.3). A more detailed summary is provided for *Paronchocerca*, *Splendidofilaria*, and *Chandlerella* in Table 26.4.

Adult female filarioids produce MF that are either blood-borne or skin-inhabiting, although in an "occult infection" adult worms will be present but not MF (Table 26.2). For many decades, only blood-borne MF were known for avian filarioids. Skin-inhabiting MF in birds were first discovered in Africa in Little Swifts (*Apus affinis* = *Cypselus affinis*) infected with *Eulimdana cypseli* (=*Filaria cypseli*). Unfortunately, the observation went unnoticed for years, as it was but a brief statement within a larger manuscript discussing a dog filarioid (Nelson 1962).

New awareness of skin-inhabiting MF in birds came with studies of *P. fulicaeatrae* in American Coots (Bartlett and Anderson 1987b) and various species of *Eulimdana* in birds in the order Charadriiformes (Bartlett et al. 1989; Bartlett and Anderson 1990; Bartlett 1992, 1993). However, we do not yet have a detailed understanding of the host range of filarioids that produce skin-inhabiting MF in birds.

The genera *Eulimdana* and *Pelecitus* are now known to have some species that produce skin-inhabiting MF and other species that produce blood-borne MF. Other genera where only blood-borne MF are currently known require additional study as this same diversity may occur elsewhere.

Some species of avian filarioids exhibit life history traits that are not known among the filarioids of other vertebrate hosts. Reproductive senescence and ephemerality were first recognized in studies of *P. fulicaeatrae* in American Coots and species of *Eulimdana* in charadriiforms (Bartlett et al. 1989; Bartlett and Anderson 1990; Bartlett 1992, 1993). These avian filarioids have skin-inhabiting MF and are transmitted by lice (order Phthiraptera). Reproductive senescence (curtailment of MF production while adult worms continue to live) and emphemerality (death of adult worms soon after females produce MF) both lead to a short period of MF production. This appears to be an adaptation to limit within-host transmission where continuous ingestion of MF might kill the louse vectors. Thus, reproductive senescence may have evolved in species that occupy sites where, if they were to die, they might initiate life-threatening inflammation (e.g., *Pelecitus fulicaeatrae* near joints in legs) (Anderson and Bartlett 1994). Ephemerality may have evolved in species in which adults occupy sites where, when they die, they are harmlessly resorbed (e.g., species of *Eulimdana* in the neck) (Anderson and Bartlett 1994).

Vectors

Since adult filarioids occur in tissue sites that do not connect with the passageways of the gastrointestinal, renal, or respiratory tracts of the bird host, they have evolved specialized means of transmission: all use hematophagous arthropod vectors (Anderson 2000). Vectors are known for 18 species of avian filarioids and include, in order of discovery, lice (order Phthiraptera) and flies (order Diptera, families Simuliidae, Culicidae, and Ceratopogonidae) (Table 26.5). Details of parasite development first emerged in studies using Simuliidae (Anderson 1956) and Ceratopogonidae (Hibler 1963).

At the generic level, it is apparent that avian filarioids may use more than one family or order of vector (Table 26.5). At the species level among those that use dipteran vectors, there appears to be restriction to one family of vectors, but more than one species in a particular vector genus may be used. *Splendidofilaria fallisensis* develops in two species of *Simulium* (Anderson 1956, 1968), and *E. longicaudata*, *Chandlerella quiscali*, and *Chandlerella chitwoodae* develop in at

Table 26.2. Knowledge, resources, and information to guide searches for adult filarioid worms in birds at necropsy.

Subject	Use	Resources to consult or information to consider	Additional information required (to be determined by examiner)	
			MF in bird*	Other
Host distribution	Determine possible filarioid genus or genera present in the species of bird being examined	Table 26.1	+ or −	Host species identification
Microfilarial morphology	Determine possible filarioid genus or genera present in the individual bird being examined	Table 26.7; Figures 26.2–26.15	+	Morphology of MF present
Sites occupied by adult worms, by genus	Determine priority location(s) to examine in bird	Tables 26.3 and 26.4	+	Possible filarioid genus or genera present
Ephemerality	Know that adult worms will not be present	In some filarioid species, adults die and are resorbed soon after they produce MF but MF are long lived	+	Possible filarioid genus or genera present
Occult infection	Know that adult worms will be present but MF will not be found in blood	In some host species, adult female filarioids produce MF but MF become trapped in inflammatory tissues around adult worms	−	
Prepatent infection	Know that adult worms will be present but MF will not be found	MF are not produced when adults are still immature	−	
Sterile infection	Know that adult worms will be present but MF will not be found	MF are not produced when only female or only males worms are present	−	

Note: The order of presentation is not prescriptive; circumstances at necropsy should determine use, sequence, and/or combination.
*This information must be determined by looking for microfilariae (MF) in the blood and skin of the bird. Microfilariae might be found (i.e., are present: +) or they might not be found (i.e., are absent: −).

least two species of *Culicoides* (Hibler 1963; Robinson 1971; Bartlett and Anderson 1980b). There may be less vector specificity among filarioids that use louse vectors; *Eulimdana bainae* and *Eulimdana wongae* develop in both ischnoceran and amblyceran lice (Bartlett 1993), which are generally viewed as different suborders of Phthiraptera (Chapter 29).

Bird–filarioid relationships, including the roles vectors play, have been discussed most recently by Bartlett and Anderson (1980a, b, 1986, 1987b), Bartlett and Greiner (1986), Hoberg (1986), and Bartlett (1992). Broad host ranges have been suggested for many avian filarioids (Bartlett et al. 1985; Bartlett and Greiner 1986). Filarioids that are not host specific can be sporadic, occult, or common in different bird species in the same bird community (Bartlett and Anderson 1980a). In contrast, Bartlett (1992) suggested narrower host distributions for other avian filarioids, especially those transmitted by lice, which are host specific. A different perspective, namely that most species of avian filarioids have very narrow host ranges, regardless of vector, leads to a proliferation of descriptions

Table 26.3. Sites in the avian host occupied by adult avian filarioids, by subfamily and genus.

Subfamily	Genus	Sites
Dirofilariinae	*Pelecitus*	• Near joints of legs, feet, or toes • Other*: subcutaneous in neck and around esophagus
Onchocercinae	*Struthiofilaria*	Body cavity
Splendidofilariinae	*Paronchocerca*	Diverse, see Table 26.4
	Pseudlemdana	• Generally: connective tissue around trachea and esophagus • Occasionally: subcutaneous connective tissue of head, neck, thorax, or thighs
	Striatofilaria	• Subcutaneous connective tissue of neck and connective tissue around trachea • Other†: thoracic cavity
	Aproctella	• Generally: body cavity or in association with heart • Occasionally: subcutaneous tissue of neck • Rarely†: kidney
	Cardiofilaria	• Generally: body cavity or in association with heart • Rarely†: subcutaneous connective tissue, lungs, hepatic portal vein
	Andersonfilaria	In fossa in dorsal wall of pelvic girdle under middle region of right kidney
	Chandlerella	Diverse, see Table 26.4
	Splendidofilaria	Diverse, see Table 26.4
	Dessetfilaria	• Outer wall of aorta • Other‡: cervical air sacs
Lemdaninae	*Lemdana*	• Subcutaneous connective tissue of head and neck, or connective tissue around trachea, esophagus, or crop • Other†: body cavity
	Aproctiana	Abdominal cavity
	Sarconema	• *Sarconema eurycerca*: heart • *Sarconema pseudolabiata*: subcutaneous connective tissue of neck
	Eulimdana	• Generally: subcutaneous connective tissue of head and neck, or connective tissue around trachea, esophagus, or crop • Rarely: body cavity
	Eufilaria	• Generally: subcutaneous connective tissue of head and neck, or connective tissue around trachea, esophagus, or crop • Occasionally: subcutaneous connective tissue of groin or legs

*Reports are likely misidentifications of species of *Lemdana* or *Eulimdana* (subfamily Lemdaninae).
†Report(s) requires confirmation and/or clarification.
‡Report is undoubtedly an error resulting when delicate air sacs are disrupted at necropsy.

Table 26.4. Sites in the avian host occupied by adult filarioids in the genera *Paronchocerca, Splendidofilaria,* and *Chandlerella.*

Sites in host		*Paronchocerca*	*Splendidofilaria*	*Chandlerella*
Circulatory system	Heart	*Paronchocerca ciconiarum* *Paronchocerca limboonkengi* *Paronchocerca tonkinensis* *Paronchocerca rousseloti* *Paronchocerca mirzai* *Paronchocerca straeleni* *Paronchocerca mansoni* *Paronchocerca bumpae* *Paronchocerca francolina* *Paronchocerca sonini*	*Splendidofilaria pavlovskyi* *Splendidofilaria travassosi* *Splendidofilaria brevispiculum* *Splendidofilaria verrucosa* *Splendidofilaria californiensis* *Splendidofilaria kasmirensis* *Splendidofilaria wehri* *Splendidofilaria alii* *Splendidofilaria pachacuteci* *S. longicaudata* *Splendidofilaria osmaniae*	*Chandlerella bosei* *Chandlerella skrjabini* *Chandlerella lerouxi* *Chandlerella pelecani* *Chandlerella singhi* *Chandlerella buckleyi* *Chandlerella himalayansis* *Chandlerella alii* *Chandlerella apusi* *Chandlerella longicaudata* *Chandlerella sultana*
	Behind heart valves		*Splendidofilaria picacardina*	
	Aorta		*Splendidofilaria travassosi*	
	Pulmonary arteries (walls and/or lumen)	*Paronchocerca ciconiarum* *Paronchocerca rousseloti*	*Splendidofilaria periarterialis* *Splendidofilaria algonquinensis* *Splendidofilaria caperata*	
	Other blood vessels			*Chandlerella sinensis* *Chandlerella apusi*
	Lumen of blood vessels			*Chandlerella bosei*
Abdominal cavity		*Paronchocerca bambusicolae* *Paronchocerca ibanezi* *Paronchocerca schelupovi*	*Splendidofilaria smithi* *Splendidofilaria verrucosa* *Splendidofilaria grettillati* *Splendidofilaria kasmirensis*	*Chandlerella bosei* *Chandlerella stantchinski** *Chandlerella sinensis* *Chandlerella columbigallinae* *Chandlerella lienalis* *Chandlerella thapari* *Chandlerella columbae* *Chandlerella inversa*
Liver				*Chandlerella bosei* *Chandlerella sinensis* *Chandlerella lienalis* *Chandlerella hepatica*
Spleen				*Chandlerella lienalis*
Kidney				*Chandlerella shaldybini*
Lungs		*Paronchocerca papillatus* *Paronchocerca thapari* *Paronchocerca francolina* *Paronchocerca sonini* *Paronchocerca struthiones*	*Splendidofilaria falconis*	*Chandlerella bosei* *Chandlerella sinensis*
Connective tissues	Trachea/esophagus		*Splendidofilaria skrjabini*	*Chandlerella sinensis* *Chandlerella chitwoodae* *Chandlerella striatospicula*
	Blood vessels (general)			*Chandlerella chitwoodae*
	Splenic artery			*Chandlerella striatospicula*
	Intestine			*Chandlerella striatospicula*
	Lymphatics			*Chandlerella bushi*

(*continues*)

Table 26.4. (*Continued*)

Sites in host		*Paronchocerca*	*Splendidofilaria*	*Chandlerella*
Subcutaneous tissues	Nonspecific locations		*Splendidofilaria smithi* *Splendidofilaria fallisensis* *Splendidofilaria singhi* *Splendidofilaria columbensis* *Splendidofilaria hibleri*	*Chandlerella robinsoni*
	Head	*Paronchocerca rousseloti*	*Splendidofilaria singhi*	
	Neck	*Paronchocerca rousseloti*	*Splendidofilaria singhi*	
	Chest		*Splendidofilaria pectoralis*	
	Thighs		*Splendidofilaria gedoelsti*	
Brain		*Paronchocerca helicina*		*Chandlerella quiscali*
Eyes			*Splendidofilaria smithi* *Splendidofilaria rotundicephala*	*Chandlerella petrowi*
Bursa of knee			*Splendidofilaria mavis*	
Feet	Calcaneal region		*Splendidofilaria bohmi*	
	Subcutaneous tissues		*Splendidofilaria tuvensis*	

Note: References available at www.integrativescience.ca.
*Correction of original report as "air sacs."

of species; this perspective is not held by this author.

CLINICAL SIGNS

Little has been reported about the clinical signs of infections with avian filarioids because very few species are pathogenic and, even with those, infections are generally subclinical. Moreover, clinical disease, when noted, may involve only a few birds. Reported signs include reduced body weight (Seegar 1979a) and acute depression (www.wildlifeinformation.org[1]) in association with *Sarconema eurycerca*, swollen joints and/or lameness with *Pelecitus* spp. (Greve et al. 1982; Paster 1983; Allen et al. 1985; Kummerfeld and Daugschies 1989), feather loss with *Eulimdana clava* (Eslami 1987; Gharagozlou 1988; Pizarro et al. 1994), and torticollis and progressive ataxia with *C. quiscali* (Law et al. 1993).

PATHOLOGY

MF are generally considered nonpathogenic although there are exceptions. Of note with respect to bloodborne MF is the chronic inflammation caused by MF in the walls of the pulmonary arteries of American Crows (*Corvus brachyrhynchos*) infected with *Splendidofilaria caperata*. MF are trapped in situ and the infection is "occult" (see Point #4 below). Of note with respect to skin-inhabiting MF is the possibility that they are the cause of the disease associated with *E. clava* in domestic Rock Pigeons (*Columba livia*) (see Point #3 below).

Few adult avian filarioids provoke overt disease in a live bird or gross tissue damage that can be observed at necropsy. Adults of a species of avian filarioid that are pathogenic in one or more species of birds may not be pathogenic in other species. Similarly, a species that is pathogenic in an individual bird may not be pathogenic in other birds of the same species. Known pathogens include the following:

1. *Splendidofilaria eurycerca.* This filarioid has been reported from various species of geese and swans (Anatidae). Adults live under the epicardium or within the myocardium. Pathology includes weakness and enlargement of the heart, myocardial hemorrhage, and myocardial inflammation, necrosis, and eventually fibrosis (www.wildlifeinformation.org; Quortrup and Holt 1940; Cowan 1946; Kluge 1967; Irwin 1975; Cole 1999) (Figure 26.1). Severity of lesions increases with increasing numbers of worms.
2. *Pelecitus* spp. Swellings and nodules in the legs and feet, especially near joints, and tenosynovitis

[1]S. Boardman, and Bourne, D. C. (eds). Waterfowl: Health and management waterfowl diseases, Available at http://wildlife1.wildlifeinformation.org/List_Vols/Waterfowl_Mod/Wildpro_Waterfowl_Cont.htm; and *Sarconema eurycerca*, Available at http://wildlife1.wildlifeinformation.org/S/00dis/Parasitic/Heartworm_Sarconema_Infection.htm.

Table 26.5. Known vectors of avian filarioids.

Vector	Filarioid species	Avian or arthropod host	Reference
Order Phthiraptera	*Eulimdana cypseli*	Little Swift (*Apus affinis* =*Cypselus affinis*)	Dutton (1905); Nelson (1962)
	Eulimdana bainae	Whimbrel (*Numenius phaeopus*)	Bartlett (1992)
	Eulimdana wongae	Marbled Godwit (*Limosa fedoa*)	Bartlett (1992)
	Sarconema eurycerca	Tundra Swan (*Cygnus columbianus*)	Seegar et al. (1976); Cohen et al. (1991)
	Pelecitus fulicaeatrae	American Coot (*Fulica americana*)	Bartlett and Anderson (1987a, b)
Order Diptera			
Family Simuliidae	*Splendidofilaria fallisensis*	American Black Duck (*Anas rubripes*); domestic duck (*Anas platyrhynchos*)	Anderson (1956, 1968)
	Splendidofilara sp. (presumed*)	Blackfly† (*Simulium* sp.)	Fukuda et al. (2005)
Family Culicidae	*Cardiofilaria nilesi*	Mosquito‡ (*Mansonia crassipes*)	Niles (1962); Niles et al. (1965); Dissanaike and Fernando (1965); Dissanaike and Niles (1967)
	Pelecitus ceylonensis	Mosquito§ (*Mansonia crassipes*)	Niles et al. (1965); Dissanaike (1967); Dissanaike and Niles (1967)
	Aproctella alessandroi	Blue-gray Tanager (*Thraupis episcopus*)	Bain et al. (1981)
Family Ceratopogonidae	*Chandlerella striatospicula*	Black-billed Magpie (*Pica hudsonia*)	Hibler (1963)
	Chandlerella quiscali	Common Grackle (*Quiscalus quiscula versicolor*)	Robinson (1971)
	Chandlerella chitwoodae	Common American Crow (*Corvus brachyrhynchos*)	Bartlett and Anderson (1980b)
	Splendidofilaria picacardina	Black-billed Magpie (*Pica hudsonia*)	Hibler (1963)
	Splendidofilaria californiensis	California Quail (*Callipepla californica*)	Weinmann et al. (1979); Atchley and Wirth (1975)
	Eufilari longicaudata	Black-billed Magpie (*Pica hudsonia*)	Hibler (1963)
	Eufilaria bartlettae	Eurasian Blackbird (*Turdus merula*)	Bain (1980)
	Eufilaria delicata	Eurasian Blackbird (*Turdus merula*)	Bain (1980)
	Eufilaria kalifai	Eurasian Magpie (*Pica pica pica*)	Millet and Bain (1984)

Note: Filarioid species are grouped by vector, chronological order of discovery, and then parasite genus.

*The larvae found were presumed to be a species of *Splendidofilaria*.

†The wild bird host was not known.

‡The wild bird host was not known by Niles (1962) who found infective filarioid larvae in mosquitoes. These were experimentally inoculated into chickens by Niles et al. (1965) and the adult worms recovered were described as the new species *Cardiofilaria nilesi* by Dissanaike and Fernando (1965). The infective larvae were described by Dissanaike and Niles (1967).

§The wild bird host was not known by Niles et al. (1965) who found two types of infective filarioid larvae in mosquitoes. Larvae of one of these types were similar to those of Niles (1962). Larvae of the second type were experimentally inoculated into "ash-doves" by Dissanaike (1967) who described the adult worms recovered as the new species *Pelecitus nilesi*, and also reported the species in wild "crows." The infective larvae were described by Dissanaike and Niles (1967).

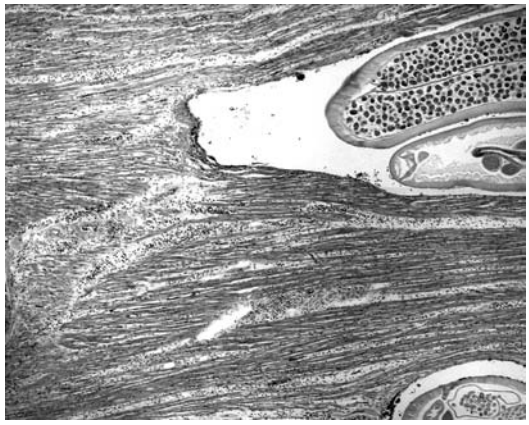


Figure 26.1. Heart of a Tundra Swan (*Cygnus columbianus*) with myocarditis due to *Sarconema eurycerca*. At this magnification, separation of myofibers is evident with interstitial hemorrhage, and infiltration with mixed inflammatory cells including heterophils, eosinophils, macrophages, and perivascular lymphocytes. Developing embryos are visible in the ovary of the parasite (top section). In more advanced cases, necrosis and fibrosis may be present in the myocardium. Hematoxylin and eosin; 400×. Photomicrograph Courtesy of US Geological Survey, National Wildlife Health Center.

have been reported in psittaciformes that harbor *Pelecitus* spp. (e.g., Greve et al. 1982; Paster 1983; Allen et al. 1985; Kummerfeld and Daugschies 1989). Reports of disease in psittaciformes due to *"Pelecitus*" in the neck region are likely misidentifications of worms in the genus *Eulimdana*. The adults of *Pelecitus fulicaeatrea* occur near the ankles and generally provoke swellings in Red-necked Grebes (*Podiceps grisegena*) but rarely in American Coots (Bartlett and Anderson 1989). In such rare cases in coots, worms were generally within soft, thin-walled capsules but a note was also made of worms within a fibrous, thick-walled capsule. Histopathologic changes in the tendon sheaths adjacent to adult worms included synovial cell hypertrophy, hypervascularization, inflammatory cell infiltration, and mild fibrosis.

3. *Eulimdara clava*. Disease has been associated with infections of *E. clava* in domestic Rock Pigeons in Iran (Eslami 1987; Gharagozlou 1988). Clinical signs included loss of feathers in the head, neck, back, and wings. One report indicated that MF were not found in blood where they were expected although MF were present in female worms (Eslami 1987). It is possible these clinical signs were related to skin-inhabiting MF, which are now known for other species of *Eulimdana* and may also occur with *E. clava*. In 1994, Pizarro et al. reported that a pigeon that harbored adult *E. clava* (identified as "*Pelecitus clavus*") had MF in subcutaneous tissues immediately adjacent to adult worms in the neck but the broader possibility that MF were skin-inhabiting was not considered. "Peritracheal filariosis" described in pigeons (Guidal and Settnes 1968; Rutherford and Black 1974) undoubtedly refers to adult *E. clava* and the MF reported in the lungs (i.e., in blood) were probably of a different species and not those of *E. clava*.
4. *Splendidofilaria caperata*. In American Crows, infection with *S. caperata* is accompanied by chronic inflammation that is caused by MF near adults living in tortuous channels within the walls of the pulmonary arteries; MF become trapped in the inflamed tissues and are not found in the blood (Bartlett and Anderson 1981). Such amicrofilaremic infections are called "occult." Some inflammation was reported around adult worms in Black-billed Magpies infected with this same parasite but MF were present in blood (Hibler 1963). Pathology has not been noted with *S. caperata* in various other wild birds, including five more passeriform species and a species in each of Strigiformes, Coraciiformes, and Gruiformes (Bartlett and Anderson 1985, 1987a, c).
5. *Species with adults in "sensitive" locations.* Adults of species of *Paronchocerca, Chandlerella*, and *Splendidofilaria* live in various locations in birds (Table 26.4), and lesions have been reported in some cases where adults live in the muscles or lumen of the heart, walls or lumen of major blood vessels, or brain. The occurrence of *S. californiensis* in nodules on the luminal surface of the aorta elicits a relatively mild inflammation, but can occlude 20–60% of the aortic orifice and interfere with movement of aortic valves, thus compromising cardiac output (Weinmann et al. 1979). In addition to *S. caperata*, adults of *Splendidofilaria algonquinensis* live in the pulmonary arteries and cause lesions in House Sparrows (*Passer domesticus*) (Huizinga et al. 1971), although infections are not occult. Adults of *C. quiscali* live in the brain. Clinical signs and pathology were associated with this parasite in farmed Emus (*Dromaius novaehollandiae*) (Law et al. 1993), but lesions have

not been reported in infected wild passeriforms in North America including American Crows, Blue Jays (*Cyanocitta cristata*), Brown-headed Cowbirds (*Molothrus ater*), Common Grackles (*Quiscalus quiscula versicolor*), and (introduced) European Starlings (*Sturnus vulgaris*). While adult *Paronchocerca ciconarium* were associated with cardiovascular lesions in a Marabou Stork (*Leptoptilos crumeniferus*) that died in a zoo (Ensley 1978), the role of *Paronchocerca* spp. in the deaths of other zoo birds is not clear (Bartlett and Anderson 1986; Nicholls et al. 1995). *Splendidofilaria eurycerca* in anatids is another example of a filarioid whose adults live in a "sensitive location," namely, the muscles of the heart. It can be pathogenic although this is not inevitable. Intensity of infection and overall condition of the avian host are important.

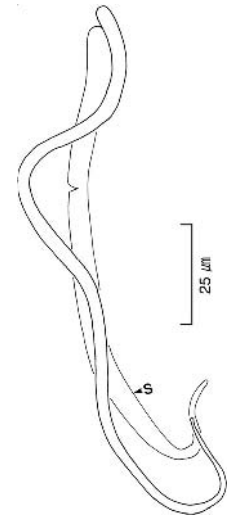


Figure 26.2. Morphology of microfilariae of *Andersonfilaria africanus*, Giemsa-stained thin blood smear. S, sheath (which has detached from body). *Note:* Microfilaria was obtained from the blood or skin of the avian host unless otherwise indicated. Caution is advised when morphological information based on microfilariae from uteri of female worms is used (see text). Adapted from Figure 73 in Bartlett and Bain (1987) and reproduced with permission of the *Comparative Parasitology*, formerly *Proceedings of the Helminthological Society of Washington*.

DIAGNOSIS

Finding adult worms at necropsy is a critical step in identifying species and can be aided by diverse information (Table 26.2) including the genera of filarioids previously reported in the relevant avian host family (Table 26.1) and the locations that adult parasites in these genera tend to occupy in the bird (Tables 26.3 and 26.4). A key to genera has been provided by Bartlett and Anderson (1987a), along with the 31 synonyms for the 16 recognized genera. References for recent taxonomic reviews (or for the original generic proposals) are provided in Table 26.6. An exhaustive list of species, synonyms, taxonomic authorities, and other references can be found at www.integrativescience.ca.

The finding of adult filarioid worms at necropsy can be greatly facilitated by determining what morphological type(s) of MF are present (Table 26.7, Figures 26.2–26.15). It is important, therefore, to use appropriate techniques to look for MF. When a carcass (fresh, refrigerated, or thawed) is examined, a small piece of lung and skin can be torn into tiny pieces in a few drops of physiologic saline on a microscope slide. After removing tissue bits, an equal volume of 2–5% formalin is added, and the preparation is covered with a vaseline-ringed microscope cover glass. The resulting wet mount is then examined by microscopy for MF using a compound microscope with a 10× objective lens or higher magnification. A slight modification involves the addition of a vital stain (brilliant cresyl blue or Giemsa) to a saline-only preparation before the cover glass is applied. Skin snips (2–3 mm^2) should be taken from sites at or near the locations where adult worms are expected (e.g., legs for *Pelecitus* spp. and neck for *Eulimdana* spp.), regardless of whether birds are dead or alive.

When a live bird is available, blood samples can be taken, preferably using techniques that do not involve drying the blood. The hematocrit centrifuge technique enables study of MF in a wet mount preparation (Woo 1971). After centrifugation, the hematocrit capillary tube is broken at the interface between serum and cells where MF concentrate and MF are then expressed onto

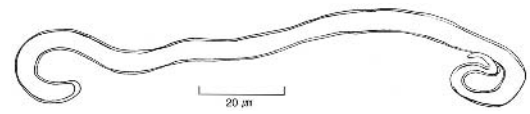


Figure 26.3. Morphology of microfilariae of *Aproctella stoddardi*, Giemsa's stain. Note as given in Figure 26.2. Adapted from Figure 7 in Anderson (1957) and reproduced with permission of the *Canadian Journal of Zoology*.

Table 26.6. The 16 genera of avian filarioid nematodes from the family Onchocercidae.

Subfamily	Number of valid species	Reference for recent taxonomic review (or proposal for new genus)
Dirofilariinae		
1. *Pelecitus*	16	Bartlett and Greiner (1986)
Onchocercinae		
2. *Struthiofilaria*	1	Noda and Nagata (1976)
Splendidofilariinae		
3. *Paronchocerca*	17	Bartlett and Anderson (1986)
4. *Pseudlemdana*	1	Sonin (1968)
5. *Striatofilaria*	1	Sonin (1968)
6. *Aproctella*	7	Anderson (1957)
7. *Cardiofilaria*	14	Bartlett and Anderson (1980a)
8. *Andersonfilaria*	2	Bartlett and Bain (1987)
9. *Chandlerella*	26	Bartlett and Anderson (1980a)
10. *Splendidofilaria*	31	Bartlett and Anderson (1980a)
11. *Dessetfilaria*	2	Bartlett and Bain (1987)
Lemdaninae		
12. *Lemdana*	9	Bartlett and Anderson (1987a)
13. *Aproctiana*	1	Sonin (1968)
14. *Sarconema*	2	Sonin (1966)
15. *Eulimdana*	16	Bartlett et al. (1985) and Bartlett (1992)
16. *Eufilaria*	14	Bartlett and Anderson (1980a)

Note: Genera are grouped by subfamily, with information provided as to the numbers of valid species and references for recent taxonomic reviews or original generic proposals (see www.integrativescience.ca for the taxonomic authorities, 31 generic synonyms, and exhaustive references).

Table 26.7. Key morphological traits of microfilariae of avian filarioids in different genera.

Morphological traits of microfilariae	*Pelecitus*	*Struthiofilaria*	*Paronchocerca*	*Pseudlemdana*	*Striatofilaria*	*Aproctella*	*Cardiofilaria*	*Andersonfilaria*	*Chandlerella*	*Splendidofilaria*	*Dessetfilaria*	*Lemdana*	*Aproctiana*	*Sarconema*	*Eulimdana*	*Eufilaria*
Sheath																
Present	+	+	+			+		+	+			+		+	+	
Absent			+			+	+			+	+					+
Tail																
Broadly rounded			+						+	+	+					
Sharply pointed	+	+					+								+	+
Tapering rounded	+		+			+		+				+				
Length																
$\leq$200 μm	+		+						+	+	+				+	+
$\sim$ 200 μm	+					+		+				+				
$\geq$200 μm	+	+					+					+		+		

Note: Parasite genera are arranged to group closely related taxa, and all 16 genera are included to highlight where information is lacking. References available at www.integrativescience.ca. Information in this table should be viewed as "suggestive" rather than "diagnostic" since exceptions may occur in the pattern portrayed for each genus.

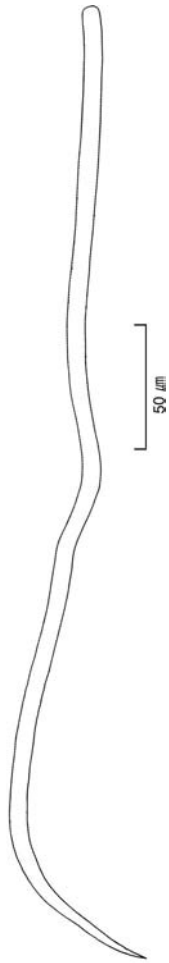


Figure 26.4. Morphology of microfilariae of *Cardiofilaria pavlovskyi,* cresyl blue vital stain in wet mount. Note as given in Figure 26.2. Adapted from Figure 3 in Bartlett and Anderson (1980a) and reproduced with permission of the *Systematic Parasitology.*

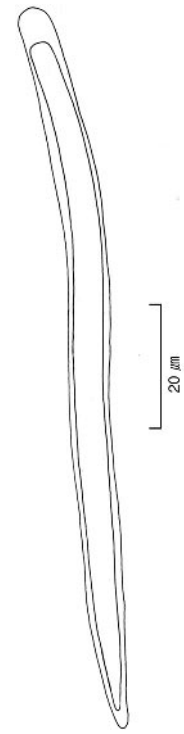


Figure 26.5. Morphology of microfilariae of *Paronchocerca ciconiarum,* in glycerin from uterus of female worm. Note as given in Figure 26.2. Adapted from Figure 55 in Bartlett and Anderson (1986) and reproduced with permission of the *Canadian Journal of Zoology.*

a microscope slide for preparation of a wet mount. Techniques described for mammalian blood do not necessarily work well for the nucleated erythrocytes of avian blood and Seegar (1979b) and Holmstad et al. (2003) have explored more appropriate techniques. Dried, stained thin blood smears are useful within the limitations previously mentioned.

Some information about MF morphology in the scientific literature (e.g., Figures 26.5 and 26.6) is based on specimens from the vagina or uteri of female worms (especially females preserved in fixatives required for taxonomic study). Caution is advised as these MF tend to be shorter than MF in blood or skin and the presence or absence of a sheath can be difficult to definitively ascertain.

IMMUNITY

Very little is known about avian immunity in the context of filarioid infections in wild birds. The unusual life history traits of ephemerality and reproductive senescence may be genetically controlled, but it is also possible that they are mediated by host immune processes (Anderson and Bartlett 1994).

MF of *C. nilesi* disappeared from the blood of experimentally infected chickens (*Gallus gallus*), leading Gooneratne (1969) to suggest an immune response that affects MF but not adults. Niles and Kulasiri (1970) indicated such immunity might not be relevant and further study was required. It should be noted that chickens (order Galliformes) are not the normal host of *C. nilesi* and that the parasite has been reported

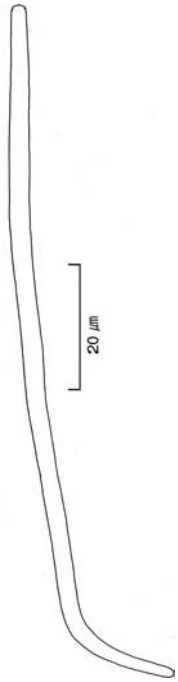


Figure 26.6. Morphology of microfilariae of *Paronchocerca struthionus*, in glycerin from uterus of female worm. Note as given in Figure 26.2. Adapted from Figure 21 in Bartlett and Anderson (1986) and reproduced with permission of the *Canadian Journal of Zoology*.

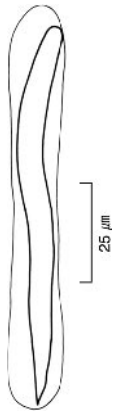


Figure 26.7. Morphology of microfilariae of *Eulimdana metcalforum*, in 2–5% formalin-saline wet mount. Note as given in Figure 26.2. Adapted from Figure 22 in Bartlett (1992) and reproduced with permission of the *Systematic Parasitology.*

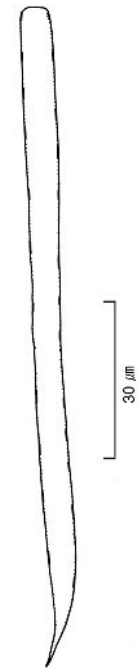


Figure 26.8. Morphology of microfilariae of *Eufilaria longicaudata,* cresyl blue vital stain in wet mount. Note as given in Figure 26.2. Adapted from Figure 46 in Bartlett and Anderson (1980a) and reproduced with permission of the *Systematic Parasitology.*

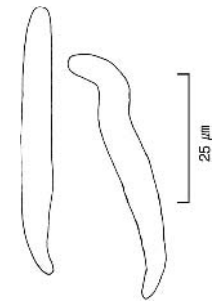


Figure 26.9. Morphology of microfilariae of *Dessetfilaria guianensis*, hematein-stained thin smear. Note as given in Figure 26.2. Adapted from Figure 72 in Bartlett and Bain (1987) and reproduced with permission of the *Comparative Parasitology*, formerly *Proceedings of the Helminthological Society of Washington.*

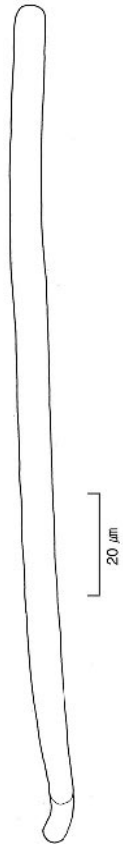


Figure 26.10. Morphology of microfilariae of *Chandlerella bushi*, Giemsa-stained thin smear. Note as given in Figure 26.2. Adapted from Figure 11 in Bartlett and Anderson (1987c) and reproduced with permission of the *Canadian Journal of Zoology*.

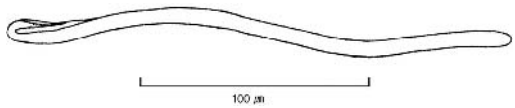


Figure 26.11. Morphology of microfilariae of *Struthiofilaria megalocephala*, unspecified preparation technique. Note as given in Figure 26.2. Adapted from Figure 8 in Noda and Nagata (1976) and reproduced with permission of the *Bulletin of the University of Osaka Prefecture*.

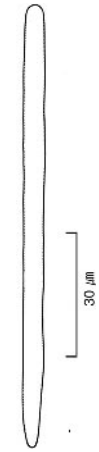


Figure 26.12. Morphology of microfilariae of *Splendidofilaria caperata*, cresyl blue vital stain in wet mount. Note as given in Figure 26.2. Adapted from Figure 28 in Bartlett and Anderson (1980a) and reproduced with permission of the *Systematic Parasitology*.

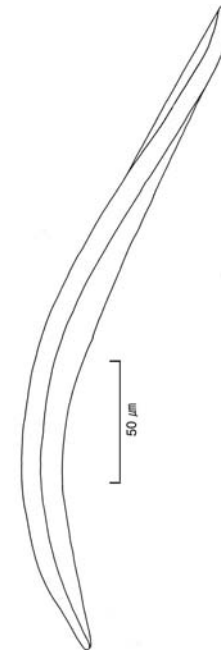


Figure 26.13. Morphology of microfilariae of *Lemdana wernaarti*, cresyl blue vital stain in wet mount. Note as given in Figure 26.2. Adapted from Figure 18 in Bartlett and Anderson (1987a) and reproduced with permission of the *Canadian Journal of Zoology*.

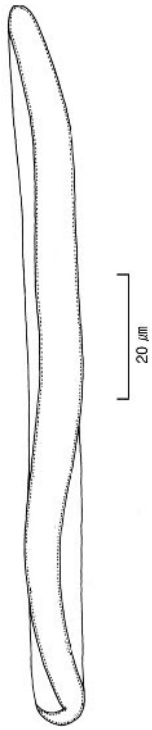


Figure 26.14. Morphology of microfilariae of *Pelecitus fulicaeatrae,* cresyl blue vital stain in wet mount. Note as given in Figure 26.2. Adapted from Figure 6 in Bartlett and Anderson (1987b) and reproduced with permission of the *Canadian Journal of Zoology.*

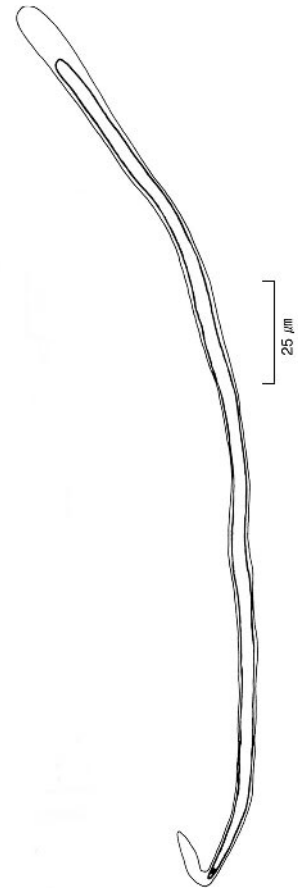


Figure 26.15. Morphology of microfilariae of *Pelecitus tubercauda,* Giemsa-stained thin smear. Note as given in Figure 26.2. Adapted from Figure 3 in Vanderburgh et al. (1984) and reproduced with permission of the *Canadian Journal of Zoology.*

in wild Spotted Doves (*Streptopelia chinensis*) (order Columbiformes) (Lee and Amin-Babjee 1986).

PUBLIC AND DOMESTIC ANIMAL HEALTH CONCERNS

There are few known public or domestic animal health concerns associated with avian filarioids in wild birds and the possibility has received little study. Clinical signs and pathology were associated with *C. quiscali* in farmed Emus (Law et al. 1993).

WILDLIFE POPULATION IMPACTS

Little is known about the impact of filarioids on bird populations and this aspect of their biology has been rarely studied. Even with *S. eurycerca*, which is the best known pathogen among all avian filarioids, the overall conclusion is that "this parasite has not received sufficient study for its full host range, its relative frequency of occurrence in different species, or its significance as a mortality factor for wild birds to be determined" (Cole 1999). Lead poisoning, gunshot wounds and other trauma, an abundance of lice, and the presence of other nematode parasites and pathogens confound efforts to determine the impact of *S. eurycerca* on the health of individual wild birds as well as populations (Quortrup and Holt 1940; Cowan 1946; Holden and Sladen 1968; Irwin 1975; McKelvey and MacNeill 1981; Cohen et al. 1991). Two studies attempted to document population impacts from filarioids in galliforms. Data were inconclusive about the impact of *S. californiensis*, a heartworm, in populations of California Quail (*Callipepla californica*) (Weinmann et al. 1979).

Splendidofilaria smithi (identified as "*Splendidofilaria papillocerca*") in Willow Ptarmigan (*Lagopus lagopus*) was negatively correlated with population growth rates of the host (Holmstad et al. 2005).

TREATMENT AND CONTROL

Infections with avian filarioids are rarely of concern and usually no treatment is indicated. Levamisole hydrochloride was ineffective in eliminating MF from the blood of an otherwise clinically normal Marabou Stork in a zoo (Ensley 1978). Use of levamisole, fenbendazole, and mebendazole has been suggested for psittaciformes (Paster 1983). Treatment of Emus with ivermectin appeared to prevent clinical signs (Law et al. 1993) and this compound has been used to treat an Alexandrine Parakeet (*Psittacula eupatria*) that had adult worms removed from one eye (Kummerfeld and Daugschies 1989). Drugs that kill adult worms in situ may lead to more severe consequences than those posed by the original infection since dead worms may initiate inflammatory responses and lesions that adversely affect normal physiological function. Nonintervention is likely best and is consistent with the fact that neither disease nor pathology has been reported for the vast majority of the 160 known species of avian filarioids.

ACKNOWLEDGMENTS

This chapter is dedicated to the late Roy C. Anderson, who made substantial contributions to the taxonomy, classification, and biology of the avian filarioids. The author acknowledges, with gratitude, the invaluable assistance of Prune Harris and Kristy Read in the preparation of this chapter and of Cathy Chisholm in the facilitation of online access to the CABI database.

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27
Capillarid Nematodes

Michael J. Yabsley

INTRODUCTION

Avian parasites that belong to the genera *Baruscapillaria*, *Capillaria*, *Echinocoleus*, *Eucoleus*, *Ornithocapillaria*, *Pterothominx*, and *Tridentocapillaria* are collectively referred to as the capillarids. The capillarids are small thin nematodes in the superfamily Trichinelloidea, family Trichuridae, and subfamily Capillarinae, and are related to *Trichinella* and *Trichuris*. The taxonomy of this group has changed extensively but currently there are 24 genera of capillarids that infect all classes of vertebrates. These parasitize various tissues including the gastrointestinal tract, respiratory system, urinary bladder, subcutaneous tissues, and liver. Representatives from seven of these genera infect birds and all of them parasitize regions of the gastrointestinal tract, either singly or in combination, which may be specific for particular capillarid species. For example, *Eucoleus annulatus* and *Eucoleus contortus* are found in the crop and esophagus of chickens, *Pterothominx caudinflata*, *Pterothominx bursata*, and *Baruscapillaria obsignata* are found in the small intestine, and *Capillaria anatis* is found in the ceca.

Although prevalence of infection in some avian species can be high, clinical disease among free-ranging birds is uncommon and effects are more at the level of an individual than at the level of entire populations. This is likely because the intensity of infection is often low. The life cycle of many capillarids is direct, so there is a high risk of transmission and disease among captive birds in poorly maintained facilities where worm burdens can be high.

The natural history of several species that infect domesticated birds is well known, but the ecology of capillarids that infect wild birds is poorly understood. Detailed morphologic examination is necessary to identify capillarids to the proper genus and species. Their lack of distinguishing characteristics has led to numerous revisions in classification and large numbers of synonyms for recognized species.

SYNONYMS

Capillariasis, hairworm, threadworm, cropworm.

HISTORY

The capillarids have historically been placed in numerous genera. Many of the early species descriptions were inadequate and based on only a few specimens or, in some cases, fragments of worms. This led to extensive revisions in taxonomy and changes in our understanding of capillarid–host relationships since *C. anatis* was first described from a clinically normal domestic duck over 200 years ago (see Gower 1939). Skrjabin et al. (1957) conducted an extensive review of capillarid taxonomy and proposed five genera: *Capillaria*, *Eucoleus*, *Hepaticola*, *Skrjabinocapillaria*, and *Thominx*. During the next 20 years, 15 additional genera were described. Anderson and Bain (1982) synonymized all 20 genera of capillarids into the genus *Capillaria* because of confusion stemming from morphologic criteria that were used to define genera. Although none of the proposed schemes are universally accepted, the classification proposed by Moravec (1982) and used for subsequently described genera is the most widely accepted. This taxonomy will be retained in this chapter. Twenty-four genera have been proposed to date (Moravec 1982; Moravec et al. 1987; Barus and Sergejeva 1990a, b; De and Maity 1995; Moravec et al 1999): *Amphibiocapillaria*, *Aonchotheca*, *Baruscapillaria*, *Calodium*, *Capillaria*, *Capillostrongyloides*, *Crocodylocapillaria*, *Echinocoleus*, *Eucoleus*, *Freitascapillaria*, *Huffmanela*, *Indocapillaria*, *Ornithocapillaria*, *Paracapillaria*, *Paracapillaroides*, *Paratrichosoma*, *Pearsonema*, *Piscicapillaria*, *Pseudocapillaria*, *Pseudocapillarioides*, *Pterothominx*, *Schulmanela*, *Tenoranema*, and *Tridentocapillaria*. The capillarid species that infect birds are found in seven of these genera: *Baruscapillaria*, *Capillaria*, *Echinocoleus*, *Eucoleus*, *Ornithocapillaria*, *Pterothominx*, and *Tridentocapillaria*. Where older species names are still commonly

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used, I have listed the currently accepted name first and followed it by the old name. Because the taxonomy is confusing, some proposed synonyms are not included because most scientists consider them to be valid species. For example, Madsen (1951) and Cram (1936) consider *Eucoleus perforans* and *E. annulatus* to be synonyms of *E. contortus*, but most researchers accept all three as distinct species.

DISTRIBUTION AND HOST RANGE

The majority of capillarid species have been reported from two or more related avian species and a few are known to infect a wide range of avian host species (e.g., *E. contortus* infects birds in nine orders). These highly adaptive species have a cosmopolitan distribution because of the wide geographic range of the numerous hosts they infect. Some capillarids have a narrower host range and are only found in areas where suitable hosts are present. For example, *Pterothominx moraveci* infects only Port Lincoln Parrots (*Barnardius zonarius*) while *Eucoleus frugilegi* infects a range of species in the family Corvidae.

Reports of avian genera infected with six cosmopolitan species are shown in Table 27.1. The remaining capillarid species have a narrower host range and a detailed list of these capillarid species and hosts is given in Table 27.2. Table 27.2 only includes reports based on identification of adult worms to species. Reports of adults identified to level of genus, for example, "*Capillaria* sp.," or based on detection of capillarid eggs in feces are not included.

ETIOLOGY

Capillarids are small hairlike nematodes that parasitize the gastrointestinal tract of all classes of vertebrates. Capillarids have an esophagus that is characteristic of nematodes in the superfamily Trichinelloidea. The esophagus has a short muscular anterior part and a long glandular posterior portion (stichosome) that is composed of cuboidal gland cells called stichocytes (Figure 27.1). Eggs of trichurids and capillarids are also distinct in that they have bipolar plugs and may be nearly clear to a deep golden color; the surface ranges from smooth to variably textured (Figure 27.2).

EPIZOOTIOLOGY

Detailed life history traits are primarily known for those species that infect domestic fowl. With few exceptions, for example, *Pterothominx philippinensis*, unembryonated eggs are passed in the feces and develop to the first larval stage (L1) in 9–14 days. For those species with a direct life cycle, eggs containing L1 larvae are infective to the next avian host. For capillarids with indirect life cycles, various species of earthworms serve as required intermediate hosts. One exception, however, is *P. philippinensis*, which uses fish as a required intermediate host. After eggs are ingested by an earthworm, they hatch in the gastrointestinal tract and larvae migrate through tissues of the earthworm until it is ingested by an appropriate avian host. Among some capillarid species, larvae are immediately infective after hatching in the earthworm, while in other species further development of the larvae must occur in the earthworm prior to ingestion by an avian host. If an earthworm is required in the life cycle, eggs must pass through the worm before they hatch (Moravec et al. 1987; McDougald 2003).

Interestingly, an extreme female sex bias has been observed in capillarid infections of birds and other vertebrates. Since most birds infected with capillarids have few adult worms, a bias that favors adult females will increase parasite reproductive potential. The mechanism by which this sex bias develops is unknown (Lewis 1968; Joy and Scott 1997; Poulin 1997; Martin et al. 2006).

The study of capillarids in wild birds is complicated by the lack of knowledge of life cycles and taxonomy of many species. Many studies are restricted to reports of capillarid eggs in fecal samples. Fecal surveys likely underestimate the true prevalence of capillarid infection because most free-ranging birds have very low-intensity infections and low fecal egg counts (Prestwood 1968). Ecological studies based on fecal egg detection are limited further because the capillarid species cannot be identified by egg morphology, coinfections with multiple capillarid species may be missed, and coinfecting capillarid species may use different life cycle strategies.

The effects of age, sex, and season on capillarid infection seem to vary greatly depending on the geographic region and host and parasite species. For example, greater numbers of adult Wild Turkeys (*Meleagris gallopavo*) and Rock Pigeons (*Columba livia*) are infected with *Capillaria* spp. (Prestwood 1968) while more young American Woodcocks (*Scolopax minor*) and Clapper Rails (*Rallus longirostris*) are infected with *Capillaria* spp. (Heard 1967; Pursglove 1969). Senlik et al. (2005) reported that adult pigeons and those sampled in the autumn were more likely to be infected with *B. obsignata* than fledglings or nestlings (Senlik et al. 2005). Seasonal trends were not associated with capillarid infections in chickens in Tanzania or infections of *P. caudinflata* in Willow Ptarmigan (*Lagopus lagopus*) from Norway (Permin et al. 1997; Schei et al. 2005), but were associated with infections of

Table 27.1. Genera of birds infected with six cosmopolitan capillarids species.*

Parasite species	Location in host	Avian order	Avian genera reported as host
Capillaria anatis	Cecum, sometimes in the small intestine	Anseriformes	*Aix, Anas, Anser, Aythya, Branta, Bucephala, Chen*, and *Lophodytes*
		Galliformes	*Alectoris, Meleagris, Perdix*, and *Phasianus*
		Passeriformes	*Attila, Corvus, Garrulus*, and *Pica*
Eucoleus annulatus	Esophagus and crop	Anseriformes	*Anas* and *Cairina*
		Galliformes	*Alectoris, Bonasa, Colinus, Gallus, Lyrurus, Meleagris, Numida, Perdix, Phasianus*, and *Tetrao*
Eucoleus contortus†	Pharynx, esophagus, and crop	Anseriformes	*Aix, Anas, Aythya, Bucephala, Clangula, Netta, Somateria*, and *Tadorna*
		Charadriiformes	*Actitis, Alle, Calidris, Capella, Charadrius, Chlidonias, Gallinago, Gelochelidon, Himantopus, Larus, Pavoncella, Philomachus, Pluvialis, Rissa, Tringa, Recurvirostra, Sterna, Thalasseus, Uria*, and *Vanellus*
		Ciconiiformes	*Botaurus, Cochlearius, Eudocimus, Plegadis*, and *Platalea*
		Falconiformes	*Accipiter, Buteo, Falco*, and *Haliaeetus*
		Galliformes	*Alectoris, Bonasa, Colinus, Crossoptilon, Lofortyx, Meleagris, Oreortyx, Perdix*, and *Phasianus*
		Gruiformes	*Crex, Fulica*, and *Gallinula*
		Passeriformes	*Corvus, Cyanocitta, Erithacus, Passerella, Phoenicurus*, and *Sturnus*
		Pelecaniformes	*Pelecanus* and *Phalacrocorax*
		Podicipediformes	*Podiceps* and *Tachybaptus*
Baruscapillaria obsignata	Small intestine and cecum	Anseriformes	*Aix, Branta, Chen*, and *Anser*
		Ciconiiformes	*Ardea*
		Columbiformes	*Columba* and *Zenaida*
		Galliformes	*Colinus, Coturnix, Gallus, Meleagris, Pavo, Perdix*, and *Zenaida*
		Piciformes	*Ramphastos*
		Psittaciformes	*Agapornis, Barnardius*, and *Melopsittacus*
Pterothominx bursata	Small intestine	Galliformes	*Gallus, Meleagris, Numida, Perdix, Phasianus*, and *Tetrao*
Pterothominx caudinflata	Small intestine	Anseriformes	*Anas, Anser, Branta, Cairina*, and *Chen*
		Columbiformes	*Columba* and *Streptopelia*
		Galliformes	*Alectoris, Bonasa, Callipepla, Coturnix, Chrysolophus, Gallus, Lagopus, Meleagris, Numida, Perdix, Phasianus*, and *Tetrao*
		Gruiformes	*Otis*
		Passeriformes	*Erithacus, Ixoreus, Sturnus, Passer*, and *Turdus*
		Tinamiformes	*Nothoprocta*

*References: Walton (1923), Gower (1939), Read (1949), Madsen (1951), Yamaguti (1961), Hodasi (1963), Wakelin (1967), Kellogg and Prestwood (1968b), Hair and Forrester (1970), Wehr (1971), Gibson (1972), Moravec (1982), Moravec et al. (1987), Barus and Sergejeva (1989), Barus and Sergejeva (1990c), Barus and Sergejeva (1990d), Ching (1990), Uchida et al. (1991), Borgsteede et al. (2003), Forrester and Spalding (2003), McDougald (2003).

†Some researchers (see Barus and Sergejeva 1989) consider *Eucoleus contortus* to be a complex of at least two species—*Eucoleus contortus* from the oral cavity and esophagus of aquatic birds in the orders Anseriformes, Charadriiformes, Ciconiiformes, Pelecaniformes, and Podicipediformes and *Eucoleus dispar* from the oral cavity and esophagus of terrestrial birds. Other researchers consider these two species to occur in both aquatic and terrestrial birds.

Table 27.2. Reports of capillarids in avian hosts excluding *Capillaria anatis, Eucoleus annulatus, Eucoleus contortus, Baruscapillaria obsignata, Pterothominx bursata,* and *Pterothominx caudinflata* (Table 27.1). Only reports that included identification of adult worms to species are included. Reports of capillarid eggs in the feces or unspeciated capillarid adults are not included. Unless otherwise specified, all reports are from free-ranging birds.

Avian host	Capillarid species	Location in host	Disease signs/lesions	Location	References
Struthioniformes					
Greater Rhea (*Rhea americana*)	*Capillaria parvumspinosa*	Not given	None reported	Europe	Yamaguti (1961)
Tinamiformes					
Spotted Nothura (*Nothura maculosa*)	*Eucoleus penidoi*	Crop	None reported	Argentina, Brazil	Freitas and Almeida (1935) and Pinto et al. (2006)
Gray Tinamou (*Tinamus tao*)	*Eucoleus crypturi*	Esophagus	None reported	Brazil	Yamaguti (1961)
Gaviiformes					
Common Loon (*Gavia immer*)	*Baruscapillaria mergi*	Small intestine	None reported	USA	Kinsella and Forrester (1999)
Red-throated Loon (*Gavia stellata*)	*Baruscapillaria mergi*	Large intestine	None reported	Unknown	Baruš and Sergejeva (1990c)
Podicipediformes					
Red-necked Grebe (*Podiceps grisegena*)	*Baruscapillaria ryjikovi*	Small intestine, cecum	None reported	USSR	Baruš and Sergejeva (1990c)
Eared Grebe (*Podiceps nigricollis*)	*Baruscapillaria ryjikovi*	Small intestine, cecum	None reported	Czechoslovakia	Baruš and Sergejeva (1990c)
Pied-billed Grebe (*Podilymbus podiceps*)	*Baruscapillaria ryjikovi* (=*Baruscapillaria podicipitis)*	Cloaca	None reported	USA	Baruš and Sergejeva (1990c) and Forrester and Spalding (2003)
Little grebe (*Tachybaptus ruficollis*)	*baruscapillaria ryjikovi*	Cloaca	None reported	Czechoslovakia, Japan, USSR	Baruš and Sergejeva (1990c) and Uchida et al. (1991)
Pelecaniformes					
Anhinga (*Anhinga anhinga*)	*Carcharhinus perezi*	Unknown	Unknown	Cuba	Yamaguti (1961)
Double-crested Cormorant (*Phalacrocorax auritus*)	*Ornithocapillaria carbonis*	Small intestine, ceca, and large intestine	None reported	USA	Threlfall (1982) and Forrester and Spalding (2003)
	Baruscapillaria spilculata	Large intestine	None reported	USA	Fedynich et al. (1997)

Neotropic Cormorant (*Phalacrocorax brasilianus*)	*Ornithocapillaria appendiculata*	Large intestine	None reported	Brazil, Mexico, USA	Freitas (1933b), Vicente et al. 1995, and Moravec et al. 2000
	Baruscapillaria spilculata	Large intestine	None reported	Brazil, USA	Freitas (1933b) and Fedynich et al. (1997)
Great Cormorant (*Phalacrocorax carbo*)	*Baruscapillaria rudolphii*	Intestine	None reported	Czech Republic	Moravec et al. (1994)
	Ornithocapillaria carbonis	Intestine	None reported	Russia, Poland, Czech Republic	Dubinin and Dubinina (1940), Frantova (2001), and Frantova (2002)
	Baruscapillaria jaenschi	Not given	None reported	Australia	Johnston and Mawson (1945)
Black-faced Cormorant (*Phalacrocorax fuscescens*)	*Baruscapillaria jaenschi*	Not given	None reported	Australia	Johnston and Mawson (1945)
Little Pied Cormorant (*Phalacrocorax melanoleucos*)	*Baruscapillaria jaenschi*	Not given	None reported	Australia	Johnston and Mawson (1945)
Pygmy Cormorant (*Phalacrocorax pygmaeus*)	*Ornithocapillaria phalacrocoraxi*	Bursa of Fabricius and cloaca	None reported	Tajikistan	Baruš and Sergejeva (1990b)
Little Black Cormorant (*Phalacrocorax sulcirostris*)	*Baruscapillaria jaenschi*	Not given	None reported	Australia	Johnston and Mawson (1945)
Australian Pelican (*Pelecanus conspicillatus*)	*Baruscapillaria jaenschi*	Not given	None reported	Australia	Johnston and Mawson (1945)
American White Pelican (*Pelecanus erythrorhynchos*)	*Baruscapillaria mergi*	Small and large intestine, cecum	None reported	USA	Kinsella et al. (2004)
Brown Pelican (*Pelecanus occidentalis*)	*Baruscapillaria mergi*	Intestine	None reported	USA	Courtney and Forrester (1974)

(*continues*)

Table 27.2. (*Continued*)

Avian host	Capillarid species	Location in host	Disease signs/lesions	Location	References
Ciconiiformes					
Great Egret (*Ardea alba*)	*Capillaria herodiae*	Small intestine	None reported	USA	Sepúlveda et al. (1999)
Great Blue Heron (*Ardea herodias*)	*Capillaria herodiae*	Small and large intestine	None reported	USA	Boyd (1966) and Forrester and Spalding (2003)
Little Blue Heron (*Egretta caerulea*)	*Baruscapillaria mergi*	Small intestine	None reported	USA	Sepúlveda et al. (1996)
Wood Stork (*Mycteria americana*)	*Capillaria avellari*	Esophagus	None reported	USA, Brazil	Yamaguti (1961) and Forrester and Spalding (2003)
	Baruscapillaria mergi	Small intestine	None reported	USA	Forrester and Spalding (2003)
Black-crowned Night-Heron (*Nycticorax nycticorax*)	*Capillaria brasiliana*	Unknown	Unknown	Brazil	Freitas (1933a) and Yamaguti (1961)
Roseate Spoonbill (*Platalea ajaja*)	*Baruscapillaria mergi*	Gizzard, small intestine, and large intestine	None reported	USA	Sepúlveda et al. (1994)
Anseriformes					
Wood Duck (*Aix sponsa*)	*Capillaria spinulosa*	Cecum	None reported	USA	Ogburn-Cahoon (1979) and Forrester and Spalding (2003)
	Pterothominx exilis	Intestine	None reported	USA, England (captive)	McDonald (1969) and Baruš and Sergejeva (1990d)
Eurasian Teal (*Anas crecca*)	*Baruscapillaria mergi*	Cecum	None reported	USSR	Baruš and Sergejeva (1990c)
Mottled Duck (*Anas fulvigula*)	*Capillaria spinulosa*	Cecum	None reported	USA	Forrester and Spalding (2003)
Mallard (*Anas platyrhynchos*)	*Capillaria nyrocinarum*	Intestine	None reported	Japan	Motohiro et al. (2000)
	Eucoleus perforans	Esophagus	None reported	Asia, Europe	Baruš and Sergejeva (1989)
Ring-necked Duck (*Aythya collaris*)	*Capillaria spinulosa*	Cecum	None reported	USA	Forrester and Spalding (2003)

Greater Scaup (*Aythya marila*)	*Capillaria nyrocinarum*	Intestine	None reported	Denmark	Yamaguti (1961)
Ferruginous Pochard (*Aythya nyroca*)	*Capillaria nyrocinarum*	Intestine	None reported	Denmark	Yamaguti (1961)
	Capillaria spinulosa	Cecum	None reported	Europe	Gower (1939)
Common Goldeneye (*Bucephala clangula*)	*Baruscapillaria mergi*	Intestine	None reported	Canada, Denmark	Yamaguti (1961), Mahoney and Threlfall (1978), and Baruš and Sergejeva (1990c)
	Capillaria nyrocinarum	Intestine	None reported	Denmark	Yamaguti (1961)
Long-tailed Duck (*Clangula hyemalis*)	*Baruscapillaria mergi*	Small and large intestine	None reported	Denmark	Gower (1939), Yamaguti (1961), and Baruš and Sergejeva (1990c)
	Capillaria nyrocinarum	Intestine	None reported	Denmark	Yamaguti (1961)
White-winged Scoter (*Melanitta fusca*)	*Capillaria nyrocinarum*	Intestine	None reported	Canada	U.S. National Parasite Collection, Accession No. 076891
White-winged Scoter (*Melanitta fusca*)	*Baruscapillaria mergi*	Small and large intestine	None reported	Denmark	Yamaguti (1961) and Baruš and Sergejeva (1990c)
	Capillaria nyrocinarum	Intestine	None reported	Denmark	Yamaguti (1961)
Black Scoter (*Melanitta nigra*)	*Capillaria nyrocinarum*	Intestine and cecum	None reported	Canada, Denmark	Yamaguti (1961) and U.S. National Parasite Collection Accession No. 076911
Surf Scoter (*Melanitta perspicillata*)	*Capillaria nyrocinarum*	Cecum	None reported	Canada	U.S. National Parasite Collection, Accession No. 076865
Common Merganser (*Mergus merganser*)	*Baruscapillaria mergi*	Small intestine	None reported	Denmark, USSR	Yamaguti (1961) and Baruš and Sergejeva (1990c)

(*continues*)

Table 27.2. (*Continued*)

Avian host	Capillarid species	Location in host	Disease signs/lesions	Location	References
Red-breasted Merganser (*Mergus serrator*)	*Baruscapillaria mergi*	Small intestine	None reported	Denmark	Yamaguti (1961)
Common Eider (*Somateria mollissima*)	*Capillaria nyrocinarum*	Intestine	None reported	Netherlands, Denmark, Canada	Yamaguti (1961), Bishop and Threlfall (1974), and Borgsteede et al. (2005)
	Baruscapillaria mergi	Small intestine	None reported	Denmark	Yamaguti (1961)
King Eider (*Somateria spectabilis*)	*Capillaria nyrocinarum*	Intestine	None reported	Denmark	Yamaguti (1961)
Black Swan (*Cygnus atratus*)	*Capillaria ellisi*	Intestine	None reported	Australia	Johnston and Mawson (1945)
Tundra Swan (*Cygnus columbianus*)	*Capillaria gigantoteca* (some authors consider this a synonum of *Baruscapillaria obsignata*)	Small intestine	None reported	Russia	Yamaguti (1961)
Black-necked Swan (*Cygnus melancoryphus*)	*Capillaria droummondi*	Unknown	Unknown	Brazil	Yamaguti (1961)
Mute Swan (*Cygnus olor*)	*Capillaria pudendotecta*	Intestine	None reported	Russia	Yamaguti (1961)
Falconiformes					
Northern Goshawk (*Accipiter gentilis*)	*Eucoleus dispar* (sometimes as *Trichosomum contorta*)	Esophagus	None reported	Germany, Poland, Spain	Okulewicz (1988), Krone (2000), and Ferrer et al. (2004)
	Capillaria tenuissima	Small intestine, sometimes gizzard	None reported	Germany	Krone (2000)
	Baruscapillaria falconis	Small intestine	None reported	Czech Republic	Frantova (2002)
Eurasian Sparrow Hawk (*Accipiter nisus*)	*Eucoleus dispar*	Esophagus	None reported	Spain	Ferrer et al. (2004) and Sanmartin et al. (2004)
	Capillaria tenuissima	Small intestine, sometimes gizzard	None reported	Spain, Germany, Czech Republic	Krone (2000), Frantova (2002), and Sanmartin et al. (2004)

	Baruscapillaria falconis (as *Trichosomum contortum*)	Small intestine	None reported	Europe	Read (1949) and Yamaguti (1961)
Crested Goshawk (*Accipiter trivirgatus*)	*Baruscapillaria falconis*	Small intestine	None reported	Taiwan	Su and Fei (2004)
Eurasian Buzzard (*Buteo buteo*)	*Capillaria tenuissima*	Small intestine, sometimes gizzard	None reported	Netherlands, Spain, Poland, Germany	Okulewicz (1988), Illescas et al. (1993), Krone (2000), Borgsteede et al. (2003), and Sanmartin et al. (2004)
	Eucoleus dispar	Esophagus	None reported	Netherlands, Germany, Czech Republic, Spain	Lierz et al. (2002), Borgsteede et al. (2003), and Ferrer et al. (2004)
	Baruscapillaria falconis	Small intestine	None reported	Czech Republic	Frantova (2002)
Red-tailed Hawk (*Buteo jamaicensis*)	*Eucoleus dispar* (reported as *Capillaria contorta*)	Esophagus	None reported	USA	Forrester and Spalding (2003)
	Baruscapillaria falconis	Small intestine	None reported	USA	Read (1949) and Forrester and Spalding (2003)
Red-shouldered Hawk (*Buteo lineatus*)	*Eucoleus dispar* (reported as *Capillaria contorta*)	Esophagus	None reported	USA	Forrester and Spalding (2003)
	Baruscapillaria falconis	Small intestine	None reported	USA	Forrester and Spalding (2003)
Short-toed Eagle (*Circaetus gallicus*)	*Eucoleus dispar*	Esophagus	None reported	Spain	Ferrer et al. (2004)
Western Marsh-Harrier (*Circus aeruginosus*)	*Eucoleus dispar*	Esophagus	None reported	Germany, Spain	Lierz et al. (2002) and Ferrer et al. (2004)
Montagu's Harrier (*Circus pygargus*)	*Eucoleus dispar*	Esophagus	None reported	Spain	Sanmartin et al. (2004)
	Capillaria tenuissima	Small intestine, sometimes gizzard	None reported	Spain	Sanmartin et al. (2004)

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Table 27.2. (*Continued*)

Avian host	Capillarid species	Location in host	Disease signs/lesions	Location	References
Peregrine Falcon (*Falco peregrinus*)	*Eucoleus dispar*	Esophagus	None reported	Germany, Spain	Lierz et al. (2002) and Ferrer et al. (2004)
American Kestrel (*Falco sparverius*)	*Baruscapillaria falconis*	Small intestine	None reported	USA	Forrester and Spalding (2003)
	Ornithocapillaria cylindrica	Intestine	None reported	Cuba	Baruš and Sergejeva (1990c)
Eurasian Hobby (*Falco subbuteo*)	*Eucoleus dispar*	Esophagus	None reported	Spain	Ferrer et al. (2004)
Eurasian Kestrel (*Falco tinnunculus*)	*Eucoleus dispar* (sometimes as *Eucoleus contortus* or *Eucoleus suppereri*)	Esophagus	None reported	Spain, Germany	Lierz et al. (2002) and Sanmartin et al. (2004)
	Capillaria tenuissima	Small intestine, sometimes gizzard	None reported	Spain	Sanmartin et al. (2004)
White-tailed Eagle (*Haliaeetus albicilla*)	*Eucoleus dispar*	Esophagus	None reported	Germany, Finland	Krone et al. (2003) and Krone et al. (2006)
	Capillaria tenuissima	Small intestine	None reported	Germany	Krone et al. (2003)
Bald Eagle (*Haliaeetus leucocephalus*)	*Eucoleus dispar* (reported as *Capillaria contorta*)	Esophagus	None reported	USA	Kinsella et al. (1998)
	Baruscapillaria falconis	Small intestine	None reported	USA	Forrester and Spalding (2003)
Red Kite (*Milvus milvus*)	*Eucoleus dispar*	Esophagus	None reported	Spain	Sanmartin et al. (2004)
Osprey (*Pandion haliaetus*)	*Baruscapillaria falconis*	Small intestine	None reported	USA	Kinsella et al. (1996)
Chimango Caracara (*Milvago chimango*)	*Capillaria tenuissima*	Small intestine	None reported	Chile	Martin et al. (2006)

Galliformes					
Vulturine Guinea Fowl (*Acryllium vulturinum*)	*Eucoleus perforans*	Esophagus	Weakness, anorexia, vomiting, thickening of mucosa, extensive damage to epithelium	USA (captive)	De Rosa and Shivaprasad (1999)
Golden Pheasant (*Chrysolophus pictus*)	*Capillaria phasianina*	Cecum	None reported	Denmark (captive)	Madsen (1951)
domestic chicken (*Gallus gallus domestica*)	*Baruscapillaria montevidensis*	Cecum	None reported	Uruguay	Calzada (1937)
Wild Turkey *(Meleagris gallopavo)* and domesticated turkey	*Capillaria phasianina*	Cecum	None reported	USA	Schorr (1988)
	Thomix tridens	Small intestine	None reported	USA	Davidson et al. (1975)
	Eucoleus perforans (=Capillaria or *Eucoleus combologiodes)*	Esophagus	None reported	Worldwide	Baruš and Sergejeva (1989)
	Pterothominx meleagridis	Small intestine (submucosa)	None reported	Worldwide	Baruš and Sergejeva (1990d)
Helmeted Guinea Fowl (*Numida meleagris*)	*Eucoleus perforans*	Esophagus	Severe esophagitis	Worldwide	Baruš and Sergejeva (1989) and Menezes et al. (2001)
Gray Partridge (*Perdix perdix*)	*Capillaria phasianina*	Cecum	Mild to severe necrosis, mortality	Denmark, England	Clapham (1949) and Madsen (1951)
	Eucoleus perforans	Esophagus	None reported	Worldwide	Baruš and Sergejeva (1989)
	Pterothominx blomei (some consider a synonym of *Pterothominx caudinflata*)	Small intestine	None reported	England, Switzerland	Baruš and Sergejeva (1990d)

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Table 27.2. (*Continued*)

Avian host	Capillarid species	Location in host	Disease signs/lesions	Location	References
Ring-necked Pheasant (*Phasianus colchicus*)	*Eucoleus perforans*	Crop and esophagus	Lethargy, emaciation, diarrhea, thickened, small nodules, congestion, and petechial hemorrhage of the mucosa	Brazil (captive, domesticated)	Pinto et al. (2004) and Gassal and Schmäschke (2006)
	Echinocoleus cyanopicae (has only been reported in *Cyanopica cyaneus*, this may be a misidentification)	Cecum	Necrosis of epithelium and glandular crypts, some inflammation in the mucosa	Romania (captive, domesticated)	Iulia and Pavlovic (2003)
	Capillaria phasianiana	Small intestine and cecum	None reported (low burdens) to necrosis, fibrosis, sloughing of the epithelium (heavy burdens)	Worldwide Poland, Brazil, Romania, Denmark, Hungary (captive, domesticated)	Madsen (1951), Kellogg and Prestwood (1968a), Iulia and Pavlovic (2003), Pinto et al. (2004), Tampieri et al. (2005), and Gassal and Schmäschke (2006)
	Capillaria uropapillata	Crop and esophagus	None reported	Brazil (captive, domesticated)	Pinto et al. (2004)
	Pterothominx meleagridis	Small intestine (submucosa)	None reported	Worldwide (captive, domesticated)	Baruš and Sergejeva (1990d)
	Pterothominx blomei (some consider a synonym of *Pterothominx caudinflata*)	Small intestine	None reported	England, Switzerland (captive, domesticated)	Baruš and Sergejeva (1990d)

Black Grouse (*Tetrao tetrix*)	*Pterothominx alpina*	Duodenum near pylorus	None reported	Austria, Germany	Baruš and Sergejeva (1990d) and Fischbacher (2007)
Eurasian Capercaillie (*Tetrao urogallus*)	*Pterothominx alpina*	Duodenum near pylorus	None reported	Austria, Germany	Baruš and Sergejeva (1990d) and Fischbacher (2007)
Gruiformes					
Black-crowned-Crane (*Balearica pavonina*)	*Eucoleus obtusiuscula*	Gizzard	None reported	Africa	Baruš and Sergejeva (1989)
American Coot (*Fulica americana*)	*Capillaria fulicae*	Cecum	None reported	USA	Kinsella (1973)
Common Moorhen (*Gallinula chloropus*)	*Capillaria fulicae*	Cecum	None reported	USA	Forrester and Spalding (2003)
Whooping Crane (*Grus americana*)	*Eucoleus obtusiuscula*	Gizzard	None reported	USA (captive)	Spalding et al. (1996)
	Capillaria fulicae	Small intestine, cecum, and large intestine	None reported	USA (captive)	Forrester and Spalding (2003)
Sandhill Crane (*Grus canadensis*)	*Eucoleus obtusiuscula*	Gizzard	None reported	USA	Spalding et al. (1996)
Common Crane (*Grus grus*)	*Eucoleus obtusiuscula*	Gizzard	None reported	Europe	Yamaguti (1961)
Purple Gallinule (*Porphyrio martinica*)	*Capillaria fulicae*	Cecum	None reported	USA	Forrester and Spalding (2003)
Charadriiformes					
Common Sandpiper (*Actitis hypoleucos*)	*Pterothominx totani*	Intestine	None reported	Europe, USSR	Yamaguti (1961) and Baruš and Sergejeva (1990d)
Spotted Sandpiper (*Actitis macularius)*	*Pterothominx totani*	Intestine	None reported	Canada	Gibson (1972)
Red Knot (*Calidris canutus*)	*Capillaria cecumitis*	Large intestine	None reported	USA	Forrester and Spalding (2003)
Common Ringed Plover (*Charadrius hiaticula*)	*Pterothominx totani*	Intestine	None reported	USSR	Baruš and Sergejeva (1990d)

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Table 27.2. (*Continued*)

Avian host	Capillarid species	Location in host	Disease signs/lesions	Location	References
Killdeer (*Charadrius vociferus*)	*Eucoleus obtusiuscula (=Capillaria vanelli)*	Gizzard	None reported	USA	Forrester and Spalding (2003)
Whiskered Tern (*Chlidonias hybrida*)	*Baruscapillaria jaenschi*	Not given	None reported	Australia	Johnston and Mawson (1945)
Dunlin (*Calidris alpina*)	*Capillaria cecumitis*	Large intestine	None reported	USA	Ching (1990)
Willet (*Tringa semipalmata inornata*)	*Capillaria cecumitis*	Large intestine	None reported	USA	Ching (1990)
Common Snipe (*Gallinago gallinago*)	*Baruscapillaria belopolskaiae*	Small intestine	None reported	Europe	Baruš and Sergejeva (1990c)
Silver Gull (*Larus novaehollandiae*)	*Baruscapillaria jaenschi*	Not given	None reported	Australia	Johnston and Mawson (1945)
American Avocet (*Recurvirostra americana*)	*Capillaria recurvirostrae*	Small intestine	None reported	USA	Ahern and Schmidt (1976) and Garcia and Canaris (1987)
Northern Lapwing (*Vanellus vanellus*)	*Eucoleus obtusiuscula*	Gizzard	None reported	Europe, Tunisia	Yamaguti (1961), Bernard (1989), and Baruš and Sergejeva (1989)
Terek Sandpiper (*Xenus cinereus*)	*Pterothominx totani*	Intestine	None reported	USSR	Baruš and Sergejeva (1990d)
Columbiformes					
Spot-winged Wood-Quail (*Odontophorus capueira*)	*Capillaria vasi*	Intestine	None reported	Brazil	Madsen (1951)
Psittaciformes					
Caatinga Parakeet (*Aratinga cactorum*)	*Capillaria plagiaticia*	Not given	None reported	Brazil (captive)	Freitas et al. (1959)
Port Lincoln Parrot (*Barnardius zonarius*)	*Pterothominx moraveci*	Small intestine	None reported	Captive (Germany/Czech Republic)	Baruš et al. (2005)
Kea (*Nestor notabilis*)	*Capillaria plagiaticia*	Not given	None reported	New Zealand (captive)	Wakelin (1967)

Strigiformes					
Northern Saw-whet Owl (*Aegolius acadicus*)	*Baruscapillaria falconis*	Small intestine	None reported	USA	Read (1949)
Northern Long-eared Owl (*Asio otus*)	*Capillaria tenuissima*	Small intestine, sometimes gizzard	None reported	Austria, Germany	Kutzer et al. (1982) and Krone (2000)
	Baruscapillaria falconis	Small intestine	None reported	Spain, USA	Read (1949) and Illescas et al. (1993)
Little Owl (*Athene noctua*)	*Capillaria tenuissima*	Small intestine, sometimes gizzard	None reported	Austria, Germany	Kutzer et al. (1982) and Krone (2000)
Eurasian Eagle-Owl (*Bubo bubo*)	*Baruscapillaria falconis*	Small intestine	None reported	Spain	Illescas et al. (1993)
Great Horned Owl (*Bubo virginianus*)	*Baruscapillaria falconis*	Small intestine	None reported	Canada, USA	Read (1949), Ramalingam and Samuel (1978), and Kinsella et al. (2001)
	Capillaria tenuissima	Small intestine	None reported	USA	Kinsella et al. (2001)
Tawny Owl (*Strix aluco*)	*Eucoleus dispar*	Esophagus	None reported	Poland	Okulewicz (1988)
	Capillaria tenuissima	Small intestine, sometimes gizzard	None reported	Austria, Germany	Kutzer et al. (1982) and Krone (2000)
Spotted Owl (*Strix occidentalis*)	*Baruscapillaria falconis*	Small intestine	None reported	USA	Hoberg et al. (1989)
Ural Owl (*Strix uralensis*)	*Capillaria tenuissima*	Small intestine	None reported	Japan	Uchida et al. (1991)
	Capillaria tenuissima	Small intestine	None reported	Austria	Kutzer et al. (1982)
Barred Owl (*Strix varia*)	*Eucoleus dispar*	Esophagus	None reported	USA	Kinsella et al. (2001)
	Baruscapillaria falconis	Small intestine	None reported	USA	Kinsella et al. (2001)
	Capillaria tenuissima	Small intestine	None reported	USA	Kinsella et al. (2001),
Barn Owl (*Tyto alba*)	*Capillaria tenuissima*	Small intestine, sometimes gizzard	None reported	France, Germany	Thebault (1988), and Krone (2000)
Australian Masked-Owl (*Tyto novaehollandiae*)	Described as *Capillaria strigis* (homonym so renamed *Capillaria newzealandica*)	Not given	None reported	New Zealand	Johnston and Mawson (1944) and Yamaguti (1961)

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Table 27.2. (*Continued*)

Avian host	Capillarid species	Location in host	Disease signs/lesions	Location	References
Caprimulgiformes					
Large-tailed Nightjar (*Caprimulgus macrurus*)	*Thomix tridens*	Small intestine	None reported	Philippines	U.S. National Parasite Collection
Apodiformes					
Common Swift (*Apus apus*)	*Tridentocapillaria hirundinis*	Intestine	None reported	Europe	Yamaguti (1961)
Piciformes					
Maroon Woodpecker (*Blythipicus rubiginosus*)	*Thomix tridens*	Small intestine	None reported	Borneo	Baruš and Sergejeva (1990a)
Northern Flicker (*Colaptes auratus*)	*Capillaria longistriata*	Small intestine	None reported	USA	Walton (1923)
	Thomix tridens	Small intestine	None reported	USA	Bolette (1998)
	Capillaria venusta	Small intestine	None reported	Cuba	Baruš (1971)
Great Spotted Woodpecker (*Dendrocopos major*)	*Ornithocapillaria picorum*	Small intestine	None reported	USA	Walton (1923) and Yamaguti (1961)
Pileated Woodpecker (*Dryocopus pileatus*)	*Capillaria longistriata*	Small intestine	None reported	USA	Forrester and Spalding (2003)
Red-bellied Woodpecker (*Melanerpes carolinus*)	*Thomix tridens*	Small intestine	None reported	USA	Foster et al. (2002)
Northern Flicker (*Colaptes auratus*)	*Ornithocapillaria picorum*	Small intestine	None reported	Mexico	Leidy (1856)
Golden-olive Woodpecker (*Piculus rubiginosus*) (=*Colaptes maximus*)	*Ornithocapillaria picorum*	Small intestine	None reported	Brazil	Walton (1923) and Yamaguti (1961)
Gray-faced Woodpecker (*Picus canus*)	*Ornithocapillaria picorum*	Small intestine	None reported	Europe	Walton (1923) and Yamaguti (1961)
	Tridentocapillaria eurycerca	Small intestine	None reported	USSR	Baruš and Sergejeva (1990a)
Green Woodpecker (*Picus viridis*)	*Ornithocapillaria picorum*	Small intestine	None reported	Europe	Walton (1923) and Yamaguti (1961)

Black-necked Aracari (*Pteroglossus aracari*)	*Capillaria venusta*	Not given	None reported	Brazil	Yamaguti (1961)
Toco Toucan (*Ramphastos toco*)	*Capillaria venusta*	Not given	None reported	Brazil	Pinto et al. (1996)
Channel-billed Toucan (*Ramphastos vitellinus*)	*Capillaria venusta*	Not given	None reported	Brazil	Pinto et al. (1996)
Passeriformes					
Sedge Warbler (*Acrocephalus schoenobaenus*)	*Eucoleus dispar*	Esophagus	None reported	Europe	Baruš and Sergejeva (1989)
Red-winged Blackbird (*Agelaius phoeniceus*)	*Thomix tridens*	Small intestine	None reported	USA	Read (1949)
	Ornithocapillaria ovopunctata	Intestine	None reported	USA	Cooper and Crites (1974b)
Eurasian Skylark (*Alauda arvensis*)	*Pterothominx exilis*	Intestine	None reported	Asia, Europe	Baruš and Sergejeva (1990d)
	Pterothominx longifilla	Small intestine	None reported	Czechoslovakia	Baruš and Sergejeva (1990d)
Olive-backed Pipit (*Anthus hodgsoni*)	*Baruscapillaria emberizae*	Small intestine	None reported	Japan	Uchida et al. (1991)
Meadow Pipit (*Anthus pratensis*)	*Ornithocapillaria ovopunctata* (reported as *Capillaria ornate* or *Baruscapillaria inflexa*)	Intestine	None reported	Europe	Yamaguti (1961)
Tree Pipit (*Anthus trivialis*)	*Pterothominx longifilla*	Intestine	None reported	France	Yamaguti (1961)
European Greenfinch (*Carduelis chloris*)	*Pterothominx exilis*	Intestine	None reported	Asia, Europe	Baruš and Sergejeva (1990d)
Gray-cheeked Thrush (*Catharus minimus*)	*Ornithocapillaria ovopunctata*	Small intestine	None reported	Cuba	Baruš and Sergejeva (1990c)
Brown Dipper (*Cinclus pallasii*)	*Baruscapillaria cincli*	Small intestine	None reported	Japan	Uchida et al. (1991)

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Table 27.2. (*Continued*)

Avian host	Capillarid species	Location in host	Disease signs/lesions	Location	References
White-capped Redstart (*Chaimarrornis leucocephalus*)	*Ornithocapillaria ovopunctata*	Small intestine	None reported	Nepal	Baruš and Daniel (1976)
Black-faced Cuckoo-shrike (*Coracina novaehollandiae*)	*Capillaria graucalina*	Not given	None reported	Australia	Johnston and Mawson (1941)
American Crow (*Corvus brachyrhynchos*)	*Baruscapillaria resecta (=Baruscapillaria corvorum)*	Small intestine	None reported	Canada	Andrews and Threlfall (1975)
Eurasian Nutcracker (*Nucifraga caryocatactes*)	*Baruscapillaria resecta (=Baruscapillaria corvorum)*	Intestine	None reported	Europe	Yamaguti (1961) and Baruš and Sergejeva (1990c)
Common Raven (*Corvus corax)*	*Baruscapillaria resecta (=Capillaria corvorum)*	Intestine	None reported	Tunisia	Bernard (1989)
Carrion Crow (*Corvus corone*)	*Eucoleus dispar*	Esophagus	None reported	Europe	Baruš and Sergejeva (1989)
	Baruscapillaria resecta (=Baruscapillaria corvorum)	Intestine	None reported	Europe	Baruš and Sergejeva (1990c)
Rook (*Corvus frugilegus*)	*Eucoleus dispar*	Esophagus	None reported	Not given	Baruš and Sergejeva (1989)
	Baruscapillaria resecta (=Baruscapillaria corvorum)	Intestine	None reported	Europe	Baruš and Sergejeva (1990c) and Frantova (2002)
	Pterothominx exilis	Small intestine	None reported	Asia, Europe	Baruš and Sergejeva (1990d)
Eurasian Jackdaw (*Corvus monedula*)	*Baruscapillaria resecta (=Baruscapillaria corvorum)*	Intestine	None reported	Europe	Yamaguti (1961), Fronska-Popiel et al. (2001), and Frantova (2002)

Azure-winged Magpie (*Cyanopica cyanus*)	*Echinocoleus cyanopicae*	Cecum	None reported	Spain	Lopez-Neyra (1947) and Baruš and Sergejeva (1989)
Yellow-throated Warbler (*Dendroica dominica*)	*Thomix tridens*	Small intestine	None reported	Cuba	Baruš and Sergejeva (1990a)
Blackburnian Warbler (*Dendroica fusca*)	*Thomix tridens*	Small intestine	None reported	Cuba	Baruš and Sergejeva (1990a)
Yellowhammer (*Emberiza citrinella*)	*Baruscapillaria emberizae*	Small intestine	None reported	New Zealand	Johnston and Mawson (1944)
Reed Bunting (*Emberiza schoeniclus*)	*Pterothominx exilis*	Small intestine	None reported	Asia, Europe	Baruš and Sergejeva (1990d)
Yellow Bunting (*Emberiza sulphurata*)	*Baruscapillaria emberizae*	Small intestine	None reported	Japan	Uchida et al. (1991)
Gray Bunting (*Emberiza variabilis*)	*Baruscapillaria emberizae*	Small intestine	None reported	Japan	Uchida et al. (1991)
Eurasian Jay (*Garrulus glandarius*)	*Capillaria avicola*	Unknown	Unknown	Russia	Yamaguti (1961)
	Baruscapillaria resecta (=Baruscapillaria corvorum, sometimes reported as *Capillaria anatis*)	Intestine	None reported	Japan	Uchida et al. (1991)
	Pterothominx exilis	Small intestine	None reported	Asia, Europe	Baruš and Sergejeva (1990d)
Magpie-lark (*Grallina cyanoleuca*)	*Baruscapillaria grallinae*	Not given	None reported	Australia	Johnston and Mawson (1945)
Australasian Magpie (*Gymnorhina tibicen*)	*Capillaria gymnorhinae*	Not given	None reported	Australia	Johnston and Mawson (1947)
Barn Swallow (*Hirundo rustica*)	*Tridentocapillaria hirundinis*	Intestine	None reported	Europe	Yamaguti (1961)
Varied Thrush (*Ixoreus naevius*)	*Ornithocapillaria quiscali*	Intestine	None reported	Canada	Ching (1993)

(*continues*)

Table 27.2. (*Continued*)

Avian host	Capillarid species	Location in host	Disease signs/lesions	Location	References
Long-tailed Shrike (*Lanius schach*)	*Thomix tridens*	Small intestine	None reported	Russia, Philippines	Hromada et al. (2000) and U.S. National Parasite Collection
Siberian Rubythroat (*Luscinia calliope*)	*Baruscapillaria calliopsis*	Small intestine	None reported	Japan	Uchida et al. (1991)
Common Nightingale (*Luscinia megarhynchos*)	*Thomix tridens*	Small intestine	None reported	France, Poland	Yamaguti (1961) and Okulewicz (1982)
Black-and-white Warbler (*Mniotilta varia*)	*Thomix tridens*	Small intestine	None reported	Cuba	Baruš and Sergejeva (1990a)
Brown-headed Cowbird (*Molothrus ater*)	*Thomix tridens*	Small intestine	None reported	USA	Cooper et al. (1973)
Blue Rock-Thrush (*Monticola solitarius*)	*Ornithocapillaria ovopunctata* (reported *Baruscapillaria inflexa*)	Intestine	None reported	Europe	Yamaguti (1961)
Great Tit (*Parus major*)	*Thomix tridens*	Small intestine	None reported	Poland	Okulewicz (1991)
Green-backed Tit (*Parus monticolus*)	*Tridentocapillaria parusi*	Small intestine	None reported	Taiwan	Baruš and Sergejeva (1990a)
Spanish Sparrow (*Passer hispaniolensis)*	*Capillaria rigidula*	Intestine	None reported	Tunisia	Bernard (1989)
Fox Sparrow (*Passerella iliaca*)	*Capillaria freitasi*	Small intestine	None reported	USA	Read (1949)
Eurasian Magpie (*Pica pica pica*)	*Baruscapillaria corvorum* (=*Capillaria resectum*, sometimes reported as *Capillaria anatis*)	Intestine	None reported	USA, Europe	Yamaguti (1961) and Todd and Worley (1967)
	Eucoleus dispar	Esophagus	None reported	Not given	Baruš and Sergejeva (1989)
Black-billed Magpie (*Pica hudsonia*)	*Baruscapillaria corvorum* (reported as *Capillaria anatis)*	Small intestine	None reported	USA	Todd et al. (1967)

Eastern Towhee (*Pipilo erythrophthalmus*)	*Thomix tridens*	Small intestine	None reported	Canada	Hodasi (1963)
Scarlet Tanager (*Piranga olivacea*)	*Thomix tridens* (reported as *Capillaria pirangae*)	Small intestine	None reported	USA	Durbin (1952)
Summer Tanager (*Piranga rubra*)	*Thomix tridens*	Small intestine	None reported	Cuba	Baruš and Sergejeva (1990a)
White-browed Babbler (*Pomatostomus superciliosus*)	*Capillaria pomatostomi*	Not given	None reported	Australia	Johnston and Mawson (1945)
Alpine Accentor (*Prunella collaris*)	*Ornithocapillaria ovopunctata*	Small intestine	None reported	Nepal	Baruš and Daniel (1976)
	Capillaria rigidula	Large Intestine	None reported	Nepal	Baruš and Daniel (1976)
Dunnock (*Prunella modularis*)	*Capillaria rigidula*	Intestine	None reported	France	Yamaguti (1961)
	Pterothominx longifilla	Small intestine	None reported	Czechoslovakia	Baruš and Sergejeva (1990d) and Frantova (2002)
Yellow-billed Chough (*Pyrrhocorax graculus*)	*Pterothominx exilis*	Small intestine	None reported	Asia, Europe	Baruš and Sergejeva (1990d)
Common Grackle (*Quiscalus quiscula*)	*Ornithocapillaria ovopunctata*	Small intestine	None reported	USA, Canada	Hodasi (1963), Cooper and Crites (1974b), and Badley and Dronen (1979)
	Ornithocapillaria quiscali	Small intestine	None reported	USA	Read (1949)
	Pterothominx exilis	Intestine	None reported	USA	Cooper and Crites (1974b)
Eastern Bluebird (*Sialia sialis*)	*Pterothominx exilis*	Intestine	None reported	England (captive)	Baruš and Sergejeva (1990d)
Eurasian Nuthatch (*Sitta europaea*)	*Tridentocapillaria parusi*	Small intestine	None reported	Taiwan	Baruš and Sergejeva (1990a)
	Pterothominx longifilla	Small intestine	None reported	Asia, Europe	Baruš and Sergejeva (1990d)

(*continues*)

Table 27.2. (*Continued*)

Avian host	Capillarid species	Location in host	Disease signs/lesions	Location	References
Chestnut-cheeked Starling (*Sturnia philippensis*)	*Baruscapillaria emberizae*	Small intestine	None reported	Japan	Uchida et al. (1991)
Rosy Starling (*Pastor roseus*)	*Ornithocapillaria ovopunctata*	Small intestine	None reported	Tadzhikistan	Baruš and Sergejeva (1990b)
European Starling (*Sturnus vulgaris*)	*Pterothominx exilis*	Intestine	None reported	United Kingdom, Poland, Europe	Yamaguti (1961), Hair and Forrester (1970), and Baruš and Sergejeva (1990d)
	Ornithocapillaria ovopunctata	Intestine	None reported	Worldwide	Yamaguti (1961) and Baruš and Sergejeva (1990b)
	Capillaria similis	Unknown	Unknown	United Kingdom, Russia	Hair and Forrester (1970)
	Eucoleus dispar	Esophagus	None reported	Europe	Baruš and Sergejeva (1989)
Spotless Starling (*Sturnus unicolor)*	*Thomix tridens*	Small intestine	None reported	Tunisia	Bernard (1989)
Redwing (*Turdus iliacus*)	*Pterothominx exilis*	Intestine	None reported	Asia, Europe	Baruš and Sergejeva (1990d)
Eurasian Blackbird (*Turdus merula*)	*Pterothominx exilis*	Intestine	None reported	Europe	Yamaguti (1961), Baruš and Sergejeva (1990d), and Frantova (2002)
	Ornithocapillaria ovopunctata	Intestine	None reported	Asia, Europe, and North Africa	Yamaguti (1961) and Frantova (2002)
American Robin (*Turdus migratorius*)	*Pterothominx exilis*	Intestine	None reported	Canada	Rayner (1932) and Cooper and Crites (1974a)
	Ornithocapillaria ovopunctata	Intestine	None reported	USA	Cooper and Crites (1974a)

Song Thrush (*Turdus philomelos*)	*Baruscapillaria ovopunctata* (also reported as *Baruscapillaria inflexa*)	Intestine	None reported	Europe	Yamaguti (1961) and Kopociǹska et al. (2004)
	Pterothominx exilis	Intestine	None reported	Poland, Czech Republic	Frantova (2002) and Kopociǹska et al. (2004)
	Ornithocapillaria quiscali	Intestine	None reported	Canada	Ching (1993)
Fieldfare (*Turdus pilaris*)	*Capillaria similis*	Unknown	Unknown	Europe	Yamaguti (1961)
Dark-throated Thrush (*Turdus ruficollis*)	*Pterothominx exilis*	Intestine	None reported	Asia, Europe	Baruš and Sergejeva (1990d)
Mistle Thrush (*Turdus viscivorus*)	*Baruscapillaria ovopunctata*	Intestine	None reported	Europe	Yamaguti (1961)
	Pterothominx exilis	Intestine	None reported	Asia, Europe	Baruš and Sergejeva (1990d)
White-eyed Vireo (*Vireo griseus*)	*Thomix tridens*	Small intestine	None reported	Cuba	Baruš and Sergejeva (1990a)
Hooded Warbler (*Wilsonia citrina*)	*Thomix tridens*	Small intestine	None reported	Cuba	Baruš and Sergejeva (1990a)

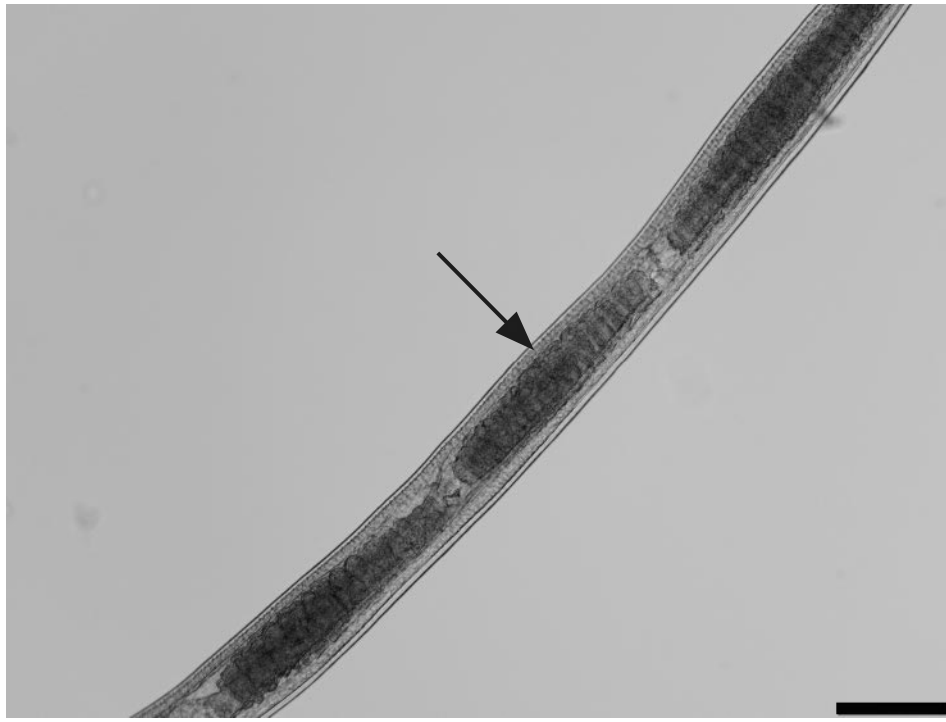


Figure 27.1. *Eucoleus contortus* stichosome (glandular esophagus). The middle of one of the three individual stichocytes depicted in the photomicrograph is marked with arrow. Scale bar = 100 μm.

Eucoleus contortus in Wood Ducks (*Aix sponsa*) from Louisiana (Ogburn-Cahoon 1979).

Details about the natural history of a limited number of the more thoroughly studied capillarids are provided in the following sections. These species primarily infect species of Anseriformes, Columbiformes, or Galliformes or are zoonotic and have been extensively studied because of their importance to avian or human health.

Pterothominx caudinflata (=*Capillaria caudinflata*, =*Aonchotheca caudinflata*)

This species is a cosmopolitan parasite of birds in the orders Anseriformes, Columbiformes, Galliformes, Gruiformes, Passeriformes, and Tinamiformes (Table 27.1). Eggs passed in the feces of hosts must develop in the environment for 11–12 days prior to ingestion and further development in earthworms. Required intermediate hosts include *Allolobophora caliginosa*, *Allolobophora parva*, *Aporrectodea rosea*, *Dendrobaena octaedra*, *Dendrobaena adaiensis* (synonym *Dendrobaena smidti*), *Dendrodrilus rubidus*, *Eisenia foetida*, *Helodrilus caliginosus*, and *Lumbricus terrestris* (Moravec et al. 1987; McDougald 2003).

Pterothominx bursata

This species infects many species of Galliformes (Table 27.1) and adults are found in the mucosa of the small intestine. Development of larvae within the egg takes 8–15 days after eggs are passed in feces. Eggs hatch once they are ingested by a required earthworm intermediate host. Larvae penetrate into the earthworm body cavity to molt and develop further. Larvae are infective to the definitive host approximately 22–25 days after infecting the earthworms. Ingested larvae develop to adults in 20–26 days (Moravec et al. 1987; McDougald 2003).

Eucoleus contortus (=*Thominx spirale*, =*Eucoleus spiralis*, =*Capillaria venteli*)

This is a cosmopolitan species that infects the oral cavity, esophagus, and crop of at least eight orders of birds (Table 27.1). Adults are found in both the mucosa and submucosa (Anderson 2000). The life cycle is direct and eggs passed in feces develop for 24–40 days before they are infective. Under ideal conditions of suitable moisture and no sunlight, embryonated eggs can remain viable for up to 15 months. The prepatent period



Figure 27.2. Eggs of *Eucoleus contortus* from Northern Bobwhite (*Colinus virginianus*). Scale bar = 20 μm. Note the distinctive bipolar plugs that characterize capillarid and trichurid eggs.

is 29–54 days. This species will occasionally cause disease in free-ranging birds including Northern Bobwhite (*Colinus virginianus*), raptors, and, rarely, other species (Cram 1930; Colglazier et al. 1967; Kellogg and Prestwood 1968b; Helmboldt et al. 1971; Clausen and Gudmundsson 1981; Bosch et al. 2000.

***Baruscapillaria obsignata* (=*Capillaria columbae*)**

This species is a cosmopolitan parasite of Galliformes and Columbiformes, but is also found in birds in four other orders (Table 27.1). Chickens, turkeys, partridges, pigeons, guinea fowl, and quail are the most frequent hosts. The life cycle is direct; eggs passed in the feces develop to the infective stage within 6–7 days (Moravec et al. 1987). Embryonated eggs exposed to temperatures below −3.5°C, temperatures higher than 50°C, or desiccation for 14 days are killed (Levine 1937; McDougald 2003). When protected from direct sunlight, embryonated eggs of *B. obsignata* survive exposure to natural environmental conditions for at least 384 days. When exposed to the sun, they can survive at least 129 days (Levine 1937). This species is typically only associated with disease in captive birds.

***Eucoleus annulatus* (=*Capillaria annulata*)**

This cosmopolitan species infects the esophagus and crop of Galliformes and, rarely, Anseriformes. It is considered to be a synonym of *E. contortus* by some researchers. Earthworms in the genera *Eisenia*, *Allolobophora*, *Lubricus*, and *Dendrobaena* are required intermediate hosts (Wehr 1936). Larvae develop to the infective stage 14–21 days after eggs are ingested by a suitable earthworm. Although common in domestic turkeys and capable of causing significant disease in captive Wild Turkeys, prevalence in free-ranging Wild Turkeys is <5% nad no disease has been documented (Maxfield et al. 1963; Hurst et al. 1979; Castle and Christensen 1984; Sasseville et al. 1988).

Eucoleus contortus, Eucoleus dispar, Baruscapillaria falconis,* and *Capillaria tenuissima

These four species are common in raptors (Table 27.2). Although most reports of *E. contortus* and *E. dispar* (possible synonyms, see Table 27.1 note) are from clinically normal birds (Tables 27.1 and 27.2), capillarids present in oral lesions are rarely identified to species and could represent one or both of these species. Oral

lesions can occur in stressed birds with extremely intense infections, although presumably healthy wild birds seem to harbor infections of high intensity with no signs of disease. Mortality associated with unidentified oral capillarids has been reported in captive and free-ranging Gyrfalcons (*Falco rusticolus*), Rednecked Falcon (*Falco chicquera*), and Peregrine Falcons (*Falco peregrinus*) (Brüll 1932; Trainer et al. 1968; Cooper 1969; Clausen and Gudmundsson 1981). *Eucoleus dispar* has an indirect life cycle with earthworms as intermediate hosts, but a direct life cycle is suspected (Barus and Sergejeva 1989). The life cycles of *B. falconis* and *C. tenuissima* are unknown. If earthworm intermediate hosts are required, rodents are believed to serve as paratenic hosts (Olsen 1974).

Pterothominx philippinensis (=*Calodium philippinensis*, =*Capillaria philippinensis*)

Numerous species of birds are competent definitive hosts for *P. philippinensis* including White-breasted Waterhens (*Amaurornis phoenicurus*), Chinese Pond-Herons (*Ardeola bacchus*), Black-crowned Night-Heron (*Nycticorax nycticorax*), Cattle Egret (*Bubulcus ibis*), Yellow Bittern (*Ixobrychus sinensis*), Common Moorhen (*Gallinula chloropus*), Greater Painted-snipe (*Rostratula benghalensis*), pigeons, ducks (*Anas* spp.), and chickens (Bhaibulaya and Indra-Ngarm 1979; Cross and Basaca-Sevilla 1983). Many different species of fish can serve as a required intermediate host for *P. philippinensis* (Bhaibulaya and Indra-Ngarm 1979; Cross 1992) and eggs of *P. philippinensis* are not directly infective to avian or mammalian hosts (Cross 1992). The life cycle of *P. philippinensis* is unusual in that two biologically different populations of adult worms are produced. When larvae are ingested by consumption of an infected fish, they develop into adults in the intestine of the host within 10–11 days. Larviparous females begin to release L1 larvae in 13–14 days (Cross 1992). These L1 reinfect (autoinfect) the same host and develop into adults in 22–24 days. Male and female worms in this second population of adults mate, and females produce typical capillarid eggs that are passed with the feces.

Experimentally infected birds with intense infections became listless, anorexic, have mucoid diarrhea, and may die (Bhaibulaya and Indra-Ngarm 1979). Since free-ranging birds are unlikely to develop infections of high intensity because of lower rates of exposure to infected fish, clinical signs and mortality are not expected. Birds are currently considered to be the natural host for this zoonotic parasite on the basis of their susceptibility in experimental studies and a single report from a naturally infected bittern (*Ixobrychus* sp.) (Cross 1992).

CLINICAL SIGNS

Clinical signs are dependent both on locations within a host where particular species of capillarids develop and on intensity of infection. Birds with low numbers of parasites may not exhibit any clinical signs (Pinto et al. 2004). While most free-ranging wild birds have low intensity infections, those that die from capillarid infections are usually found dead and no clinical signs are observed. Birds harboring large numbers of *B. obsignata*, *E. perforans*, *E. annulatus*, or *P. caudinflata* may have nonspecific signs, which include emaciation, diarrhea, ruffled feathers, anorexia, and reduced water intake. Birds may also exhibit ataxia and weakness and will frequently die if such symptoms are evident (Cram 1936; Reis and Nobrega 1938; Kellogg and Prestwood 1968a; Wehr 1971; Hurst et al. 1979; Mathey and Gutter 1979; De Rosa and Shivaprasad 1999; McDougald 2003; Pinto et al. 2004). Clinical signs may not always be a good indicator of disease. Ring-necked Pheasants (*Phasianus colchicus*) infected with *E. perforans* did not exhibit signs of infection; however, gross and histopathologic lesions were apparent (Pinto et al. 2004).

Few studies have investigated the clinical pathology of capillarid infections and findings vary depending on intensity of infection, extent of tissue damage, and severity of clinical signs. There are reports of normal white blood cell counts and hematocrit values in chickens infected with *B. obsignata* (Wakelin 1965) while others describe heterophilia and eosinophilia (Olson and Levine 1939). Some infected chickens may have slightly elevated globulins and total protein (Berghen 1966). In contrast, pigeons with intense infections of *B. obsignata* have decreased total protein and albumen that is likely due to severe diarrhea (Chubb et al. 1964).

PATHOLOGY

As with clinical signs, the severity of gross and microscopic lesions is dependent on intensity of infection, host species, and parasite species. Low numbers of worms in a normal host rarely cause any appreciable lesions, remain in the superficial mucosa, and are incidental findings (Figure 27.3). Significant lesions and secondary bacterial infections can develop in aberrant hosts however when parasites migrate in the submucosa.

Capillarid species that infect the upper gastrointestinal tract (oral cavity, esophagus, crop) can cause inflammation (Figure 27.4), dilatation of the crop or esophagus, thickening of mucosa, ulceration, bacterial colonization (Figure 27.4), exudation, and fibrinonecrotic plaques (Figure 27.4) and are generally

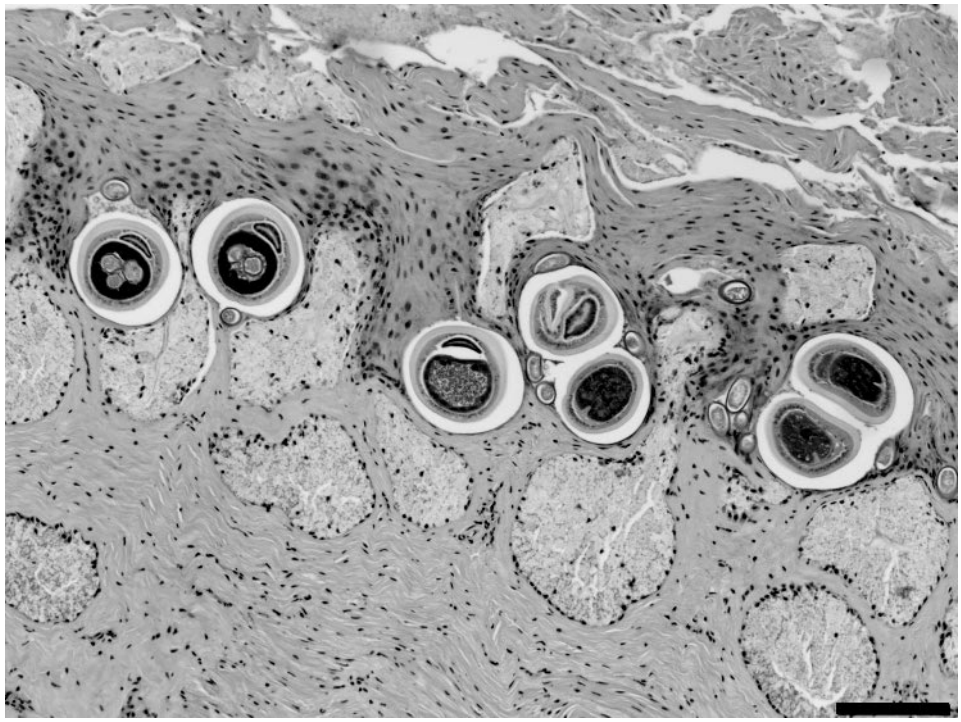


Figure 27.3. Esophagus, Purple Martin (*Progne subis*). Cross sections of adult capillarids and eggs in the mucosa. Little inflammation or disruption may be caused by low-intensity infections in a normal avian host. Hematoxylin and eosin stain. Scale bar = 100 μm. Courtesy of R. W. Gerhold, University of Georgia.

considered to be more pathogenic than intestinal species (Helmboldt et al. 1971; Hurst et al. 1979).

A Blue Jay (*Cyanocitta cristata*) infected with *E. contortus* was emaciated and dehydrated and had a diphtheritic membrane extending from the oral cavity to the proventriculus (Helmboldt et al. 1971). Gyrfalcons heavily infected with *E. contortus* or *E. dispar* had multifocal aggregates of thick, granular, yellow exudate in the oral cavity, pharynx, and esophagus (Clausen and Gudmundsson 1981). Epithelial necrosis and sloughing with occasional ulceration are evident in birds with lesions. Large numbers of adult parasites and eggs can be present in the lamina propria and are frequently surrounded by prominent mononuclear infiltrates. Similar infiltrates are also present in the epithelium and around mucous glands in the esophagus (Cram 1936; Clausen and Gudmundsson 1981). Marked necrosis and sloughing of the epithelium of the esophagus and crop also occurs in Northern Bobwhite infected with *E. contortus* (Cram 1936). Pheasants heavily infested with *E. perforans* have a chronic esophagitis characterized by mononuclear and granulocytic infiltrates in the lamina propria (Pinto et al. 2004). Although the vast majority of lesions are confined to the mucosa, in extreme cases lesions may extend into the muscularis (Hung 1926). Additionally, hyperplasia or squamous metaplasia of the esophageal mucous glands, hyperkeratosis, severe inflammation, bacterial overgrowth, and intercellular edema may be observed.

Intestinal and cecal dwelling capillarid species can cause thickening of the mucosa, erosion or ulceration, intraluminal fluid accumulation, petechiae, exudates, and diarrhea (with or without blood). Microscopic lesions may include epithelial necrosis with erosion or ulceration. Although gross lesions were absent in pheasants infected with *Capillaria phasianiana*, a chronic typhlitis with mononuclear cell infiltrate was noted by microscopy (Pinto et al. 2004).

DIAGNOSIS

Diagnosis of capillarid infection can be made by detection of eggs in fecal samples or mucosal scrapings or by detection of eggs or adult worms in histologic sections of tissues (Figures 27.3 and 27.4). Eggs of capillar-

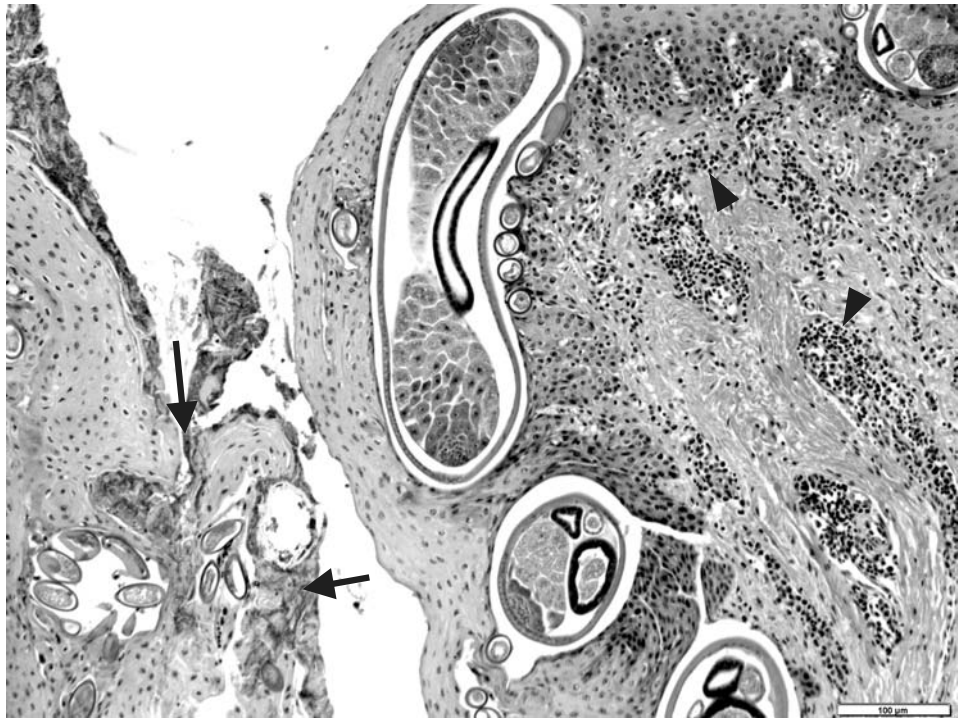


Figure 27.4. Crop, Red-tailed Hawk (*Buteo jamaicensis*). Numerous cross sections of adult capillarids and eggs are present in the mucosa. Note heterophilic inflammation in the submucosa (arrowheads) and bacterial colonies (arrows). Hematoxylin and eosin stain. Scale bar = 100 µm. Courtesy of A. E. Ellis, University of Georgia.

ids are easily recognized by the characteristic bipolar plugs (Figure 27.2); however, egg morphology alone cannot be used to reliably identify the parasite to genus or species. Eggs can be demonstrated by direct examination of fecal material or concentration and flotation of feces with either saturated sugar solution (specific gravity = 1.275), zinc sulfate solution (specific gravity = 1.18), or sodium nitrate (specific gravity = 1.2). Because worms are extremely thin, coiled through the mucosa, and easily broken, acquiring intact worms from tissue can be challenging. Gentle teasing apart of fresh tissue is the easiest way to acquire intact worms.

Identification of capillarids to species depends on knowledge of the site of infection, examination of the vulvar region of the female worms, and detailed study of the posterior and anterior ends of adult worms of both sexes. However, small thin worms embedded in the mucosa that have a stichosome esophagus can be reliably identified as a capillarid. Infection with capillarids is common in many host species; therefore, diagnosis of disease requires demonstration of appropriate clinical signs and/or lesions (gross or microscopic) and a lack of other pathogens.

Differential diagnoses in sick birds include other agents that can cause weakness, emaciation, diarrhea, ruffled feathers, anorexia, and reduced water intake. Gross lesions associated with oral capillariasis in raptors can mimic lesions caused by trichomoniasis or "frounce" (Trainer et al. 1968; Clausen and Gudmundsson 1981), yeast infections, or any agent causing caustic damage to the epithelium.

IMMUNITY

Little is known about the development of immunity to capillarids. Experimental studies with *P. philippinensis* and *Baruscapillaria resecta* suggest that some immunity develops following infection. Attempts to reinfect birds that had cleared an infection with *P. philippinensis* were not successful, suggesting development of immunity (Bhaibulaya and Indra-Ngarm 1979; Cross and Basaca-Sevilla 1983). Antibodies to antigens of *B. resecta* were detected in sera from infected Eurasian Jackdaw (*Corvus monedula*) (Fronska-Popiel et al. 2001); however, cross-reactive antigens appear to be present within the superfamily Trichinelloidea

because sera samples from humans infected with *P. philippinensis* reacted with antigens from *B. obsignata*, *Trichinella sprialis*, and *Trichuris vulpis* (Banzon et al. 1975). The exact mechanism of immunity, if any develops, is currently unknown.

PUBLIC HEALTH CONCERNS

The only known avian capillarid that poses a zoonotic threat is *P. philippinensis*. This species is a zoonosis in parts of Southeast Asia, Egypt, India, and Iran (Cross 1992). This parasite can cause severe enteritis in humans and is acquired by ingestion of raw or undercooked freshwater and brackish-water fish (Cross 1998). Unlike all other capillarids, eggs can embryonate within the human host, hatch, and autoinfect (Cross 1998).

DOMESTIC ANIMAL HEALTH CONCERNS

Capillariasis is an important disease of many species of domesticated and captive wild birds and is associated with how birds are managed in captivity. Several species of capillarids (Table 27.1) infect both wild and domestic birds; however, wild birds do not pose any unique threat to domestic birds because these parasites are easily maintained in captive flocks without exposure to wild birds.

WILDLIFE POPULATION IMPACTS

Capillarid infections rarely cause clinical disease in wild free-ranging birds in spite of being common and large outbreaks have not been reported. Wild birds that are kept in zoological parks or other captive facilities are more likely to develop capillariasis than their free-ranging counterparts. Captive birds are often exposed to large numbers of eggs or infected earthworms in facilities where birds are crowded (Maxfield et al. 1963; Hurst et al. 1979; Castle and Christensen 1984; Sasseville et al. 1988). This problem can be compounded in facilities where large numbers of closely related host species are housed together because several capillarid species have low host-specificity.

TREATMENT, MANAGEMENT IMPLICATIONS, AND CONTROL

Management of capillarid infection in wild birds is not feasible and generally not warranted due to the limited pathogenicity associated with most infections. Captive wild birds should be tested and treated as appropriate to prevent the development of clinical disease associated with stress or acquisition of intense infections while in captivity. Sanitation of living quarters and raising birds on wire bottom cages will greatly decrease intensity of infection and risk of disease in captive birds because transmission of capillarids is dependent on fecal contamination and exposure to either eggs or infected earthworms. Mixing of wild birds of different species is discouraged and wild birds should be excluded from enclosures.

Fenbendazole, febantel, and levamisole are highly efficacious for treatment of capillariasis in numerous avian species including chickens, turkeys, pheasants, partridges. They have been used successfully to treat pigeons infected with *B. obsignata* and raptors infected with an oral capillarid (Lawrence 1983; Kirsch 1984; Santiagno et al. 1985; Norton et al. 1991; Baert et al. 1993; Taylor et al. 1993; El-Kholy and Kemppainen 2005). Subcutaneous injection of ivermectin is effective against infections with *P. caudinflata* in captive guinea fowl (Okaeme and Agbontale 1989). Interestingly, albendazole improved body condition of birds coinfected with *P. caudinflata* and *Heterakis gallinarum* but, because worm burdens were unaffected, it was not considered an effective treatment (Villanúa et al. 2007). Piperazine was not effective in treating *P. caudinflata* in guinea fowl (Ayeni et al. 1983).

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Section IV: Leeches

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28
Leech Parasites of Birds

Ronald W. Davies, Fredric R. Govedich, and William E. Moser

INTRODUCTION

Leeches (Euhirudinea) occur worldwide and form an important component of the benthic invertebrate fauna of most lakes and ponds, with a few species occurring in the quieter flowing sections of streams and rivers. Marine leeches typically feed on marine vertebrates including fish and turtles. Land (terrestrial) leeches can be found in moist, humid tropical and subtropical regions where they inhabit wet rainforests and feed on the blood of vertebrates (Sawyer 1986; Davies and Govedich 2001; Govedich 2001).

Most freshwater leeches are predators and feed on chironomids, oligochaetes, amphipods, and mollusks. Others are temporary, sanguivorous (i.e., bloodsucking) ectoparasites of fish, turtles, amphibians, mammals, or birds. One exception is *Placobdelloides jaegerskioeldi*, which is host-specific for hippopotamus and is an endoparasite found only in the rectum of this aquatic mammal (Oosthuizen and Davies 1994). The majority of terrestrial leeches are sanguivorous, feeding on reptiles, mammals, and birds (Sawyer 1986; Davies and Govedich 2001; Govedich 2001).

There are currently 12 families of leeches, 4 of which contain species that parasitize birds: the Glossiphoniidae, Hirudinidae, Ornithobdellidae, and Haemadipsidae. Glossiphoniids feed using a proboscis (Figure 28.1) that protrudes through a mouth located in the anterior sucker. Hirudinids, ornithobdellids, and haemadipsids all feed by using cutaneous jaws with rows of teeth to cut the skin of the host (Sawyer 1986; Davies and Govedich 2001; Govedich 2001).

Leeches in the genus *Theromyzon* are of particular interest because they tend to specialize on waterbirds. They prefer to feed in the nasal cavities and thus have the potential to cause stress. Intense leech infestations have been correlated with increased water bird mortality (Sawyer 1986; Davies and Govedich 2001).

HISTORY

Leeches are important bloodsucking parasites and predators in terrestrial, marine, and freshwater ecosystems and have been used in medicine for centuries. Many species are still used for medicinal purposes and have been transported throughout the world, particularly *Hirudo medicinalis* (Sawyer 1986; Govedich 2004). The history of the discovery of avian leeches was summarized by Bartonek and Trauger (1975) and Trauger and Bartonek (1977). Most research on leech parasitism of birds has been done in North America on a limited number of species in the glossiphoniid genus *Theromyzon* (Figure 28.2). These studies have led to substantial taxonomic revision of the North American species of the genus *Theromyzon* (Oosthuizen and Davies 1992, 1993; Davies and Oosthuizen 1993).

DISTRIBUTION AND HOST RANGE

Leeches have a variety of hosts (Table 28.1) and generally are not host-specific, tending to feed on any available hosts that live in the same habitat. Avian hosts tend to live in or near water and include waterbirds (ducks, coots, loons, herons, etc.), seabirds (shearwaters, penguins, etc.), and even large land birds such as the cassowary (Benham 1907; Mann 1961; Richardson 1969, 1981; Wilkialis 1970; Mason 1976; Sawyer 1986).

Although species of *Theromyzon* are often referred to as duck leeches (Figure 28.3), they parasitize a wide variety of waterbirds in addition to ducks (Trauger and Bartonek 1977) (Table 28.1). They commonly attack grebes (Storer 2000), coots, loons, gulls, geese, swans, herons, and any other bird that comes within their reach. *Theromyzon tessulatum* has been found in eagles (Collin 1892) and also in the eye of a Hooded Crow (*Corvus cornix*) (Christiansen 1939). *Theromyzon cooperi* will feed in the eye socket of Rock Pigeons (*Columba livia*) in the laboratory (Oosthuizen and Fourie 1985).

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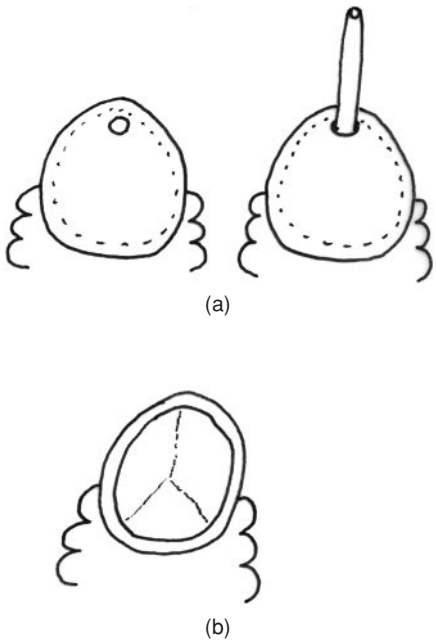


Figure 28.1. Anterior sucker of a leech that feeds with (a) a proboscis (Glossiphoniidae) and (b) a jawed leech (Hirudinidae, Ornithobdellidae, or Haemadipsidae).

Species of *Placobdella*, *Placobdelloides*, *Haementeria*, and other glossiphoniids besides *Theromyzon* have also been known to feed on birds (Table 28.1). For example, *Placobdella ornata* has been found feeding on North American birds (Moore 1964, 1966; Scudder and Mann 1968), while *Placobdella papillifera* feeds readily on fresh heparinized Mallard (*Anas platyrhynchos domesticus*) blood in the laboratory, indicating that this species might feed on waterfowl in the wild (Davies and Wilkialis 1982). *Placobdelloides maorica*, another glossiphoniid, feeds on Pacific Black Duck (*Anas superciliosa*) in New Zealand and it is likely that other members of this genus of leech also take blood meals from waterfowl as well as from reptiles and amphibians. The high frequency of leech attacks on waterfowl likely reflects the availability and abundance of birds rather than host specificity of the leeches.

Among hirudinid leeches, *H. medicinalis* has been recorded from Europe and western Asia and was imported into North America in large numbers for medical uses. This species is still farmed and sold worldwide to medical institutions. Whether or not it has established itself in the wild in North America is still questionable, but Davies (1973) collected a specimen of this species from a field site in Alberta, Canada.

Theromyzon tessulatum and *Theromyzon maculosum* are considered to be European species, but their presence in North America has been well documented. Both species feed on a variety of waterfowl including ducks and geese. Another species, *Theromyzon trizonare*, has only been recorded from the migratory routes of ducks from the Rocky Mountain flyway in the Canadian provinces and states west of the Great Lakes (Elliott and Mann 1979; Davies and Wilkialis 1980).

The African duck leech, *T. cooperi*, is the only representative of the genus reported from Africa (Oosthuizen 1993). *Theromyzon garjaewi* is recorded from Lake Baikal, *Theromyzon propinquum* from South America, and *Theromyzon matthaii* from India (Singhal and Davies 1986).

ETIOLOGY

Species of leeches that parasitize birds are found in four families: the Glossiphoniidae, Hirudinidae, Ornithobdellidae, and Haemadipsidae. Glossiphoniids have a "leaflike" body shape and have eyes located near the centerline of the head (Figures 28.4 and 28.5). They feed using a proboscis that is everted through the mouth and located in the anterior sucker (Figure 28.1). Jawed leeches (hirudinids, ornithobdellids, and haemadipsids) are long and slender in shape, typically have eyes located near the margin of the head, and cutaneous jaws to cut the skin of the host (Figures 28.1, 28.4, and 28.5).

The body of leeches is composed of 32 postoral segments, an anterior sucker, and a ventrally directed posterior sucker that is generally wider than the body at the point of attachment (Figures 28.1 and 28.5). The mouth is found within the anterior sucker (Figure 28.1) and the anus opens on the dorsal surface just anterior to the posterior sucker. Detailed reviews of the taxonomy, anatomy, ecology, and physiology together with taxonomic keys of the freshwater leeches of North America can be found in Davies and Govedich (2001) and Klemm (1985, 1995).

Members of the family Glossiphoniidae have dorsoventrally flattened triannulate bodies that are not differentiated into regions. The ventral anterior sucker is narrower than the body but fused to it. The mouth is a small pore on the ventral surface of the anterior sucker through which a muscular pharyngeal proboscis can be protruded (Figure 28.1). The genus *Theromyzon* has four pairs of eyes in comparison to *Placobdella*, *Placobdelloides*, and *Haementaria*, which have zero, one, or two pairs of eyes, respectively (Figure 28.4).

The Hirudinidae, Ornithobdellidae, and Haemadipsidae have a large mouth occupying the entire cavity of the anterior sucker (Figure 28.1). Hirudinids have three jaws (Figure 28.6), which carry 35–100 acute sharp

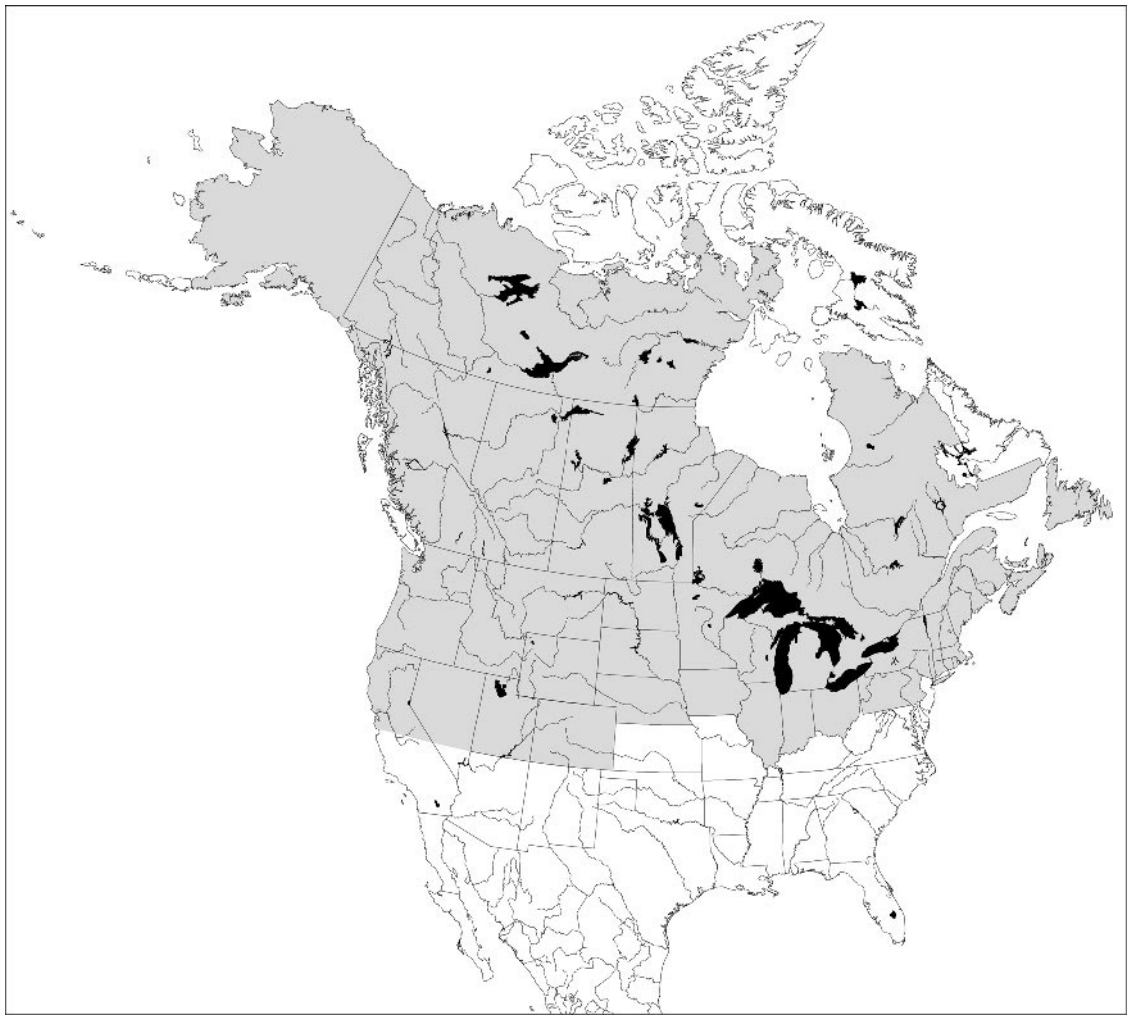


Figure 28.2. Distribution map of species of North American *Theromyzon* (Glossiphoniidae) (shaded area).

teeth in one (monostichodont) or two (distichodont) rows. The ornithobdellids also have three jaws, but the teeth are blunt, reduced, and vestigial. The haemadipsids, like the hirudinids, have sharp teeth in one row on two or three jaws. All three families have five pairs of eyes (Figure 28.4) arranged in an arch, and the body is elongated in freshwater species and only slightly flattened during swimming.

EPIZOOTIOLOGY

All leeches are hermaphrodites showing protandry or cosexuality (Davies and Singhal 1988), with reciprocal cross-fertilization being the norm. Fertilization is internal and the eggs are deposited into a cocoon secreted by the clitellum. The life cycle of all leeches consists of egg, juvenile, and mature hermaphrodite adult. Most leeches attach the cocoon to a substrate and abandon the developing eggs and young (Figure 28.7). However, members of the family Glossiphoniidae brood their eggs and young (Kutschera and Wirtz 2001; Govedich 2004) (Figure 28.8).

The majority of sanguivorous leeches are iteroparous (i.e., capable of breeding several times) and exhibit saltatory growth after reaching a mature size. Most families of leeches produce thick leathery cocoons that contain both eggs and nutrients. These are attached to a substrate and abandoned by the parent. However, the cocoons of Glossiphoniidae are very thin walled (sometimes called egg cases) and are either deposited on the substrate and immediately covered by the body of the parent or are

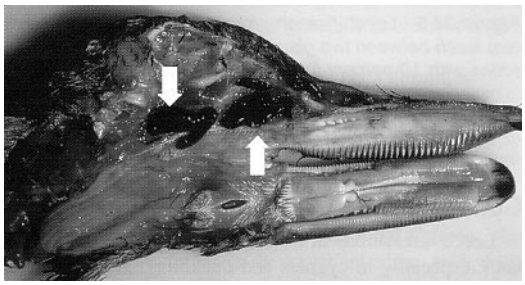


Figure 28.3. Dissected nasal passages of a Mallard (*Anas platyrhynchos*) with an infestation by *Theromyzon* sp. Reproduced from Tuggle (1999), with permission of the author.

directly attached to the ventral surface of the parent. In both cases the hatchlings are attached to the ventral body wall and carried by the parent for up to 5 months in the case of *T. tessulatum* and for about 1 month by *T. trizonare* (Wilkialis and Davies 1980). *Theromyzon trizonare* differs behaviorally from all other species of *Theromyzon* in North America by attaching its cocoon to its ventral body wall so that if disturbed, the parent can move away, carrying the cocoon with it (Davies and Wilkialis 1980).

Once on an avian host, leeches generally move to the head where most frequently they attach themselves inside the nasal passages (Figure 28.3) or, more rarely, around the eyes, particularly under the nictitating membrane and eyelids. Leeches in these positions are safe from removal by host scratching behavior. Leeches entering the nares often penetrate further into the trachea, lungs, and into the air spaces of the skull. Less common attachment sites include the mouth, cloaca, and the skin of the legs, feet, and breast.

Leeches are also intermediate hosts for some digenetic trematodes that are commonly found in ducks and grebes (Sawyer 1986; Storer 2000). Leeches can also serve as intermediate hosts for cestodes of ducks (Sawyer 1986) and can potentially serve as intermediate or paratenic hosts of cestodes, acanthocephalans, and nematodes.

Leeches are sometimes consumed as prey by other invertebrates, fish, snakes, and waterfowl. Leeches respond to the presence of a potential predator by attaching to a substrate and flattening their body, making it difficult for the predator to pull it off of the substrate. Other defensive responses include forming a ball to protect attached eggs and young, and burrowing into soft mud or leaf material.

Sanguivorous leeches spend a relatively short time (1–8 h) taking a blood meal on the host (bird) and are more frequently found free living in the benthos of the lake or river or under vegetation in moist terrestrial habitats.

Theromyzon trizonare and *T. tessulatum* and presumably other *Theromyzon* species that feed on waterfowl require a minimum of three complete blood meals in order to reach reproductive maturity (Wilkialis and Davies 1980; Davies 1984). This is significantly fewer than *H. medicinalis*, which requires 10 or more meals to reach maturity (Putter 1907, 1908; Blair 1927). Over 80% of *T. trizonare* take 3 blood meals from birds in the first 6 months after hatching. The remainder of the population overwinters after 2 blood meals and takes the third meal in the spring, so that all of the population reproduces approximately 12 months after hatching. Individuals that overwinter after 3 meals may decline in weight before the spring and require a fourth meal before reproduction commences.

Weight of *T. trizonare* increases proportionately with each successive blood meal, ranging from 7.3 g after the first meal to 20.4 g after the third (Davies 1984). Also shortly after taking a blood meal, *Theromyzon* shows a further increase in body weight, presumably from water uptake.

Sanguivorous leeches in proximity to a potential host respond to changes in illumination (shadows), vibrations in the water, chemical stimuli, and possibly differences in temperature between the host and the surrounding water. For example, species of *Theromyzon* attach to and attack a host with a body temperature in the range of 37–40°C (Dickinson and Lent 1984).

Disturbances in the water can be detected by the sensilla of a hungry leech. These are frequently the first indication of the presence of a potential host and a hungry leech will move in their direction. Although movements toward a potential host by sanguivorous leeches are not specific, the next step, determining whether or not the potential host is an appropriate source of blood, involves selectivity. When a shadow passes over a hungry sanguivorous leech, it reacts by making searching movements or even swimming toward the potential host. After attachment to the potential host with the posterior sucker, the leech makes a number of searching movements with the anterior portion of the body. If the host is not appropriate, the leech detaches. If *T. trizonare*, which primarily feeds in the nares, attaches to the feathers rather than the beak, it will move about on the bird for up to 30 minutes in search of a suitable location to feed. Suitable host recognition is presumably by chemoreception and/or mechanoreception.

When birds are present and available, hungry *Theromyzon* are found primarily in shallow waters on the upper exposed surfaces of rocks, stones, and plants where the probability of meeting a host is high.

Table 28.1. Host and distribution records for families and species of leeches that parasitize birds (Sawyer 1986).

Leech family	Leech species	Host families	Distribution
Glossiphoniidae	*Haementeria depressa*	Anatidae	Cosmopolitan
	Oosthuizobdella garoui	Podicipedidae	
	Placobdella costata	Rallidae	
	Placobdella ornata		
	Placobdella papillifera		
	Placobdelloides maorica		
	Theromyzon sp.	Anatidae	
	Theromyzon affinis	Ardeidae	
	Theromyzon bifarium	Podicipedidae	
	Theromyzon cooperi	Gaviidae	
	Theromyzon garjaewi	Rallidae	
	Theromyzon maculosum	Scolopacidae	
	Theromyzon matthaii	Recurvirostridae	
	Theromyzon mollissimum	Charadriidae	
	Theromyzon pallens	Laridae*	
	Theromyzon propinquum	Corvidae*	
	Theromyzon rude	Accipitridae*	
	Theromyzon sexoculatum		
	Theromyzon tessellatoides		
	Theromyzon tessulatum		
	Theromyzon trizonare		
Ornithobdellinae	*Hirudobdella antipodum*	Procellariidae	New Zealand
	Ornithobdella edentula	Spheniscidae	New Zealand, Snare's Islands
	Aetheobdella hirudoides	Bush birds	Southeastern Australia
Hirudinidae	*Myxobdella annandalei*	Ardeidae	Indo-Pacific
	Richardsonianus howensis	Birds hypothesized	Australia, Lord Howe Island
	Hirudo medicinalis	Phasianidae	Europe, Asia
	Hirudo nipponia	Phasianidae	Europe, Asia
	Dinobdella ferox	Phasianidae	Indo-Pacific
Haemadipsidae	*Haemadipsa zeylanica*	Phasianidae	Indo-Pacific
	Chtonobdella bilineata	Cracticidae	Indo-Pacific
	Chtonobdella limbata	Muscicapinae	Indo-Pacific
	Placobdella novabritanniae	Casuariidae	Indo-Pacific

*Atypical hosts.

Although leeches in general are strongly photonegative, hungry sanguivorous leeches are frequently phototopositive. After a meal or when carrying eggs or young, leeches remain in sheltered areas away from light and potential predators. In the winter, when feeding does not occur, leeches move into deeper water under ice cover.

Behavior of terrestrial sanguivorous leeches is similar to freshwater species with individuals moving toward shadows and vibrations while searching for potential hosts. Unlike freshwater species, *Amicobdella nigra* and other terrestrial haemadipsid species react to the presence of carbon dioxide by increasing searching movements and moving toward higher carbon dioxide concentrations. As a result, leeches move upward to the mouth, eyes, and nostrils where the majority of feeding occurs.

Direct passive transfer of leeches can occur during contact between two ducks. Davies et al. (1982) examined passive transfer of two sanguivorous glossiphoniids, *T. trizonare* and *P. papillifera*, between ducks. *Theromyzon* adults were transferred from duck to duck both when the leeches were feeding in the nares of the ducks and while they were on the body under the feathers, where feeding was never observed. *Placobdella papillifera* were never found in the nares of the ducks, but were carried on the body under the feathers where a low proportion were observed to feed.

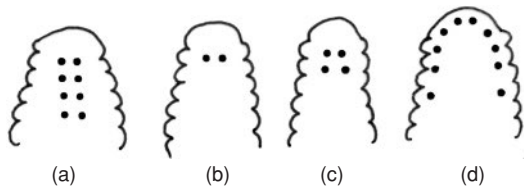


Figure 28.4. Relative eye positions of the (a–c) Glossiphoniidae and (d) Hirudinidae, Ornithobdellidae, and Haemadipsidae.

Because of the diversity of physiochemical conditions encountered in different aquatic ecosystems and while taking a blood meal, sangivorous leeches have considerable physiological plasticity. They are capable of living in waters with very low salt concentrations as well as in waters exceeding the salinity of sea water. Similarly, some species are capable of surviving in the absence or near absence of oxygen, as might occur when taking a blood meal from the nares of a bird, or in supersaturated water, as might occur when a leech leaves its host and lands in the weedy shallow waters of a warm placid lake or pond.

CLINICAL SIGNS

Infected birds can be identified by the presence of leeches in or near the nares, around the eyes, or near the cloaca where there are many capillaries and a good blood supply. Leech bite wounds may also be found following feeding, after the leech has left the host.

Infested birds exhibit various degrees of discomfort, including shaking their heads, scratching their bill with their feet, and/or forcing air through their nasal passages (sneezing), sometimes even when flying (Bartonek and Trauger 1975; Trauger and Bartonek 1977; Sawyer 1986). Engorged leeches protruding from the nares, eyes, or attached elsewhere on the host are readily visible, even from a distance using binoculars (Bartonek and Trauger 1975; Trauger and Bartonek 1977). Periorbital feathers soiled with ocular discharge, conspicuous eye irritations, eyelids matted together, and impairment of vision, including blindness, are also signs of leech infestation (Bartonek and Trauger 1975; Oosthuizen and Fourie 1985).

Symptoms of heavy parasitism by *Theromyzon* spp. are similar to botulism (Oosthuizen and Fourie 1985; Fourie et al. 1986). Heavily infested birds are lethargic and sometimes exhibit various degrees of paralysis. In Africa, after heavy infestation by *T. cooperi*, some birds are unable to walk or fly because their leg and/or wing muscles have become paralyzed. Later, the neck muscles may also become paralyzed, leading to a drooping head (Fourie et al. 1986). Heavily infested birds often also exhibit short, labored breathing (Bartonek and Trauger 1975).

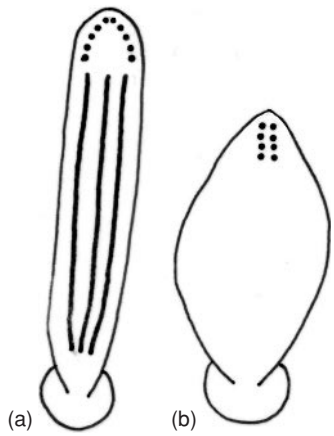


Figure 28.5. Body shape of (a) jawed leeches and (b) glossiphoniids.

PATHOGENESIS AND PATHOLOGY

Parasitism by leeches can result in temporary blindness, clouding of the cornea, and in some cases, collapse of the globe of the eye (Bartonek and Trauger 1975; Tuggle 1999). *Theromyzon trizonare* has been implicated in waterfowl deaths due to either suffocation from numerous engorged leeches in the nasal passages or stress and general weakness (Davies and Wilkialis 1981). Ducklings that were exposed in the laboratory to numbers of *T. trizonare* comparable to those recorded from wild birds developed signs of stress and debilitation. These signs included significant increases in biomass of the adrenals, spleen, and thymus, a significant decrease in liver biomass, and a significant decrease in the keel/sternum ratio, a relative measurement of emaciation. Ulceration or inflammation of the nasal mucosa of the parasitized ducks was not observed by microscopy (Davies and Wilkialis 1981).

DIAGNOSIS

The identification of *Theromyzon* (family Glossiphoniidae) is based on the number of annuli separating the male and female gonopores. In North America, the taxonomy of the genus has been revised by Oosthuizen and Davies (1992, 1993) and Davies and Oosthuizen (1993), so care must be taken to bring earlier identifications into line with this work. Fourteen species of *Theromyzon* leeches have been described from ducks

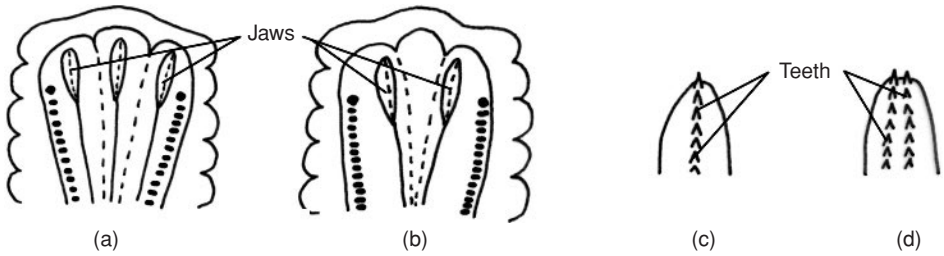


Figure 28.6. Jaw placement in (a) hirudinids and ornithobdellinids and (b) haemadipsids. Number of teeth rows on jaws: one row or monostichodont (c) and two rows or distichodont (d).

in different parts of the world. These can be divided into four groups on the basis of the number of annuli (2, 3, 4, or 5) separating the male and female gonopores.

In North America, *T. trizonare* (with three annuli), *Theromyzon rude*, *Theromyzon bifarium*, *and* *T. maculosum* (each with two) and *T. tessulatum* (with four) differ in the number of annuli between the gonopores and in several other reproductive characteristics (Oosthuizen and Davies 1993; Davies and Govedich 2001).

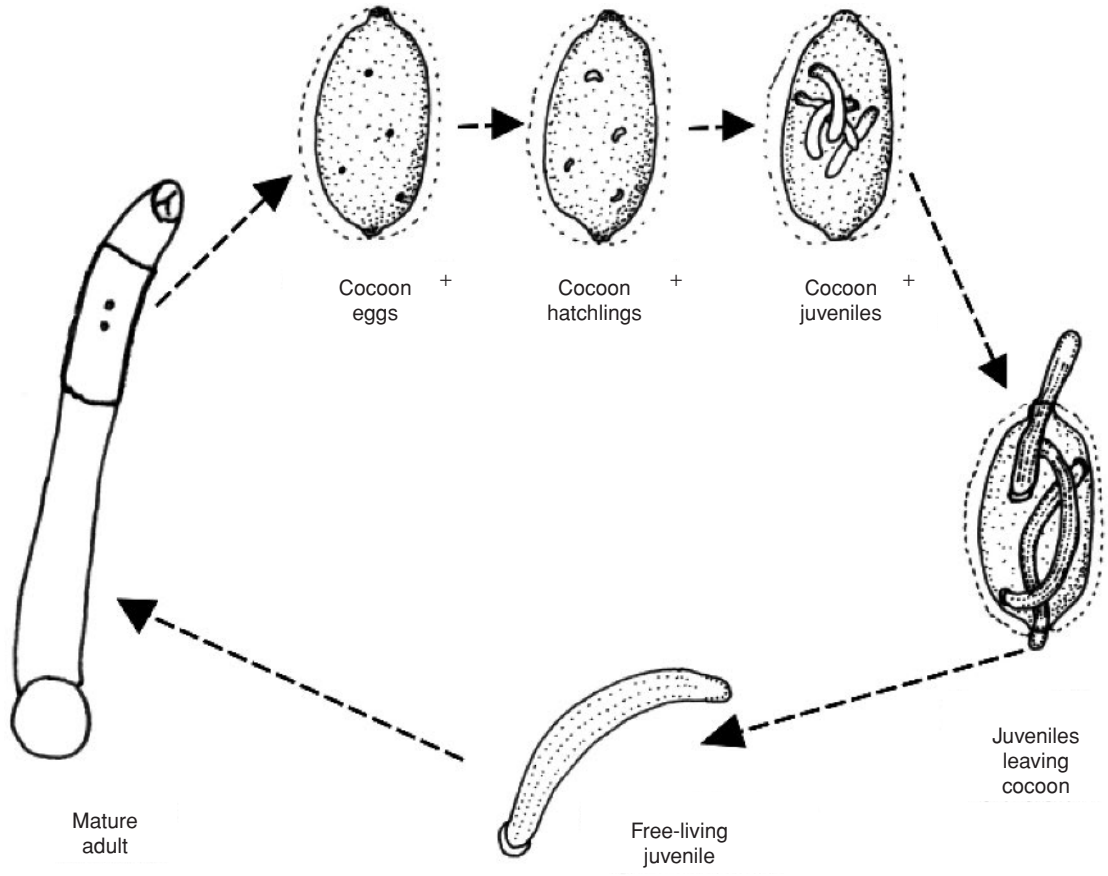


Figure 28.7. Typical life cycle of nonbrooding leeches (including hirudinids, ornithobdellinids, and haemadipsids). The cocoon is attached to a substrate and abandoned.

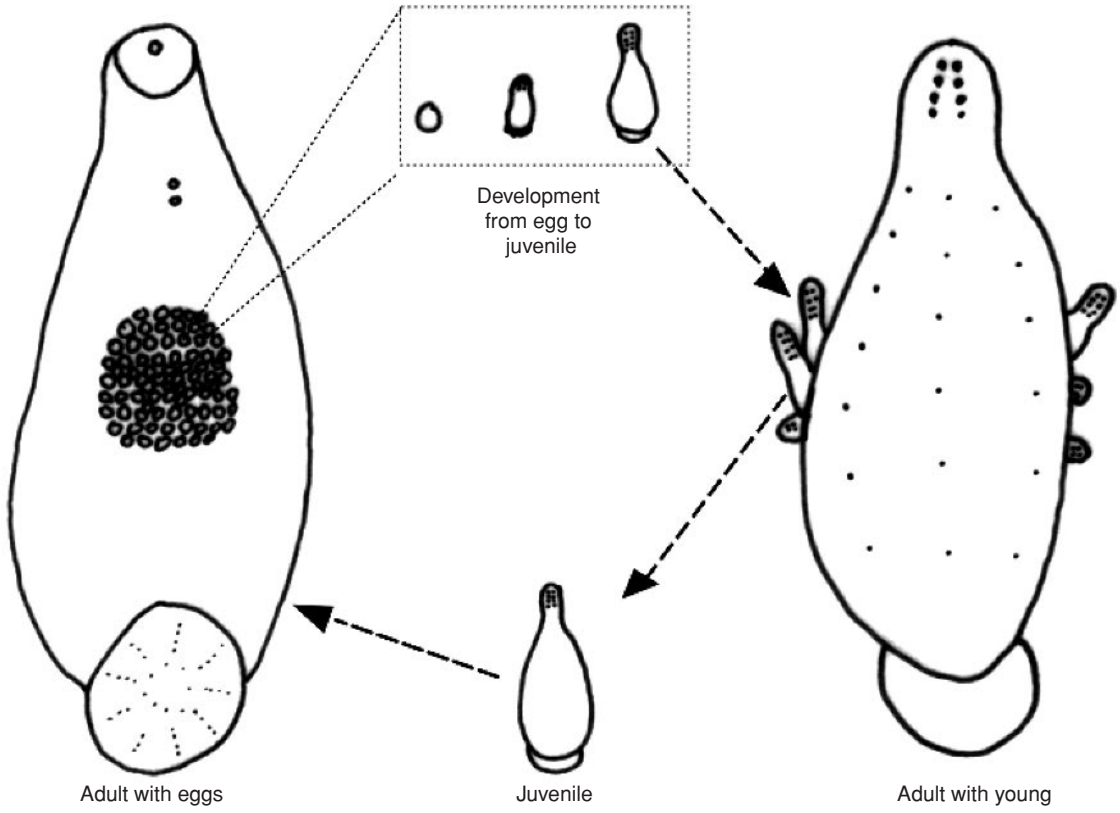


Figure 28.8. Life cycle of glossiphoniid leeches. Eggs (cocoons) may be attached to a substrate (nest) or to the parent (brooding, shown here). Young are brooded on the parent until they are mature.

The African duck leech, *T. cooperi*, is the only representative of the genus reported from Africa (Oosthuizen 1993) and, like *T. garjaewi* from Lake Baikal, has two annuli between its gonopores. Both *T. propinquum* from South America and *T. matthaii* from India (Singhal and Davies 1986) have three annuli between the gonopores.

Freshwater and terrestrial leeches in the families Hirudinidae, Ornithobdellidae, and Haemadipsidae are identified based on external features such as the presence of auricles, ocular areoli (Haemadipsidae only), and the nature of the jaws and teeth (numerous sharp teeth for Hirudinidae; reduced, blunt vestigial teeth in the Ornithobdellidae). Species within these families are determined using combinations of characters such as the color pattern, the structure of the female and male reproductive systems, and the structure of the digestive system.

Identification of leeches can be difficult, especially if they are not properly relaxed and preserved. Many species of leeches require dissection or histological sectioning for species-level identification. Notes on dorsal and ventral coloration and pattern should be made prior to preservation as leech pigmentation typically fades during the preservation process. Leeches should be narcotized before fixation. Otherwise, they contract sharply and obscure structures used in identification. A recommended method is to add 70% ethanol dropwise into a vessel of water containing the leech until the leech no longer reacts to a blunt probe. Other methods of narcotizing leeches and specific preservation protocols are available (Klemm 1985, 1995; Davies and Govedich 2001). A voucher series of specimens narcotized and then fixed in 10% formalin followed by 95% ethanol is recommended.

IMMUNITY

There is no acquired immunity to leech infestation, but leeches may be mechanically removed or die when

feeding on a host. Mortality among leeches during feeding on ducks varies with each blood meal. Mortality is highest during the first meal and lowest during the third. Most leech mortality occurs when they are ingested by the host as they attempt to feed in the nares. Leeches ingested in this manner or that were fed directly to ducks in the laboratory were completely digested and never recovered from duck feces. Following successful feeding, *Theromyzon* leeches leave the host by one of two methods. Engorged leeches either drop out of the nares by themselves or are forcibly ejected by sneezing. The latter occurs more frequently during later meals. Species of *Theromyzon* are susceptible to desiccation and will die if the bill and nares dry out.

PUBLIC HEALTH AND DOMESTIC ANIMAL HEALTH CONCERNS

Leeches are typically not host-specific and members of the Hirudinidae and Haemadipsidae have been known to feed on humans and other large mammals as well as birds. Leeches are primarily a nuisance and do not generally cause major problems for humans. However, leeches can cause problems for people allergic to their feeding secretions or who have a suppressed immune system. The site of incision during feeding can also become secondarily infected, and in a few rare cases leeches have caused problems in humans when they have entered the mouth, anus, or vagina. Members of the genus *Theromyzon* (Glossiphoniidae) are host-specific to birds although there are two cases from Europe where *T. tessulatum* was reportedly attached to a human eye and also found inside a human larynx. Both cases required medical attention (Auw-Haedrich et al. 1998; Kuehnemund and Bootz 2006). While Kuehnemund and Bootz (2006) described this leech as *T. tessulatum*, examination of the photographs in the paper suggest the leech was more likely a species of *Hirudo*.

Leeches certainly have the potential to cause stress and mortality in domestic birds, especially cygnets and ducklings. Leeches can be removed by a trained veterinarian and birds usually recover within a few hours after leeches have been removed.

TREATMENT AND CONTROL

Several chemotherapeutic remedies have been recommended for alleviation of leech infestation. Each regimen includes immersion or rinsing of the nasal chambers with an aqueous irritant for a few seconds until the leeches detach. Recommended solutions include lactic acid (4–10%), weak gastric juice, sodium chloride (10%), acetic acid, weak ammonia, and opthaine or sulfathiazole (Kuznetsova 1955; Ponomarenko 1960; Lang 1969). Removal of conspicuous leeches by hand or with forceps has also been suggested (Bartonek and Trauger 1975; Trauger and Bartonek 1977) but this is not recommended.

Measures to reduce leech populations in the field are not presently feasible or advisable as no agent (chemical or physical) selectively kills leeches. Use of nonspecific agents may damage other components of the aquatic ecosystem and have more serious effects on wildfowl populations than the leeches themselves. Keeping birds away from habitats with leeches is theoretically possible but usually impractical.

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Section V: Arthropods

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29
Phthiraptera, the Chewing Lice

Dale H. Clayton, Richard J. Adams, and Sarah E. Bush

INTRODUCTION

Chewing lice are small, wingless, dorsoventrally compressed insects that parasitize birds and some mammals. They range from $\leq$1 mm to about 10 mm in length. Avian chewing lice belong to one of two suborders: Amblycera, which occur on feathers and skin, or Ischnocera, which are more restricted to feathers. As a group, chewing lice are among the most host specific of all parasites with many species being found on only one genus or species of host. Some species of chewing lice are less specific, however, occurring on multiple host genera, families, or even orders. Most bird lice feed on feathers, dead skin, and skin products, which they appear to metabolize with the aid of endosymbiotic bacteria. Some species also feed on blood, and a few species of Amblycera feed exclusively on blood.

Chewing lice are normally found in small, subclinical infestations that are kept in check by regular host grooming, including preening with the bill and scratching with the feet. Because small numbers of lice have no apparent effect on the host, the conventional wisdom has been that chewing lice are relatively benign parasites. When present in large numbers, however, they can cause severe irritation and reduced host survival and reproductive success. They can also affect the host indirectly by serving as vectors of other parasites, including some species of filarial worms. The time and energy that birds must devote to preening to keep lice in check may also be costly. In this chapter we focus on the impact of lice on wild birds.

SYNONYMS

Infestation with lice is technically known as pediculosis, although this term is not often used in reference to wild birds.

HISTORY

Linnaeus included 23 species of chewing lice in his 10th edition of the *Systema Naturae* (Linnaeus 1758). Price et al. (2003) provided a brief history of subsequent taxonomic work on chewing lice. Blood feeding by amblyceran chewing lice was documented as early as 1778, thus establishing the possibility that chewing lice might elicit allergic reactions, transmit pathogens, and leave cutaneous wounds that can fester (Price and Graham 1997; Prelezov et al. 2006). Although blood feeding by some species has been documented in detail (Derylo and Gogacz 1974), the extent of blood feeding by most species of Amblycera remains poorly understood. Ischnoceran chewing lice are recognized vectors of some filarial worms (Bartlett 1993) and cause thermoregulatory stress (Booth et al. 1993), reduced mating success (Clayton 1990), and reduced survival in wild birds (Clayton et al. 1999).

HOST RANGE AND DISTRIBUTION

Many chewing lice are extremely host specific, but host specificity should never be assumed. The systematics of chewing lice has suffered greatly at the hands of taxonomists who describe new species on the basis of host associations, rather than on the basis of the lice themselves. This practice has made it necessary to synonymize hundreds of species over the years (Price et al. 2003).

Clayton et al. (2004) reviewed factors governing the host specificity of chewing lice, and the related phenomenon of host-parasite cospeciation. Cospeciation occurs when speciation in a host group is "mirrored" by parallel speciation in its parasites. Repeated bouts of cospeciation yield congruent host-parasite phylogenies. Page (2003) provided an overview of cospeciation, including many examples involving lice.

Nearly all species of birds that have been checked have lice. Since many avian hosts have yet to be examined, new species of lice and new host records undoubtedly await discovery, particularly in the tropics. A thoroughly revised comprehensive world checklist and biological overview of chewing lice was published

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by Price et al. (2003). It includes a key to the 253 known genera of bird lice.

The first confirmed fossil louse was recently described from Eckfeld maar, Germany (Wappler et al. 2004). This fossil is approximately 44 million years old, and feathers preserved in its foregut confirm that it was a bird louse.

Louse diversity varies considerably among avian taxa, ranging from just one species per host, as in the case of the Ostrich (*Struthio camelus)*, to more than 20 species per host, as in the case of some tinamous (Ward 1957). It is not clear what factors govern the diversity of lice among avian taxa. Host ecology and behavior may play a role. For example, birds which dive underwater have fewer genera of lice than their nondiving relatives (Felsõ and Rózsa 2006). Host morphology may also play a role. Among species of birds, the abundance component of diversity is correlated with host body size and bill morphology (Clayton and Walther 2001).

Abiotic factors such as humidity can also influence louse distributions. Species of lice that acquire water from the air are susceptible to desiccation and death in arid environments (Rudolph 1983). Consequently, birds living in arid environments tend to have fewer lice than similar birds in humid environments (Moyer et al. 2002b). Other factors, such as latitude, do not appear to affect parasite richness or abundance (Clayton et al. 1992).

The geographic distribution of lice often corresponds to that of the host. However, some lice show a more restricted distribution, being abundant on certain host populations, but rare or absent on others. For example, the louse *Quadraceps ridgwayi* occurs on Australasian populations of the Eurasian Oystercatcher (*Haematopus ostralegus)*, but is absent from this species in Africa and Eurasia (Clay 1976). In short, lice can exhibit geographic "specificity" that is nested within their host specificity.

Geographic specificity of lice can be helpful in elucidating the ecological history of the host. For example, there is little genetic structure among island populations of the endangered Galapagos Hawk (*Buteo galapagoensis)*. However, the lice *Deegeriella regalis* and *Colpocephalum turbinatum* on hawks from different islands are genetically distinct, suggesting that the hawk populations on different islands are, in fact, isolated (Whiteman and Parker 2005).

In some cases geographic specificity may explain louse distributions better than the relatedness of their hosts. For example, lice from toucans (*Ramphastos* spp.) are more likely to be found on distantly related toucans in the same geographic region than on more closely related toucans in different regions (Weckstein 2004).

Table 29.1. Higher level classification of lice (Insecta: Phthiraptera).

Suborder	Family	Genera	Species
Amblycera	**Menoponidae**	**68**	**1,039**
	Boopidae*	8	55
	Laemobothriidae	**1**	**20**
	Ricinidae	**3**	**109**
	Gyropidae	9	93
	Trimenoponidae	6	18
Ischnocera	**Philopteridae**†	**138**	**2,698**
	Trichodectidae	19	362
Rhynchophthirina	Haematomyzidae	1	3
Anoplura	(16 families)	49	532

Families in boldface are found on birds; the others are found on mammals. Data were compiled from Price et al. (2003).
*One genus (*Therodoxus*) occurs on birds (cassowaries).
†One genus (*Trichophilopterus*) occurs on mammals (lemurs).

ETIOLOGY

Chewing lice are members of the insect order Phthiraptera, which also contains the sucking lice (Table 29.1). Modern classifications divide Phthiraptera into four suborders, three of which comprise the chewing lice: Amblycera, Ischnocera, and Rhynchophthirina. Members of these three suborders all have mandibulate, chewing mouthparts. Most species of Amblycera and Ischnocera are parasites of birds, although about 12% of the species, along with the three species of Rhynchophthirina, are parasites of mammals. Members of the fourth suborder, Anoplura, all of which parasitize mammals, are known as sucking lice because they have piercing-sucking mouthparts. Durden (2001) provides an excellent review of the sucking lice, as well as taxa of chewing lice found on mammals.

Despite sharing mandibulate mouthparts, the three suborders of chewing lice are not the closest relatives to one another. Ischnocera and Anoplura share a more recent common ancestor than either suborder shares with Amblycera (Lyal 1985; Barker et al. 2002; Johnson and Whiting 2002). For this reason, modern workers classify all lice in the single order Phthiraptera, rather than following the traditional classification in which the chewing lice are placed in one order (Mallophaga), with sucking lice in another order (Anoplura). Recent molecular and morphological evidence (Johnson et al. 2004; Yoshizawa and Johnson 2006) also confirms the hypothesis that Phthiraptera is polyphyletic (Lyal 1985). The sister group of Amblycera is actually Liposcelididae, a family of nonparasitic bark/book lice in the order Pscoptera.

The two suborders of lice on birds, Amblycera and Ischnocera, are relatively easy to distinguish. Roughly 30% of all species of bird lice belong to the suborder Amblycera. Members of this suborder feed on blood and feathers, have maxillary palps, and concealed antennae with four segments (Figures 29.1a–29.1c). The majority of species of bird lice (70%) belong to the suborder Ischnocera and feed primarily on feathers and dead skin that are metabolized with the aid of endosymbiotic bacteria (Fukatsu et al. 2007). Members of the Ischnocera lack maxillary palps and have protruding 3–5 segmented antennae (Figures 29.1d–29.1g). The Amblycera are more mobile than the Ischnocera and will make short forays away from the host and abandon a dead host to search for a new one. In contrast, the Ischnocera, which are often called "feather lice," are so specialized for life on feathers that many species are not even capable of moving from feathers onto the skin of the host (e.g., Clayton et al. 1999).

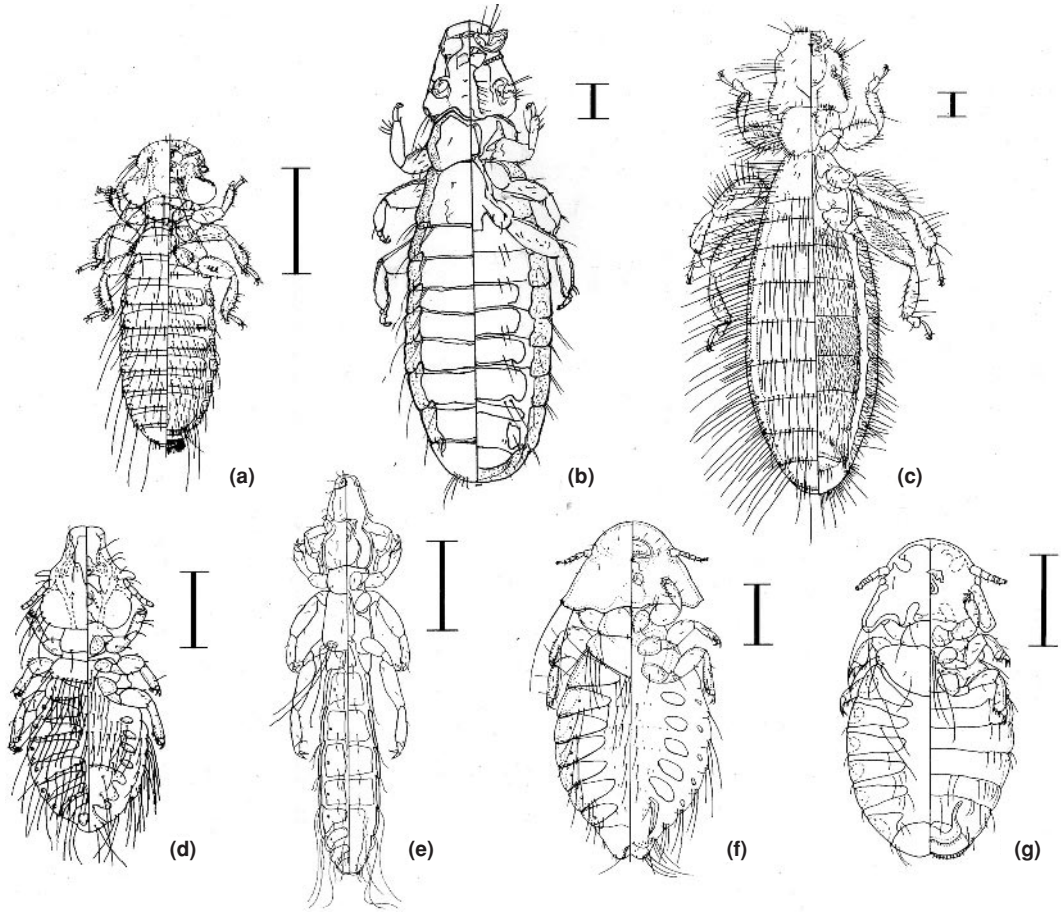


Figure 29.1. Representatives of the six families of avian chewing lice (see Table 29.1). Dorsal morphology to left of midlines, ventral morphology to right. M, male, F, female. Scale bars = 0.5 mm. (a) *Colpocephalum holzenthali* (Amblycera: Menoponidae), F, host: Barred Forest-Falcon (*Micrastur ruficollis*); (b) *Ricinus* sp. (Amblycera: Ricinidae), F, host: passeriformes spp; (c) *Laemobothrion maximum* (Amblycera: Laemobothriidae), F, host: *Buteo* sp.; (d) *Philopterus* sp. (Ischnocera: Philopteridae), M, host: passeriformes spp.; (e) *Columbicola columbae* (Ischnocera: Philopteridae) M, host: Rock Pigeon (*Columba livia*); (f) *Goniodes australis* (Ischnocera: Goniodidae), F, host: Malleefowl (*Leipoa ocellata*); (g) *Heptapsogaster* sp. (Ischnocera: Heptapsogasteridae), F, host: Tinamiformes spp. (a) Redrawn from Clayton and Price (1989); (b) redrawn from Ledger (1980); (c) redrawn from Nelson and Price (1965); (d) redrawn from Price and Hellenthal (1998); (e) by the second author; (f) redrawn from Emerson and Price (1986); (g) by the second author.

Ecologically speaking, bird lice can be divided into five categories based on overall morphology and how they escape host preening: (1) agile Amblycera that run quickly across the skin or feathers (Figure 29.1a); (2) very large Amblycera that slip sideways between the feathers (Figures 29.1b, c); (3) sluggish, triangular-headed Ischnocera that avoid preening by dwelling mainly on the head and neck (Figure 29.1d); (4) elongate Ischnocera that hide between the barbs of wing and tail feathers (Figure 29.1e); and (5) sluggish Ischnocera that burrow into the lush, downy regions of neck and abdominal feathers (Figures 29.1f, g). Although some species cannot be placed into one of these categories, this scheme illustrates the principal adaptive zones occupied by bird lice.

EPIZOOTIOLOGY

Chewing lice are obligate, permanent parasites that complete their entire life cycle on the body of the host. This cycle consists of the egg, three nymphal instars, and the adult stage. The eggs incubate for 4 to 10 days, depending on the species, and each nymphal instar requires 3–12 days for completion (Marshall 1981). Most adult lice are thought to live about a month, with females producing about one egg per day, for a total of 12–20 eggs.

Transmission of chewing lice among hosts often requires physical contact between birds, such as between mates and parents and their offspring in the nest (Hillgarth 1996; Tompkins et al. 1996). However, ischnoceran lice are also capable of moving between hosts by phoresis or "hitchhiking" on hipposboscid flies (Keirans 1975). Phoresis can be common. For example, lice were attached to 44% of 156 hippoboscid flies that were removed from European Starlings (*Sturnus vulgaris*) (Corbet 1956). Because hippoboscid flies are not generally as host specific as lice, phoresis may help explain why some lice have a wide range of taxonomically diverse avian hosts (Clayton et al. 2004; Harbison et al. in press). Although phoresis is common among the Ischnocera, it is rare among the Amblycera because members of this suborder appear to be morphologically incapable of attaching to flies (Keirans 1975).

CLINICAL SIGNS, PATHOLOGY, AND PATHOGENESIS

When present in large numbers, amblyceran lice can cause extensive feather and skin damage, leading to dermititis, puritis (itching), insomnia, and excessive preening and scratching. Although ectoparasites that feed solely on blood can cause anemia in their hosts, this has seldom been reported in the case of avian lice, perhaps because so few species feed exclusively on blood (Marshall 1981; Price and Graham 1997). Poultry lice cause reductions in food consumption, body mass, and egg production as a result of irritation (Nelson et al. 1977; Arends 1997; Prelezov et al. 2006). For example, infestations of the chicken head louse, *Cuclotogaster heterographus,* cause severe restlessness and debility (Kim et al. 1973) and sometimes kill chicks outright (Loomis 1978). Grooming rates of chickens infested with the louse *Menacanthus stramineus* also increase significantly (Brown 1974). Despite their potential effects, poultry lice are considered a relatively minor problem in modern operations because they are relatively easy to control.

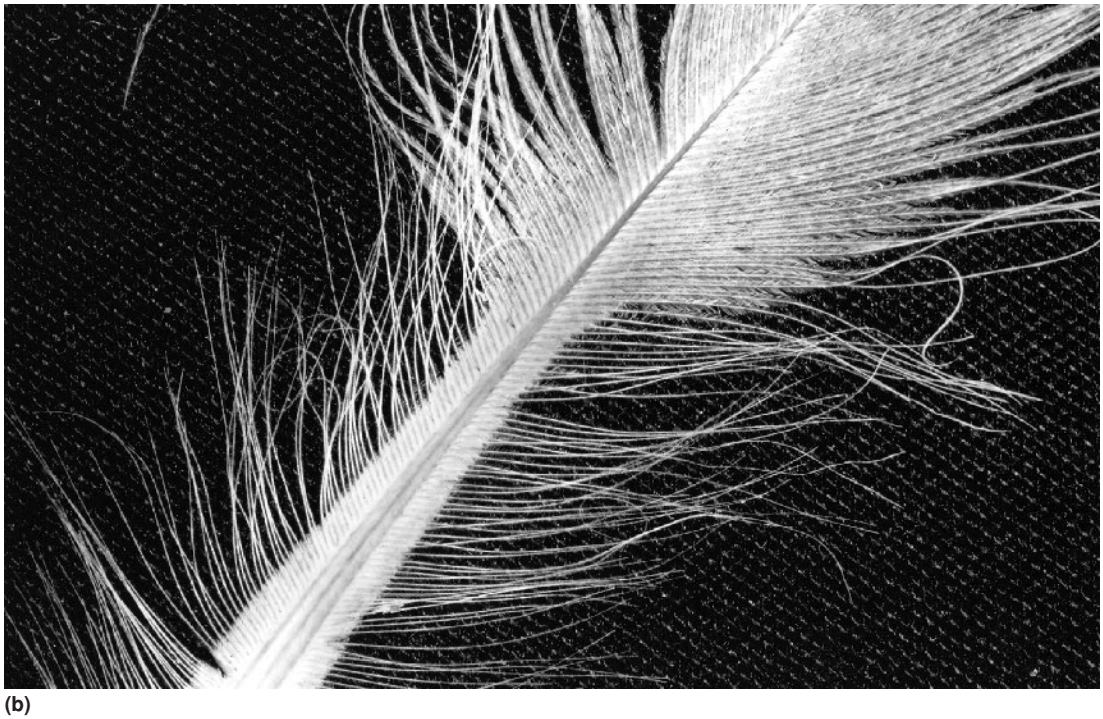
Lice also have negative effects on wild birds. Severe hemorrhagic ulcerative stomatitis and death has been documented in juvenile American White Pelicans (*Pelecanus erythrorhynchos*) infested with the menoponid louse, *Piagetiella peralis*, a species that lives within the pouch of these hosts (Samuel et al. 1982; Dik 2006). Although it is not clear whether lice were the principal cause of death, they clearly contributed to poor condition in heavily infested young pelicans.

One of the most thorough case studies of the impact of lice on wild birds involves free-ranging Rock Pigeons (*Columba livia*). Populations of the ischnoceran lice *Columbicola columbae* and *Campanulotes compar* increase dramatically on pigeons with naturally or experimentally impaired preening ability (Clayton et al. 2005). These two species feed on abdominal contour feathers (Figure 29.2) and reduce the density of the plumage. This leads to an increase in thermal conductance and a corresponding increase in the metabolic rates of their avian hosts to maintain normal core body temperatures (Figure 29.3). Metabolic rates increase by an average of 8.5% and heavily infested birds need to draw on fat reserves to keep up with these energetic costs, leading to a chronic decline in body mass over several months (Booth et al. 1993). The end result, not surprisingly, is a significant drop in survival during the winter months (Clayton et al. 1999). The impact of feather lice on energetics may also be responsible for a significant drop in the rate of male courtship display, and thus the ability of heavily infested males to attract mates (Figure 29.3) (Clayton 1990).

Studies of several other bird species have also demonstrated reductions in the potential attractiveness of lousy males to females (Clayton 1991b). For example, Barn Swallows (*Hirundo rustica*) with high louse loads have songs of shorter duration than swallows with few lice (Garamszegi et al. 2005). Adult male Satin Bowerbirds (*Ptilonorhynchus violaceus*) with the most attractive bowers had low infestations of the louse *Myrsidea ptilinorhynchi* when they were juveniles (Borgia et al. 2004).



(a)



(b)

Figure 29.2. Damage to Rock Pigeon (*Columba livia*) feathers by feeding lice *(Columbicola columbae* and *Campanulotes compar)*. (a) Abdominal contour feathers with (left to right) no damage, average damage and severe damage. The basal downy region and barbules of the basal and medial regions have been consumed (reprinted with permission from the *American Zoologist* (Clayton 1990). (b) Magnified view of severe damage showing where barbules have been removed by lice. Neither the barbs, nor the rachis itself (=shaft) are damaged, probably because they are too large for the lice to sever with their mandibles. Reprinted with permission from Oxford University Press (Clayton 1991a).

Feather damage from chewing lice can have other consequences. The menoponid louse, *Hirundoecus* (=*Machaerilaemus*) *malleus*, chews holes in the tail feathers of Barn Swallows (Kose et al. 1999). These holes may increase feather breakage as well as permeability of the feathers to air, thus altering aerodynamic efficiency (Bonser 2001). Barn Swallows with many holes flap more frequently than other swallows (Barbosa et al. 2002), presumably incurring an energetic cost. The holes also increase the potential

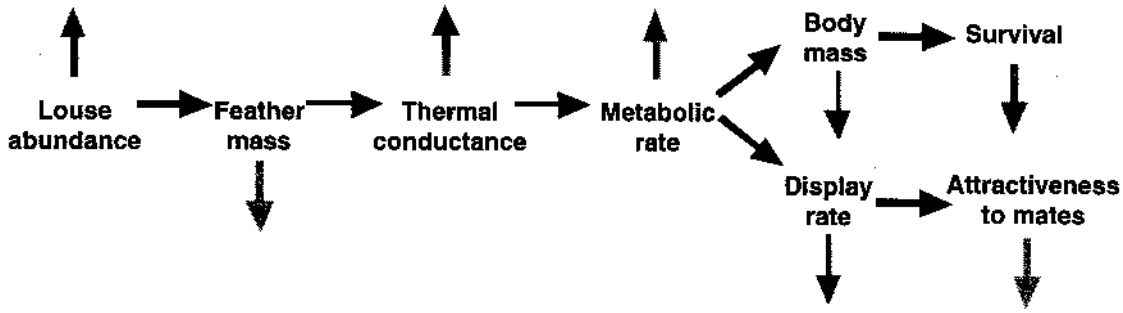


Figure 29.3. Consequences of experimentally increasing the abundance of chewing lice on Rock Pigeons (*Columba livia*) (combined results of Clayton 1990; Booth et al. 1993; and Clayton et al. 1999). See the text for discussion.

costliness of long tails, which appear to function as sexually selected "handicaps" signaling freedom from parasites (Kose et al. 1999). In another study, Cliff Swallows (*Petrochelidon pyrrhonota*) infested with lice, fleas, and bugs had significantly lower long-term survivorship relative to fumigated, parasite-free controls (Brown et al. 1995), although it was not possible to assess what fraction, if any, of the survival effect could be attributed specifically to lice, fleas, or bugs.

The time and energy required for efficient grooming to control lice may also be costly. Both numbers of lice and their species richness can influence grooming rates and the amount of time devoted to these activities (Brown 1974; Cotgreave and Clayton 1994). More time devoted to grooming may mean less time available for other activities, such as foraging and defense of territories. Increased grooming can also reduce vigilance and increase the risk of being killed by a predator (Redpath 1988).

Chewing lice can also have indirect effects on the host by acting as vectors or intermediate hosts of other parasites (Table 29.2). For example, the amblyceran louse *Trinoton anserium* transmits the common heartworm, *Sarconema eurycera*, to swans when the louse takes a bloodmeal (Seegar et al. 1976; Cohen et al. 1991; Chapter 26), while ischnoceran lice that serve as intermediate hosts for other filarid nematodes transmit these worms when they are ingested during preening (Bartlett 1993). Viruses and bacteria have also been isolated from chewing lice (Table 29.2), but it is not clear whether lice play a role in their transmission. Saxena et al. (1985) provided a detailed review of chewing lice as intermediate hosts and vectors.

DIAGNOSIS

In principle, lice are easy to detect because their life cycle is restricted to the body of the host. In practice, however, some lice are small and can be difficult to see. Some species are also restricted to microhabitats that are difficult to examine, such as the interior of quill feathers. Even lice that are normally found on the surface of feathers can be hidden in the shafts of developing pin-feathers during molt (Moyer et al. 2002a).

Methods for collecting and quantifying lice and other avian ectoparasites were reviewed by Clayton and Walther (1997). The five most commonly used methods for quantifying lice include body washing and post-mortem ruffling for dead birds, and dust ruffling, visual examination, and the use of fumigation chambers for live birds (Clayton and Drown 2001). The authors provided a decision tree for choosing among these five methods.

Louse nits (eggs) are usually white and are sometimes easier to detect than lice themselves. Although they are small (usually $\leq$1 mm in length), unhatched eggs can be obvious because they glisten in reflected light and are often laid in clusters. Hatched eggs remain attached to the feathers and are grayish and flattened in appearance. Many species of lice deposit their eggs in regions that are relatively protected from host grooming, such as the gular region (throat), vent region, or between the barbs of feathers (Nelson and Murray 1971). Most species of lice attach their eggs to the base of feathers. Representative photographs and diagnostic drawings of eggs can be found in Balter (1968), Foster (1969), Nelson and Murray (1971), Marshall (1981), and Cohen et al. (1991).

HOST DEFENSE AND IMMUNITY

Birds combat lice using a variety of defenses. The simplest defense is to avoid infection. This may be the principal advantage birds gain from choosing louse-free individuals as mates (Clayton 1991b). The most important defense of infested birds against lice is preening.

Table 29.2. Parasites and pathogens isolated from bird lice.

Bird host	Louse	Parasite/pathogen	Source
Charadriiformes			
Marbled Godwit (*Limosa fedoa)*	*Actornithophilus limosae**	‡*Eulimdana wongae*	Bartlett (1993)
	Carduiceps clayae†	‡*Eulimdana wongae*‡	Bartlett (1993)
Charadriiformes			
Whimbrel (*Numenius phaeopus*)	*Austromenopon phaeopodis**	*Eulimdana bainae*‡	Bartlett (1993)
	Lunaceps numenii†	*Eulimdana bainae*‡	Bartlett (1993)
Apodiformes			
African Swift (*Apus barbatus)*	*Dennyus hirundinis**	*Filaria cypseli*‡	Dutton (1905)
Gruiformes			
American Coot (*Fulica americana*)	*Pseudomenopon pilosum**	*Pelecitus fulicaeatrae*‡	Bartlett and Anderson (1987)
Podicipediformes			
Red-necked Grebe (*Podiceps grisegena*)	*Pseudomenopon* sp.*	*Pelecitus fulicaeatrae*‡	Bartlett and Anderson (1987)
Anseriformes			
Mute Swan (*Cygnus olor)* Tundra swan (*Cygnus columbianus*)	*Trinoton anserinum**	*Sarconema eurycera*‡	Seegar et al. (1976); Cohen et al. (1991)
Galliformes			
Red Junglefowl (*Gallus gallus*)	*Eomenacanthus stramineus (= Menacanthus)**	*Escherichia coli*§	Derylo and Jarosz (1972)
		Eastern equine¶ Encephalitis	Howitt et al. (1948)
		Pasteurella multicoida§	Derylo (1970)
		Salmonella gallinarum§	Derylo (1975)
		Streptococcus equinus§	Derylo and Jarosz (1972)
	*Menopon gallinae**	*Escherichia coli*§	Derylo and Jarosz (1972)
		Ornithosis bedsoniae§ (=*Chlamydophila*)	Eddie et al. (1962)
		Pasteurella multocida§	Derylo (1970)
		Streptococcus equinus§	Derylo and Jarosz (1972)

*Amblycera.
†Ischnocera.
‡Helminth.
§Bacteria.
¶Virus.

Wild birds with bill deformities can have enormous louse populations because they are not able to preen efficiently (reviewed by Pomeroy 1962 and Clayton 1991a). Experimental manipulations of the bill confirm that efficient self-preening is critical for controlling louse populations (Brown 1972; Clayton et al. 2005). Similarly, natural "experiments" confirm that scratching with the feet is critical for controlling louse populations on regions that cannot be preened. Birds that cannot scratch because of leg injuries sometimes have large numbers of lice and nits on the head and neck, but not on regions that the bird can still preen (Clayton 1991a). Allopreening, in which one bird preens another, may also play a role in louse control, although this possibility has not been tested carefully. Other behaviors that may help control lice include dusting, sunning, anting, and "fumigation" of nests with aromatic green vegetation (Hart 1997). Additional research is needed to determine the importance of these behaviors in louse control.

Feather chemistry is also important in defense against lice. The feathers and skin of several species of birds in the genus *Pitohui* contain the neurotoxin found in the skin of poison dart frogs (Dumbacher et al. 1992). When given a choice between these feathers and nontoxic control feathers, lice have higher mortality and avoid feeding or resting on the toxic feathers (Dumbacher 1999). A more common feather compound, the pigment melanin, makes feathers more resistant to mechanical abrasion (Bonser 1995). There is some evidence that lice on Barn Swallows avoid feeding on heavily melanized feathers (Kose et al. 1999), although a diet rich in melanin had no effect on lice of Rock Pigeons (Bush et al. 2006).

The avian immune system also provides another probable defense against lice that feed on blood or living tissue (Wikel 1996). Some early studies reported inverse correlations between avian "immunocompetence" and size of louse populations (Saino et al. 1995; Eens et al. 2000; Blanco et al. 2001). A recent comparative study by Møller and Rózsa (2005) reported a positive correlation between the number of genera of amblyceran lice on different species of altricial birds, and the T-cell-mediated immune responsiveness of nestlings of those species. The authors argued that the positive correlation reflects greater specialization of lice on species with strong immune responses. In contrast, there was no correlation between ischnoceran lice and immune responsiveness by the same species. This result makes sense because feather-feeding lice should be invisible to the immune system. More recently, Whiteman et al. (2005) found an inverse correlation between natural antibody (NAb) levels and the abundance of amblyceran lice (*Colpocephalum turbinatum*) on Galapagos Hawks. In contrast (but in parallel to Møller and Rózsa's (2005) results), the hawks showed no correlation between immunity and ischnoceran lice (*Deegeriella regalis*).

Although the results of these studies are intriguing, they should be interpreted with caution. Assessing immunocompetence on the basis of simple assays of immune function is risky. A decline in one component of the immune system can be offset by up-regulation of other components (innate, cell-mediated, or humoral), or by up-regulation of other aspects of the same component (Salvante 2006; Owen and Clayton 2007). Future studies need to explore the relationship between integrated immune responsiveness and lice, preferably in an experimental context.

TREATMENT AND CONTROL

Jackson (1985) reviewed the use of pesticides for controlling ectoparasites on wild birds in nestboxes. The safest choice is probably pyrethrum dust or spray, a "fast knock-down, slow killing" insecticide with no side effects on birds or mammals. Pyrethrum is a biodegradable derivative of chrysanthemums that breaks down within hours or days in the environment. Its kill rate is not 100%, so most commercial products use a combination of pyrethrin, a derivative of pyrethrum, and the synergist piperonyl butoxide. A 1.0% concentration of this mixture kills lice effectively, with no side effects on host nestlings or adults (Clayton and Tompkins 1995).

PUBLIC HEALTH CONCERNS

Bird lice are of little concern to humans because they cannot survive or reproduce off the body of the avian host. Although some bird lice can bite when infested birds are handled, they do not transmit human pathogens. Arthropods from ledge nesting birds occasionally enter dwellings through ventilation ducts or windows and take blood meals from people; however, such reports usually involve nest mites, not lice.

DOMESTIC ANIMAL HEALTH CONCERNS

Lice are considered a relatively minor problem in modern poultry operations because they are relatively easy to control (Williams 1992). However, they can still be a major problem for poultry kept under traditional conditions, particularly when birds are crowded or in poor health. Arends (1997) and Price and Graham (1997) review the impact of lice on poultry and other domestic birds, and provide details concerning the control of lice on domestic birds. The host specificity of lice on wild birds means that they pose little threat to domesticated birds.

WILDLIFE POPULATION IMPACTS AND MANAGEMENT IMPLICATIONS

Wildlife managers should be aware that lice can have negative effects on wild birds under certain conditions (e.g., Samuel et al. 1982; Cohen et al. 1991). Although healthy hosts normally keep their louse populations in check, lice can quickly increase on debilitated hosts. This can lead to blood loss, feather damage, irritation, and possible transmission of endoparasites and pathogens (Table 29.2), with effects on individual hosts or entire breeding populations (Samuel et al. 1982). Overcrowding of birds should be avoided because it facilitates transmission of lice, with a subsequent increase in average louse load (Clayton 1991a). For this reason, highly social birds are probably more at risk than solitary birds. Increases in lice can be either a cause or consequence of poor host health, depending on the situation. Although direct effects of lice are usually correlated with the number of lice present, their

indirect effects as vectors of other pathogens may be density independent.

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30
Acariasis

Danny B. Pence

INTRODUCTION

Although thousands of species are recognized, and hundreds of these are parasitic on or in birds, only a few endo- and ectoparasitic mites are recognized as pathogens (Krantz 1978). Mite infections/infestations in wild birds range in clinical severity from nonpathogenic to life-threatening, causing diseases of the skin, feathers, subcutaneous tissues, or respiratory tract. Clinical conditions resulting from acariasis in birds include anemia from exsanguination; dermatitis; mange on the body, feet, and face; feather damage or loss; and granulomatous inflammation in the subcutaneous tissues and in the respiratory tract (Arends 1997). While they have been known for centuries as common parasites affecting individual birds, only recently have certain mites been recognized as the cause of epizootic disease in wild bird populations (Pence et al. 1999).

It is neither the intent nor purpose of this chapter to provide a listing of the many species, genera or even families of mites that occur on avian hosts. For such, the reader is referred to the many publications on taxonomy by W. Ateyo, J. Gaud, A. Fain, and many well-known acarologists specializing on specific taxa of mites (see Krantz 1978 for a partial listing). Herein, those few genera and species that cause epizootics in wild bird populations as well as those that evoke a significant pathogenic response resulting in clinical disease will be discussed. They may include some to all of the life history stages (eggs, larvae, protonymphs, deutonymphs including the heteromorphic hypopi, and adults) of representatives from the superorders Parasitiformes (order Mesostigmata) and Acariformes (orders Astigmata and Prostigmata).

SYNONYMS

Skin diseases are commonly called bird mange; scaley leg; leg mange; podoacariasis; tassel foot; bumble foot; and scaley face. Feather problems include mite-induced depluming; permeable plumage; and feather lengthening, blackening, soiling, and droop. Endoparasitic species may cause several conditions commonly known as cystic and nodular mite disease; air sacculitis; respiratory, tracheal, and nasal acariasis; and subcutaneous acariasis.

HOST RANGE AND DISTRIBUTION

Mites are parasitic on/in almost all bird species worldwide. The few pathogenic species are best known as parasites of domestic birds such as fowl, turkeys, and pigeons or in captive caged birds such as finches and psittiforms. Since they are usually discovered sporadically as isolated cases in wild birds (Arends 1997), there are only a few reports of epizootic acariasis in wild bird populations. An epizootic of epidermoptid mange was reported in the Laysan Albatross (*Phoebastria immutabilis*) from Midway Atoll (Gilardi et al. 2001). Epizootics of knemidocoptic podoacariasis have occurred in the American Robin (*Turdus migratorius*) in the central and eastern United States; Red-winged Blackbird (*Agelaius phoeniceus*), Common Grackle (*Quiscalus quiscula*), and Brown-headed Cowbird (*Molothrus ater*) in eastern Canada; Evening Grosbeak (*Coccothraustes vespertinus*) in the southwestern United States; Chaffinch (*Fringilla coelebs*) in England and Baltic Russia; Sedge Warbler (*Acrocephalus schoenobaenus*) and European Bee-eater (*Merops apiaster*) in Nigeria; and Eurasian Tree Sparrow (*Passer montanus*) in Hong Kong (Pence et al. 1999).

ETIOLOGY

The Epidermoptidae are ectoparasitic macroscopic species including *Epidermoptes bilobatus* and *Epidermoptes phasianus* that cause head mange in domestic fowl and in Ring-necked Pheasants (*Phasianus colchicus*) (Fain 1965). A related species, *Myialges nudus*, is responsible for epizootic epidermoptid mange in Laysan Albatross fledglings (Gilardi et al. 2001). Chigger (Thrombiculidae) dermatitis caused by

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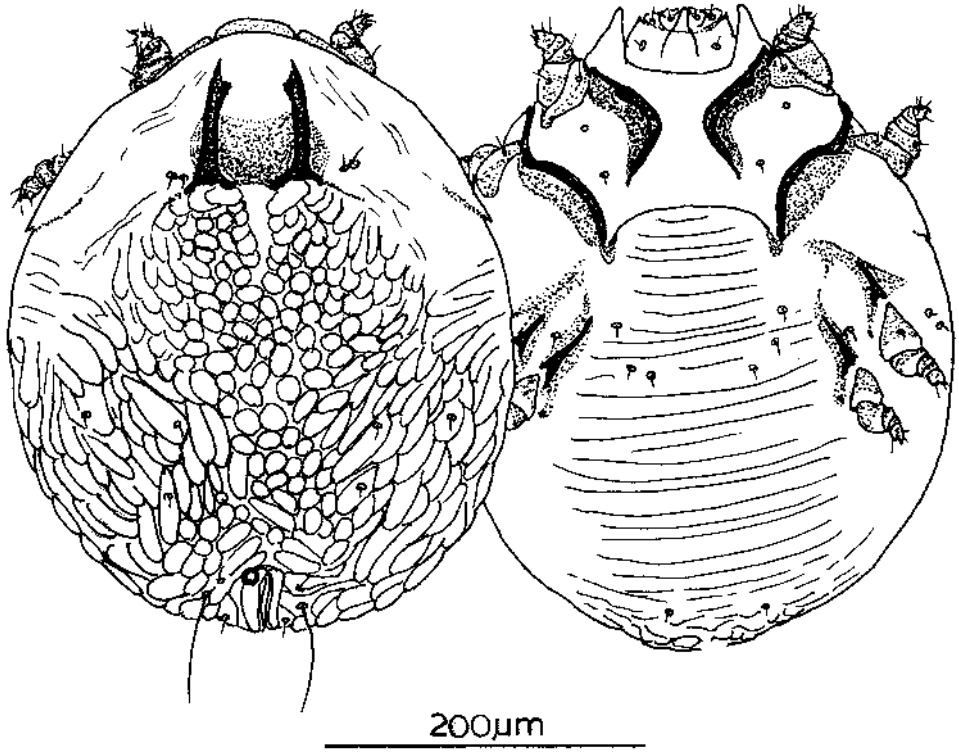


Figure 30.1. Dorsal (left) and ventral (right) views of adult female *Knemidokoptes jamaicensis* (original).

Wormersia midwayensis also has been reported in this species (Sileo et al. 1990).

The Knemidokoptidae are submacroscopic endoparasitic mites in the skin of birds. *Knemidokoptes jamaicensis* (Figure 30.1) is responsible for isolated cases of scaley leg in many species of mostly passerine birds and for epizootic podoacariasis in several species worldwide (Pence et al. 1999). The lethal head and body mange produced by *Knemidokoptes pilae* is a problem in captive budgeriegers, parrots, and macaws, and, while it occurs in wild psitticines, epizootics have not been reported (Fain and Elsen 1967). *Knemidokoptes derooi* causes mouth mange in the African Palm-Swift (*Cypsiurus parvus*).

The depluming mite, *Neoknemidokoptes laevis*, is sometimes a problem in domestic poultry and captive pheasants but has not been reported as a problem in free-ranging pheasants or the Wild Turkey (*Meleagris gallopavo*) (Arends 1997).

Species in the family Harpyrhynchidae are reported as deplumbing and itch mites of captive pigeons, canaries, and finches, but the consequences of the many species infesting wild birds is unknown. The same is true for the many species of quill mites (Syringophillidae) in wild birds that cause feather loss in domestic fowl, pigeons, and canaries. Feather mites (Cheyletiellidae, Analgesidae, and Dermoglyphidae) cause pruritis and feather damage in caged birds (Krantz 1978), but their effect on wild birds is unknown.

Of the endoparasitic mites, the cyst mite, *Laminosioptes cysticola* (Laminosioptidae) (Figure 30.2), is well known as the causative agent for fatal fowl nodular disease of the subcutaneous tissues in domestic fowl, turkeys, pheasants, geese, and pigeons (Arends 1997). Although known to occur in wild turkeys, its impact on these hosts is unknown.

Most species of birds are infected with host family-, genera-, or sometimes species-specific nasal mites of the families Rhinonyssidae, Speleognathidae, and/or Turbinoptidae (Fain 1956, 1957, 1963; Pence 1975). *Rhinonyssus rhinolethrum* is found commonly in ducks and geese. Like most other species of nasal mites, it appears not to evoke serious pathogenic lesions in its hosts. An important exception is the sometimes fatal nasal and respiratory tract rhinonyssid, *Sternostoma trachaecolum* (Figure 30.3), of caged birds such as canaries and finches (Fain and Hyland 1962). This mite also occurs in many wild birds

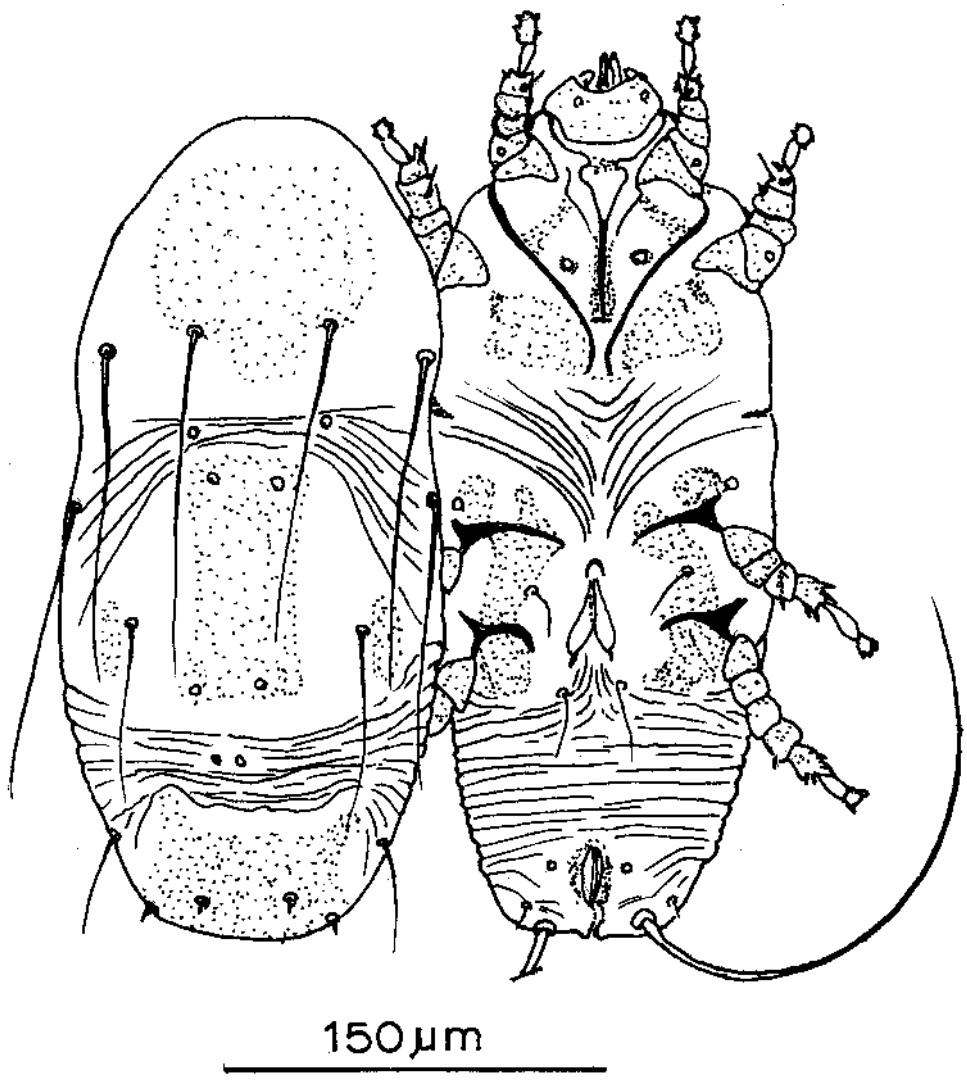


Figure 30.2. Dorsal (left) and ventral (right) views of adult female *Laminosioptes cysticola* (redrawn after Fain 1981).

worldwide and is very common in certain species such as the Great Crested Flycatcher (*Myiarchus crinitus*) in the southeastern United States. However, its effect on infected wild birds remains unknown. Another, sometimes pathogenic, air sac mite in domestic birds is the tiny *Kitodites* (=*Cytodites*) *nudus* (Figure 30.4) found in chickens, turkeys, pheasants, pigeons, canaries, and Ruffed Grouse (*Bonasa umbellus*) (Arends 1997).

Most nonpasserine orders of birds are infected with subcutaneous heteromorphic deutonymphs (hypopi) of hypoderatid mites (Hypoderatidae) of several genera, including *Phalacrodectes, Neottialges*, and *Hypodectes* (Figure 30.5) (Fain 1967). They are especially common in the subcutaneous adipose tissue beneath the skin in the axillary and groin area of most birds.

EPIZOOTIOLOGY

All birds, domestic or wild, are infested with any number of species of mites from several families occupying almost every available niche of the skin and feathers. Some of these may be fairly host specific to the bird genus or family level while other species of mites may infest many different unrelated bird hosts. Thus, wild birds may sometimes be important in the transmission of mites to domestic species. For example, the wild

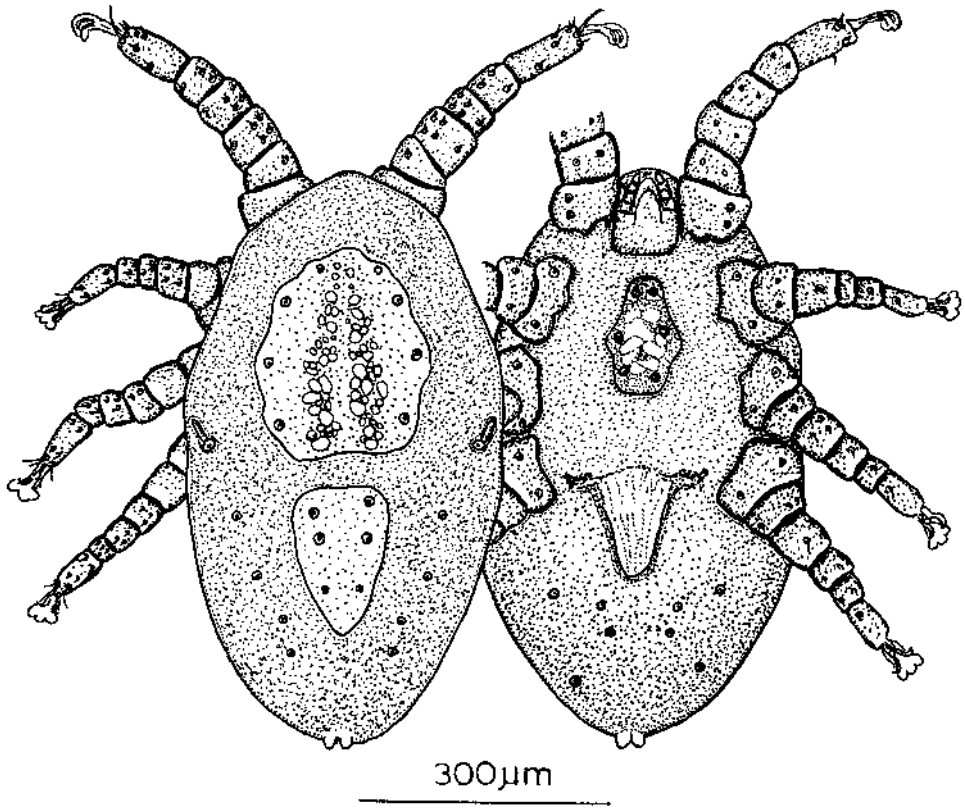


Figure 30.3. Dorsal (left) and ventral (right) views of adult female *Sternostoma trachaecolum* (original).

House Sparrow (*Passer domesticus*) appears to be the reservoir for several dermanyssid poultry mites worldwide, because of the practice of lining their nests with chicken feathers (Arends 1997). Certainly, the role of wild passerines is well known in their "seeding" of the environment with larval chigger mites (Thrombiculidae) that subsequently infest humans as well as other domestic animals and birds (Krantz 1978).

All the life history stages including eggs, larvae, nymphs, and adults of most avian acari can be found on/in the same host individual. Transmission between hosts may be by direct contact or at nest sites by actively motile larvae, nymphs, or adults (Krantz 1978). The important exceptions are the Thrombiculidae, Speleognathidae, and Hypoderidae that have parasitic larval and nymphal stages, but free-living adults (Fain 1967). The quiescent hypoderid heteromorphic deutonymphs localized in the subcutaneous fat of many birds become activated at the time of nesting, metamorphose to adults and exit through the skin of adult birds. The submacroscopic adult mites live and mate in the nesting material, the females produce eggs that hatch into larvae, and the microscopic larvae penetrate the intact skin of the nestlings. The larvae localize in the adipose tissues as heteromorphic deutonymphs that slowly mature over the next year to sixth-stage hypopi.

PATHOGENESIS, PATHOLOGY, AND CLINICAL CORRELATIONS

Similar to psoroptid mites in mammals, epidermoptid mites burrow and tunnel through the cornified layers of the skin of birds. By mechanical irritation and probably through release of excretory and secretory products, including keratinases, the mites can evoke severe mange in infected birds. Epidermoptids such as *M. nudus* in Laysan Albatross fledglings can cause granulomatous inflammation, with epithelioid histiocytes as the dominant cell type, accompanied by diffuse hyperkeratosis, multifocal dermal edema, and cellular infiltrates of heterophils with scattered eosinophils and lymphocytes in the dermis of infected birds (Gilardi et al. 2001). In occasionally fatal cases, these lesions may progress to more severe granulomatous inflammation

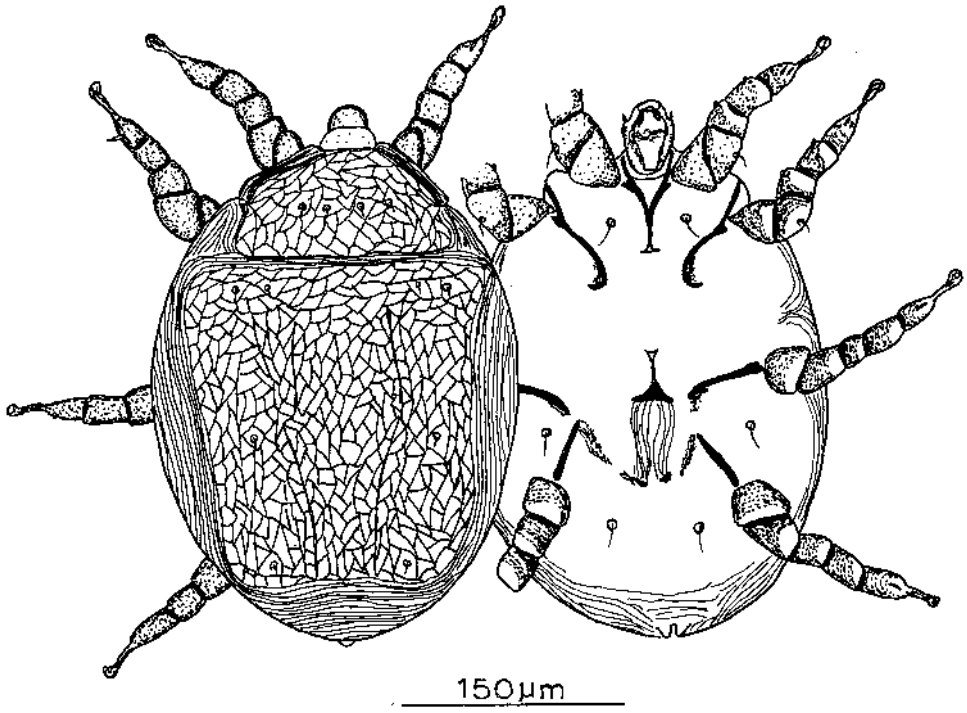


Figure 30.4. Dorsal (left) and ventral (right) views of adult female *Kytodites nudus* (original).

with Langhans' giant cells replacing large areas of the superficial dermis and obscuring the dermoepithelial junction. Mites can sometimes be observed within dilated feather follicles. Despite the severe granulomatous dermatitis, superficially the skin and feathers of infected birds appear normal (Gilardi et al. 2001).

The release of proteolytic enzymes, formation of a feeding stylostome, and subsequent ingestion of liquified tissue are a well-known mechanism of pathogenicity for the dermatitis caused by chiggers (Krantz 1978). There is a density-dependent relationship between the degree of infestation and the severity of the disease in the host. Fatal chigger dermatitis in Laysan Albatross fledglings presents as a severe necrotizing dermatitis. The effects of trombidiosis in this host ranged from mild dermatitis and edema to massive edema, focal hemorrhaging of the subcutaneous tissues of the crura and abdomen, and death with carcasses demonstrating ketosis, generalized pallor, and severe anemia (Sileo et al. 1990).

The pathogenesis of the knemidokoptids in birds resembles that of sarcoptid mites in mammals. Depending on the species, they have a predilection for the skin of the legs and feet and/or face and beak area where they burrow to the level of the stratum germativium or on the wings and thigh where they burrow into the feather follicles. Mechanical trauma from burrowing as well as excretory and secretory products released from the mites in situ contributes to the skin changes. The lesions of epizootic podoknemidokoptiasis or scaley leg caused by *K. jamaicensis* range from a white powdery scaling to proliferative epidermal overgrowth with massive crusts and scab formation resulting from massive hyperkeratosis and intense dermal inflammation on the unfeathered part of the legs and feet. Grossly, the skin may become markedly thickened and very rough, gray-white in color, desiccated, and fractured (Figure 30.6); under a stereo microscope there are numerous 0.1 mm black orifices on the surface of the skin (Pence 1975). There may be severe diffuse hyperkeratosis sometimes with sloughing of the nails, loss of digits, or traumatic amputation of the entire foot in severe cases (Pence et al. 1999). The abundant submacroscopic mites are often arrayed in a striking honeycombed pattern within the cornified epithelium (Figure 30.7), with scattered aggregates of eosinophils, lymphocytes, and histiocytes in the epidermal scale and in the underlying dermis (Pence 1970). Podoacariasis caused by *K. pilae* can occur in conjunction with scaley-face mange at the base of the beak and around the eyes and nares in captive, and probably in wild, psitticines (Fain and Elsen 1967). Lesions are similar



Figure 30.5. Dorsal (left) and ventral (right) views of two hypopi of *Hypodectes nudus* embedded in the axillary fat of a Mariana Fruit-Dove (*Ptilinopus roseicapilla*). Bar = 250 μm.



Figure 30.6. Excised leg of an American Robin (*Turdus migratorius*) showing the extensive hyperkeratosis of scaley leg from an infection with *Knemidokoptes jamaicensis*.

to those caused by *K. jamaicensis* in passerines, especially the small black orifices on the skin surface and the honeycombed pattern apparent on removing the superficial stratum corneum. Lesions may also be found on the body around the cloacal vent, on the wings, and on the thighs. As in infections with the depluming mite, *Neoknemidokoptes laevi,* there may be loss of feathers in and around the head and body lesions. Proliferation of keratinaceous overgrowths sometimes results in beak deformities including elongation, flattening, thinning, crossing, clefting, and/or splitting. In cases of depluming caused by knemidokoptids, the mites burrow into the basal shafts of the feathers on the epidermis leaving a well-defined orifice in the feather shaft. They produce the same white powdery material seen on the legs and face which may partly fill the feather follicle, evoke an intense irritation of the shaft and follicle, and cause the bird to eventually pull out the infected feather (Fain and Elsen 1967; Arends 1997). Slight thickening of the surface epithelium is noted in the lateral aspects of the internal surface of the mouth near the commissures of the bill in the African Palm-Swift infected with *K. derooi* (Fain 1970). Beak lesions and sometimes leg lesions caused by *K. fossor* are described by Fain and Elsen (1967) from several captive passerines in the Antwerp Zoo in Belgium.

The fowl cyst mite, *L. cysticola*, is found inside subcutaneous yellowish nodular granulomas of several centimeters diameter (Arends 1997). These transform into caseocalcareous deposits surrounding dead mites and are responsible for the condemnation of carcasses of heavily infected domestic poultry. The consensus of most (Kettle 1990; Arends 1997) is that the inflammation and destruction of muscle fibers cause loss of condition, emaciation, and even death of some heavily infected birds. Others contend that even massive infections of these mites are of little consequence to infected birds (Urquhart et al. 1996). Certainly, dead mites are known to evoke nodular subcutaneous granulomas, with larger numbers of nodules found in older birds. Similar lesions in the lungs are reported to cause

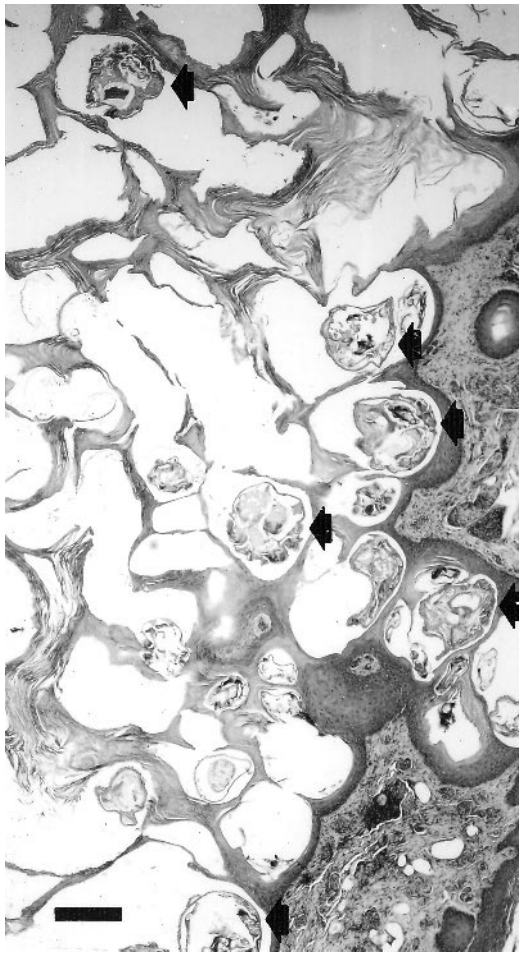


Figure 30.7. Low power magnification of a histologic section of the dermoepithelial junction of the foreleg of an Americal Robin (*Turdus migratorius*) with scaley leg from infection with *Knemidokoptes jamaicensis*. Note the honeycombed pattern in the epidermis containing numerous mites (arrows). H&E. Bar = 200 μm.

death in pigeons (Arends 1997). The effects of infection with any of the several species of laminosioptids that occur in wild birds are unknown (Fain 1981).

Tarsal claws are used to perforate the nasal mucosa by rhinonyssid mites such as *R. rhinolethrum* in the nasal passages of waterfowl; this allows them to feed on tissue and blood (Feider and Mironescu 1972). Mechanical irritation evokes hyperplasia and chronic inflammation of the nasal and respiratory mucosa produces an abundance of mucous and causes dilatation of the blood vessels which become engorged with erythrocytes (Feider and Mironescu 1972). So, heavy infestations of even those species of rhinonyssids strictly confined to the nasal passages may not always be inconsequential. Here, an edematous and bloody mucosa throughout the nasal sinuses is occasionally observed (D. B. Pence, unpublished observations). In captive canaries and goldfinches heavy infections of the nasal, tracheal, and air sac rhinonyssid, *S. trachaecolum,* may cause severe inflammatory tracheitis and airsacculitis leading to a fatal pneumonia (Fain and Hyland 1962). Although many species are reported as hosts of *S. trachaecolum* (Fain and Hyland 1962; Pence 1975), clinical disease has not been seen in wild free-living birds. It has been suggested that wild birds are more often parasitized in the nasal passages than in the lower respiratory tract and that they are refractory to the more serious stress-induced clinical conditions seen in their captive counterparts (Fain and Hyland 1962). However, it should be remembered that any heavily infected wild bird that is seriously ill with pneumonia undoubtedly would be very quickly removed from the population rendering such conditions in wild birds extremely difficult to determine.

The pathogenesis of the cytoditid air sac mites in domestic poultry, pheasants, pigeons, and Ruffed Grouse is density-dependent. Only heavy infections of *K. nudus* can cause weakness, emaciation, pneumonitis, and/or peritonitis (Arends 1997). Fatal granulomatous pneumonia results from obstruction of air passages from chronic inflammation and accumulation of mucous in the air sacs (Lindt and Kutzer 1965). Clinical signs resemble those of tuberculosis with wheezing, coughing, and weight loss in infected birds. The ramifications of this infection in Ruffed Grouse and other wild birds are unknown. Additionally, there are several other species of *Kitodites* and the related genus *Cytonyssus* that infect wild birds (Fain and Bafort 1964); clinical disease has not been reported.

Hypopi of the Hypoderatid mites appear as submacroscopic whitish to yellowish cylindrical bodies in the subcutaneous adipose tissue (Figure 30.5). They evoke a very mild to sometimes granulomatous inflammatory response in the subcutaneous tissues, especially in the dermal adipose tissue of many birds (Grünberg and Kutzer 1962; Hendrix et al. 1987; D. B. Pence, unpublished data). Having no mouth or digestive tract, it has been proposed that nutrient transport is through the exoskeleton of the hypopi or perhaps through the openings at the genital plate (Fain 1967). The somatic cells of these hypopi contain large quantities of the same neutral lipids that occur in the adjacent host adipose tissues (D. B. Pence, unpublished data). The inflammatory response to hypoderatids in

the subcutaneous adipose tissue ranges from a few foamy macrophages and plasma cells in the vicinity of the living hypopi in some hosts (Hendrix et al. 1987) to a diffuse granulomatous inflammation in many heavily infected birds (D. B. Pence, unpublished data). The latter is characterized as having only a few lymphocytes, eosinophils, and fusiform fibroblasts and with mostly epithelioid histiocytes as the predominant cell circumferential to the living hypopus, but without an attendant organized fibroplasia surrounding the lesion (D. B. Pence, unpublished data; Schwan and Sileo 1978). Severe granulomatous inflammation with a peripheral ring of Langhans' giant cells surrounding masses of epithelioid histiocytes, lymphocytes, plasma cells, and eosinophils is sometimes seen adjacent to degenerating hypopi in the subcutaneous tissues (D. B. Pence, unpublished data). There are usually no clinical signs of disease associated with infections of the subcutaneous hypopi of hyperderatid mites. They are usually discovered, sometimes in large numbers, at necropsy of birds having died of other causes. Hypopi are found most commonly in the axillary and groin fat, but sometimes they are seen in the deeper subcutaneous tissues of the esophagus and trachea, pericardial sac, abdominal cavity, and between the fascia of the large thigh muscles.

DIAGNOSIS

Because of their submacroscopic to microscopic size and predilection for unusual sites in the host, many kinds of acariases are difficult to diagnose, especially the species of endoparasitic mites. Deep skin scrapings examined in 10% KOH are very helpful in cases of epidermoptid and knemidokoptid mange. The knemidokoptids appear as tiny white immobile spheres. Examination of feather shafts under a stereo microscope is useful in diagnoses of syringophilid quill mites and depluming knemidokoptids. Ectoparasitic mites can be dislodged from the skin and feathers by brushing, especially around the head and neck with a stiff bristled brush such as an old toothbrush. The residue from these brushings should be examined with a stereo microscope. Laminosioptids are most often diagnosed by finding dead mites or their remains in the excised yellowish subcutaneous nodules crushed under a cover glass in a drop of acidulated water and examined microscopically. All rhinonyssid nasal and respiratory mites are macroscopic and usually darkly pigmented such that they are difficult to see in the anterior nasal epithelium, but they are sharply contrasted against the posterior turbinates and the nasal and respiratory mucosa. They are usually discovered at necropsy. The smaller cytoditids are more difficult to diagnose, but close inspection at necropsy may reveal white dots moving slowly over the surface of the air sacs. Hypopi usually appear at necropsy as scattered to very numerous immobile submacroscopic cylindrical whitish cystlike structures in the subcutaneous tissues, especially in the adipose tissue of the medial and lateral aspects of the thighs and in the inguineal region. Examination under a stereo microscope will reveal the characteristic deeply pigmented chitinized substructure (apodemes) of the anterior and posterior pairs of legs. Mites collected from their hosts can be fixed and stored indefinitely in 70% ethyl alcohol. Species can be identified only after light microscopic study of cleared specimens mounted in Hoyer's medium. At this point, species can usually be identified by examination of their many varied morphological features.

IMMUNITY

There have been no studies on the immune response of wild or domestic birds to acariases.

PUBLIC HEALTH CONCERNS

Dermanyssid mites that occur on domestic birds in poultry facilities can sometimes be carried by wild birds, especially House Sparrows. They can cause transient dermatitis in humans exposed to them. Also, many passerines worldwide are important in spreading chiggers (larval trombiculids) that are human pests that cause chigger dermatitis and transmit the rickettsiosis, tsutsugamushi disease (scrub typhus) caused by *Orientia tsutsugamushi* in Southeast Asia and the Indo Pacific. Otherwise, there are no public health problems associated with acariasis in wild birds.

DOMESTIC ANIMAL HEALTH CONCERNS

Host specificity varies dramatically across different species, genera, and families of mites. Thus, while cross transmission of many mite species is possible between their wild and domestic bird hosts, just how frequently this occurs is unknown. Certainly, the House Sparrow and possibly the European Starling (*Sturnus vulgaris*) and Rock Pigeon (*Columba livia*) have been responsible for the introduction of any of several dermanyssids such as the chicken mite (*Dermanyssus gallinae*), northern fowl mite (*Ornithonyssus sylviarum*), and tropical fowl mite (*Ornithonyssue bursa*) into domestic poultry flocks (Arends 1997). Recently captured wild passerines should be isolated for some time prior to mixing with long-term captive residents in a facility in order to avoid establishing knemidokoptid and other highly contagious mite infections or infestations.

WILDLIFE POPULATION IMPACTS

Epizootic knemidokoptic mange has been reported in numerous passerine populations in North America, Europe, Asia, and Africa (Pence et al. 1999). Many individuals may be lost from the population during such epizootics, but it is unknown whether or not the impact of this disease on these avian populations is compensatory with other mortality factors. The same is true for the impact of epizootic epidermoptid and trombiculid dermatitis in Laysan Albatross populations. And, while heavy infections of many other species of endoparasitic mites (tracheal and respiratory mites), subcutaneous hypopi, and massive infestations of ectoparasitic species (skin and feather mites) may extract their toll from decreased fitness and perhaps reproductive potential for individual birds, their importance to the population dynamics of their host species is unknown. So, while mites are sometimes annoying to the individual bird, and they are occasionally responsible for mortality events, it is difficult to envision any significant population impact that mites could induce on any managed game bird species. Thus, the need for any kind of management intervention in wild bird populations is questionable.

TREATMENT AND CONTROL

Permethrin spray may be used to treat for dermanyssids on transport cages or in resident facilities holding captive wild birds. Ivermectin has been used to treat wild birds with knemidokoptic podoacariasis, but it was not always successful as a prophylactic (Pence et al. 1999).

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31
Black Flies (Diptera: Simuliidae)

Douglas C. Currie and D. Bruce Hunter

INTRODUCTION

Black flies are a relatively small family of nematocerous Diptera with approximately 2,000 described species. These are distributed worldwide, particularly in areas where there are fast-flowing streams that serve as habitat for immature stages. The adults are small (1.2–6.0 mm long) hunch-backed flies with cigar-shaped antennae and broad wings. Simuliids are best known for the biting habits of adult females, which typically require a blood meal to develop their eggs. They rank among the world's most notorious pests for people and livestock, particularly in northern temperate regions. Their painful bites may incite local and/or systemic allergic reactions and can cause significant economic losses when outdoor business and recreational activities are affected. Black flies have caused serious problems in cattle raising areas in Canada and Central/Eastern Europe by harassing cattle, altering their behavior, reducing productivity, and even causing death. Black flies also act as vectors for a number of important pathogens of people, livestock, and birds, including arboviruses such as vesicular stomatitis in cattle, *Onchocerca* spp. in people and cattle, and *Leucocytozoon* spp. and *Trypanosoma* spp. in birds.

The role of black flies as the main vector of *Leucocytozoon* is well documented in wild, captive, and domestic birds (Chapter 4). There is also growing evidence that swarming, direct harassment, and blood feeding may contribute to increased nestling mortality, early fledging, and altered roosting behavior in several species of raptors and passerines.

SYNONYMS

Black fly (also black-fly and blackfly), buffalo gnat (USA), mouche noire (French-speaking Canada), sand fly (New Zealand and Australia), simuliid, reed smut, Kriebelmüke (German), pium (Brazil), borrachudo (southern Brazil), jejenes (Venezuela), bocones (Costa Rica), moshka (Russia), potu (India), mawi (Africa).

HISTORY

The earliest incontrovertible simuliid fossils date to the late Jurassic (Kalugina 1991; Currie and Grimaldi 2000); however, the fossil record of related families suggests that black flies originated during even earlier times, perhaps suggesting a Pangean or effectively Pangean origin for the family. This early origin of simuliids raises questions about what hosts might have been available to females. Records of bloodsucking on invertebrates and cold-blooded vertebrates have not been confirmed, and it must be presumed that the earliest simuliids blood fed exclusively on homeothermic animals, as they do today. Given that the earliest birds appeared only during mid-Jurassic times and that mammals (although present since the Jurassic) did not begin their radiation until the Tertiary period, it seems likely that simuliids began their sanguinary habits on another, more abundant, group of organisms, namely, homeothermic dinosaurs. Of course, the distinction between birds and dinosaurs during Jurassic times is moot, given that Aves are phylogenetically extant dinosaurs. The earliest example of an ornithophilic black fly is *Archicnephia ornithoraptor*—an Upper Cretaceous-aged fossil preserved in New Jersey amber (Currie and Grimaldi 2000).

The role of black flies as vectors of *Leucocytozoon* spp. has been known since the early 1930s with the demonstration by O'Roke (1930) and Skidmore (1931) that black flies transmit *Leucocytozoon simondi* to ducks and *Leucocytozoon smithi* to turkeys. In spite of extensive work on *Leucocytozoon* in birds since then (Chapter 4), the pathogenicity of ornithophilic black flies themselves remains poorly documented. It is only relatively recently that attention has been drawn to direct effects of black fly feeding on nestling survival and on behavior of juvenile birds (Hill 1994; Hunter et al. 1997; Smith et al. 1998; Gaard 2001).

DISTRIBUTION

Simuliids can be found virtually everywhere there is freely flowing, unpolluted water—the habitat of the

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immature stages. Only Antarctica, certain archipelagos (e.g., Hawaii, Falkland Islands) and isolated desert islands are unpopulated by members of the family. Some geographical areas (e.g., South Asia, New Guinea) remain inadequately surveyed for black flies, and it is estimated that the actual number of simuliids will easily exceed 3,000 species (Currie and Adler 2008).

HOST RANGE

Black flies are known to take blood meals from a wide variety of avian hosts and it seems unlikely that any major group of birds is immune to attack. So far, hematophagy by ornithophilic simuliids has been confirmed for at least 13 orders of birds (Crosskey 1990). The degree of host specificity varies among simuliids, with some species exhibiting a narrow host preference while others are more eclectic. However, the host preferences of most species remain poorly studied. The most host-specific black fly known is *Simulium annulus* (Lundström), which has so far been confirmed to feed only on Common Loons (*Gavia immer*). Extracts from the uropygial gland of loons are believed to play an important role in attracting females of *S. annulus* to their host (Fallis and Smith 1964; Lowther and Wood 1964). More typically, simuliids have a range of hosts that fit a particular profile (e.g., "small mammals," "ungulates," "passeriform birds," and "galliform birds").

Most female black flies can be assigned to one of two broad categories of blood-feeding habit based on their host preference for mammals (mammalophily) or birds (ornithophily). No species of black flies confines its bloodsucking to humans, but mammalophilic species that include humans as hosts are called anthropophilic. Bloodsucking records on invertebrates and cold-blood vertebrates have not been confirmed. A small number of arctic- or mountain-adapted species have reduced mouthparts that are not capable of piercing the skin (autogenous). Such obligatorily autogenous species are able to develop their eggs exclusively from nutrients gathered during the larval stage.

Bloodsucking (anautogenous) species of black flies are classified as ornithophilic or mammalophilic based on the form of the female tarsal claw (Shewell 1955). Ornithophilic species have an accessory tooth of variable size near the base of the claw, which presumably aids in grasping the feathers (Figure 31.1). In contrast, the claw of mammalophilic species is typically in the form of a simple talon, without a markedly developed subbasal tooth. Unfortunately, the distinction between the two feeding groups is not clear in all instances, with certain species taking blood from both birds and mammals regardless of claw structure. For example, the simple-clawed female of *Simulium venustum* is known to feed on various species of birds, although their numbers are low relative to well-established ornithophilic species with bifid claws (Bennett 1960). In contrast, *S. venustum* is recognized as among the most notorious pests of mammals in North America (Hunter et al. 1993; Adler et al. 2004). Despite such exceptions, a blood-meal study of black flies confirms the fidelity of claw type and host: species with bifid claws fed overwhelmingly on avian hosts, whereas species with simple claws fed predominantly on mammalian hosts (Malmqvist et al. 2004).

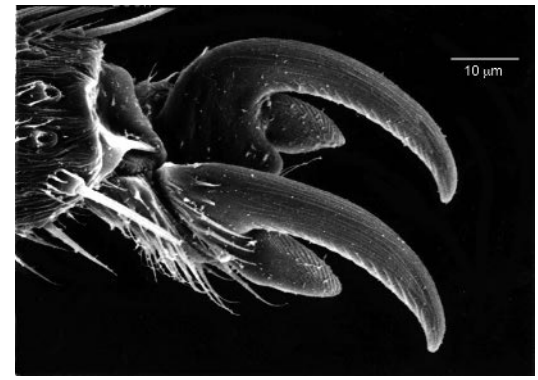


Figure 31.1. Scanning electron micrograph of the bifid claws typical of ornithophilic black flies. These claws are adaptations for grasping feathers. Courtesy of D. A. Craig, University of Alberta.

ETIOLOGY

The Simuliidae are most closely related to the Ceratopogonidae (biting midges), Chironomidae (nonbiting midges), and Thaumaleidae (solitary or trickle midges), which together comprise the nematoceran superfamily Chironomoidea. There are no comprehensive phylogenies of the Simuliidae, and different schemes of classification are favored by specialists from different geographical regions. Adler et al. (2004) provided a cladistic analysis of the Holarctic Simuliidae, which agreed largely with Moulton's (2000) suprageneric analysis of world black flies based on his analysis of molecular sequence data. Both studies recognize a two-subfamily arrangement of black flies, including the Parasimuliinae and the Simuliinae. The latter subfamily is further subdivided into two tribes: the Prosimuliini and the Simuliini. This two-subfamily and two-tribe arrangement has gained increased acceptance in recent years, although a more finely divided suprageneric classification continues to be favored by certain specialists. There is no comprehensive

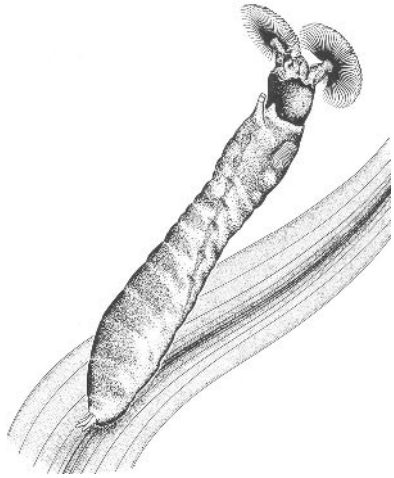


Figure 31.2. Larva of *Simulium vittatum*. Note the sausage-shaped body and labral feeding fans on head. From Currie (1986).

taxonomic treatment of the world Simuliidae, other than the world inventory of Crosskey and Howard (2004). We follow the generic classification of these authors for the purposes of this chapter.

EPIZOOTIOLOGY

Growth and Development

Like other true flies, black flies pass through four stages to complete their life history: egg, larva, pupa, adult. The first three stages are confined to running waters, which, depending on species, can range in size from tiny headwater trickles to large rivers. Eggs are deposited onto submerged or emergent substrata, or are simply dropped into the water, where they eventually settle into the bottom sediments. Larvae are roughly sausage-shaped creatures that bear a pair of rakelike appendages (labral fans) on the apex of their heads (Figure 31.2). When positioned into a current of water the fans intercept suspended particles as they pass by, providing the larva with food. Algae, bacteria, and even smaller particles, such as dissolved organic matter, are captured by the fans, which are alternatively folded and inserted into the mouth.

Larvae typically pass through a series of seven molts. Mature larvae spin a silken cocoon that varies in form from a shapeless sac to an elaborate shoe- or boot-shaped structure. The pupa more or less reflects the shape of the future adult, with the body regions and most of the appendages clearly visible through the pupal cuticle. A pair of spiracular gills arise from the anterolateral corners of the thorax and project anteriorly or anterodorally into the current (Figure 31.3). The gill, which aids in respiration, typically consists of a number of slender filaments, but in certain species the gill has a tubular or clublike form. Upon completion of a cocoon, the larva retreats inside and transforms into a pupa. Once fully developed, the adult emerges through a T-shaped slit in the pupal thorax and rises to the water surface in a bubble of gas. More detailed information about the life history of black flies can be found in Crosskey (1990).

The life span of ornithophilic black flies is not well established and likely varies among species. A study in Algonquin Park, Ontario, based on recapture of female flies (*Simulium rugglesi*) fed on ducks injected with a radioactive isotope showed that flies can live as long as 28 days, that is, sufficient time for the flies to accomplish two ovipositions (Bennet 1963).

Black fly eggs can undergo periods of dormancy (diapause) in order to survive periods of temperature extremes or drought conditions. Diapause is usually ended when environmental conditions become once again favorable for fly survival. Some northern species overwinter in the larval stage, but most overwinter as eggs and hatch when the water temperatures rise in the spring (Crosskey 1990). Water temperature and food supply regulate the rate of larval development. In northern climates usually not more than two generations of flies develop over a season, whereas further south there may be as many as four or five generations of flies.

Feeding Behavior

Many groups of black flies feed predominantly on birds and phylogenetic evidence suggests that ornithophily arose early in simuliid evolutionary history. It seems likely that simuliids first began blood feeding on the featherless progenitors of modern birds and that the specially modified tarsal claws of females arose shortly after the evolution of feathers. The subfamily Parasimuliinae is a relict group that currently consists only of autogenous (i.e., non-blood-feeding) species. In contrast, the subfamily Simuliinae has bird-feeding lineages in both the Prosimuliini and Simulini. Phylogenetic evidence is equivocal about whether ornithophily is in the ground plan of the subfamily Simuliinae or whether the habit evolved twice independently in the two tribes (Prosimuliini and Simullini). In either case, it is clear that ornithophily had an early origin in black flies, and is the predominant feeding habit among currently recognized genera.

The proportion of ornithophilic species among anautogenous black flies is difficult to assess because many regional faunas are inadequately studied. But in North America—where the simuliid fauna is most

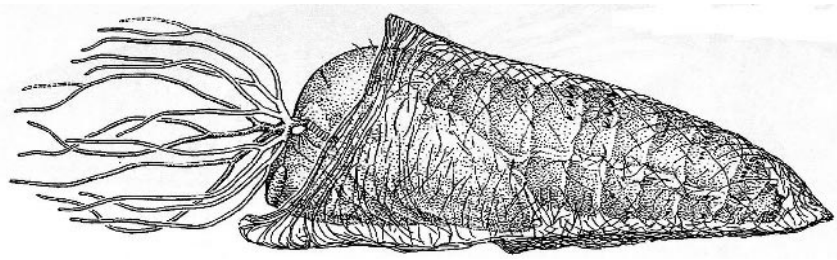


Figure 31.3. Pupa of *Simulium vittatum*. Note the silk cocoon and spiracular gills arising from the anterolateral corners of the thorax. These latter structures are important for respiration in the developing fly. From Peterson (1981).

completely known—approximately 37% of anautogenous black flies are ornithophilic compared to 63% that are mammalophilic (Adler et al. 2004). The incidence of ornithophily increases with latitude, with a proportion of 30% between 30 and 40° north and 46% between 50 and 60° north. The relative proportion of ornithophilic species at a world scale is impossible to evaluate because there are so few reliable host records outside of North America and Europe. Even in North America, about 60% of blood feeding species lack even a single confirmed host record (Adler et al. 2004). The apparent higher incidence of ornithophily at northern latitudes perhaps reflects a higher incidence of exclusively ornithophilic lineages in the Northern Hemisphere. In contrast, there are relatively few exclusively ornithophilic lineages in South America, and there are proportionally far fewer records of hematophagy on avian hosts (cf. Coscarón and Coscarón-Arias 2007). Whether this truly reflects a lower incidence of ornithophily in South America remains to be seen. Such a trend seems counterintuitive, given the relatively higher diversity of birds in tropical regions. Along similar lines, there are relatively few exclusively ornithophilic lineages of black flies in the Afrotropical and Australasian regions, and there are correspondingly few accounts about hematophagy on wild birds. This neglected aspect of simuliid biology requires much greater attention.

Bloodsucking (anautogenous) females locate their hosts using a combination of habitat features and host cues. Included among these latter are size, shape, color, odor (especially CO_2), body temperature, and phagostimulants, such as adenosines (Simmons 1985; Sutcliffe 1986, 1987; Allan et al. 1987). Visual stimuli and carbon dioxide are most important at long range, whereas odor and heat perception are thought to play a role at close range. Host products such as sweat are known to be attractive to female black flies (Schofield 1994).

At least two blood meals are needed for transmission of parasitic diseases—one to acquire the causative agent and another to transmit it. Anautogenous species that produce multiple egg batches, therefore, include the most important vectors of disease agents. Protozoa (*Leucocytozoon*, *Trypanosoma*), filarial nematodes (e.g., *Splendidofilaria fallisensis*), arboviruses (e.g., vesicular stomatitis), and possibly bacteria are transmitted to a wide variety of avian and mammalian hosts (Crosskey 1990; Adler et al. 2004; Adler 2005). The only known simuliid-borne pathogen of humans—the filarial worm *Onchocerca volvulus*, the cause of river blindness—is restricted to Africa and South and Central America.

In northern latitudes black fly activity is seasonal. Black flies emerge a few weeks after the ice leaves the streams and rivers in the spring and continues until the first frost in the fall. Peak black fly activity coincides with the nesting period for most northern bird species. Black flies are active during daylight hours in sheltered areas and particularly during cloudy periods. In open areas, they are most active in the early morning and evening shortly after sunset. Black fly activity increases at the approach of storms; however, rain and cold inhibit activity. Black flies cease flight activity when temperatures drop to 8–10°C and resume at 13–15°C (Crosskey 1990). The intensity of feeding (i.e., the prevalence, of hematophagous female black flies) is dependent on season, nest site location (proximity to open running water), when egg laying is initiated, and local weather conditions, including wind.

PATHOGENESIS

Black flies have bladelike mandibles that are used to pierce the skin and introduce salivary secretions into the wound. These salivary secretions cause vasodilation and promote blood flow to the wound, have anticoagulant activity to facilitate feeding, and modulate

components of the host immune system (Cupp and Cupp 1997). It takes between 3 and 6 min for females to fully gorge.

Salivary secretions have been studied in a number of mammalophilic black fly species. Saliva from *Simulium lineatum* and *Simulium equinum* contains histamine and several bioamines including putrescine, spermine, N1-monoacetyl-spermine, and spermidine (Wirtz 1990). Salivary apyrase and antithrombin salivary protein inhibit platelet aggregation and target components of the common pathway of the mammalian coagulation cascade (Cupp and Cupp 1997). Salivary gland extracts from *Simulium vittatum* modulate host immune responses by altering patterns of cytokine response (Cross et al. 1994).

Black fly bites result in an itchy raised bump or wheal, which may persist for up to 2 weeks, depending on the host. Repeated bites or large numbers of bites may lead to severe local or systemic allergic reactions in humans and other mammals.

Impact of Biting and Swarming on Birds

Black flies can have a negative impact on birds even without the complication of disease transmission. Blood feeding can disrupt feeding and nesting behaviors, resulting in death in extreme cases (Adler et al. 2004). Whether mortality is the result of anemia or toxemia (or a combination of these two factors) is poorly understood. Nonetheless, the deleterious effects of blood feeding by black flies are most pronounced in young birds.

The effects of bloodsucking on birds are best documented in the commercial poultry industry, where persistent attacks are known to result in inflammation of the skin, loss or reduction in appetite, egg abandonment, and death (Swenk and Mussehl 1928; Edgar 1953; Anderson and Voskuil 1963). Attacks on poultry are typically concentrated in the vicinity of the neck and head, especially around the eyes. Attacks have also been documented in the exotic bird industry in North America, with black flies implicated in the deaths of cockatoos and parrots (Mock and Adler 2002).

CLINICAL SIGNS AND PATHOLOGY

Chickens and turkeys become restless and distracted when harassed by large numbers of black flies crawling on their skin, under the wings, and biting around the eyes or on combs and wattles. Hens abandon nests, food intake decreases, and egg productivity drops. Young chicks and turkey poults may die rapidly from blood loss if infestations are intense (Crosskey 1990). Clinical pathology has not been documented, but it is likely that survivors develop a regenerative anemia with marked erythrocyte polychromasia.

Wild birds tormented by black flies also show behavioral changes. Nestling Red-tailed Hawks (*Buteo jamaicensis*) continuously exhibited annoyance behavior during infestations by continuously flapping their wings, vigorously shaking their heads, moving around in the nest, and pecking at exposed areas of their bodies (Smith et al. 1998). Occasional chicks found dead under nest sites had fractured bones but no other signs of disease, possibly the result of nestlings falling or jumping from the nests prematurely in an attempt to escape black fly harassment. Red-tailed Hawk nestlings in the Yukon, Canada, were restless and called incessantly (Doyle 2000). Similar behaviors were not observed in nestlings unaffected by black flies.

Rohner et al. (2000) documented that roost site selection in Great Horned Owls (*Bubo virginianus*) in the Kluane region of the Yukon, Canada, shifted from the winter and late spring to the summer months, coinciding with the emergence of black flies and black fly harassment. During the summer the owls roosted near ground level or on the ground in open areas compared to the traditional concealed mid-canopy locations used during the rest of year. Black fly activity was near zero in open areas at ground level and highest at mid-canopy level. The authors suggest that changes in roost site selection may be influenced by black fly avoidance behavior.

Black flies feed mostly on unfeathered areas such as the eyelids, cere, corners of the beak, ventral surface of the mandible, auricular openings, jugular groove, and ventral surfaces of the patagium (Figure 31.4) (Hunter et al. 1997; Smith et al. 1998). Eyelids may be edematous and swollen shut. Raised, inflamed erythematous areas surround small puncture wounds at black fly feeding sites and these areas are often scabbed over with dried blood (Figure 31.5). In heavy infestations adjacent feathers may be matted with blood. Microscopically, the skin lesions are characterized by increased vascularization, local edema, subcutaneous hemorrhage, local tissue necrosis, mixed inflammatory cell infiltration, and thrombosis of adjacent capillaries (Hunter et al. 1997; Smith et al. 1998). Allergic reactions, generalized toxemia, or simuliotoxicosis similar to that described in cattle and so-called black fly fever in people (headache, feverish shivering, nausea, swelling and tenderness of lymph glands, aching joints, and mental depression) have not been reported in birds.

DIAGNOSIS

Diagnosis is based on the presence of clinical signs or lesions typical of biting black flies and supported by finding the flies on or around the host. Black flies are



Figure 31.4. Common Loon (*Gavia immer*) harassed by black flies, likely *Simulium annulus* (Lundström), the most host-specific of ornithophilic black flies. Note the flies feeding along the unfeathered margins of the bill and cere. Courtesy of N. K. Dawe, Canadian Wildlife Service.

notorious for their structural homogeneity, and species-level identification often requires that specimens be examined by a specialist. It is not unusual for a single host to be attacked by more than one species of black fly.



Figure 31.5. Eyelid of a Great Horned Owl (*Bubo virginianus*) fledgling that has suffered extensive black fly bites to the eyelids and elsewhere. The eyelid is swollen and each dark crusted area is a black fly feeding site. Feathers near the margin of the eyelid are matted with dried blood. Courtesy of D. B. Hunter.

IMMUNITY

There is no literature documenting immunity or resistance to black fly bites in birds. The increased susceptibility of nestlings compared to adults is due to their small size and physical and physiological immaturity as well as their inability to escape harassment. Mortality in both Eastern Bluebirds (*Sialia sialis*) and Red-tailed Hawk nestlings was age related and decreased once protective feathering developed. In people, some individuals appear to have natural immunity and are bitten less frequently by black flies than are others. Biting rates have been shown to be partially dependent on interindividual variation in skin secretions and skin temperature (Schofield and Sutcliffe 1997) rather than host immunity. This phenomenon has not been studied in birds.

PUBLIC HEALTH CONCERNS

Mammalophilic and anthropophilic black flies cause many health concerns in people. These include being a major nuisance, causing localized dermatitis through bite wounds, inciting localized or systemic allergic reactions, and being the main vector for *Onchocerca volvulus*, the cause of human onchocerciasis or river blindness. Ornithophilic black flies pose no health hazards to humans.

DOMESTICATED ANIMAL HEALTH CONCERNS

Ornithophilic black flies have long been recognized as important pests for domestic poultry or other bird species that are raised outdoors in areas close to black fly habitats. The importance of black flies in commercial poultry production has decreased as poultry are less frequently raised in open range conditions, reducing exposure to black flies. Black flies still pose a threat to backyard poultry, organic and free range growing operations, pet birds housed in outdoor aviaries, and birds in zoos and private collections.

WILDLIFE POPULATION IMPACTS

In general, accounts about the effects of hematophagy and harassment are sparsely represented in the wild bird literature, perhaps because there is less impetus to investigate cause of death in wild (as opposed to domesticated) birds. The impact of black flies on wild birds is not well known; however, evidence suggests that it may be just as severe as that observed in the poultry and exotic bird industries. For example, attacks by members of the *S. annulus* species group are known to induce mortality in Red-tailed Hawk nestlings, either by causing anemia and dehydration or by driving

individuals from nests (Fitch et al. 1946; Smith et al. 1998; Doyle 2000). In a 4-year study (1992–1995) of Red-tailed Hawks nesting in the Kluane region of the Yukon, Canada, Doyle (2000) monitored black fly numbers at nests every 2–3 days. In these 4 years, whenever >70 flies were recorded on or around the young during a visit, and chicks were less than 20 days of age, all chicks had died by the next nest visit. Over all 4 years, the annual proportion of nest failures was significantly correlated with the mean number of flies seen per nest visit. The time of nest initiation was a factor and late breeders had a higher rate of nest failure. Smith et al. (1998) documented black fly infestations (*Simulium canonicolum*) at 42 Red-tailed Hawk nests in Wyoming. Black flies caused mortality at 6 of 42 (14%) nests where young hatched (13 of 87 nestlings) and were the only known cause of nestling mortality. The onset of infestations occurred when nestlings were 3–20 days old and usually lasted until nestlings died or fledged. Brown and Amadon (1968) reported that during wet years black fly feeding (*Prosimulium* sp.) was a significant cause of Red-tailed Hawk nestling mortality in California.

Females of *Simulium meridionale* are known to cause nestling mortality in Purple Martins (*Progne subis*), Mangrove Swallows (*Tachycineta albilinea*), and Eastern Bluebirds (Hill 1994; Gaard 2001, 2002, 2003). Affected birds are generally 6–10 days of age and feathering is incomplete. Finally, attacks by females of *Cnephia ornithophilia* may disrupt the feeding and reproductive behaviors of the critically endangered Attwater's Prairie-Chicken (*Tympanuchus cupido attwateri*), a subspecies of the Greater Prairie-Chicken (Adler et al. 2007). The vulnerability of birds, whether through the effects of blood feeding or leucocytozoonosis, is probably heightened during episodes of meager food supply (Hunter et al. 1997) and severe weather (Smith et al. 1998).

The impact of black flies on nestlings and reproductive success could be easily overlooked or missed if nests were not visited at critical times. Baby birds are impacted at an early natal down stage. Standard nesting surveys with two to three nest visits per nesting period could easily miss the impacts of black flies on nesting success. Harassment by black flies can be an important mortality factor in man-made nesting cavities for Eastern Bluebirds and Mangrove Swallows. These problems are generally local and related to the location of the nest boxes near running streams and influenced by weather conditions. Mortality is noticed because of the frequency of observation, but there is little information on black fly related mortality in natural nesting areas that are not monitored as rigorously.

Ornithophilic species of black flies can reduce the "fitness" of an avian host in a variety of ways: through direct blood loss and harassment during the nestling period; by the increased investment of energy in inflammation and repair and immunological defense; and by transmitting potential pathogens such as *Leucocytozoon* or *Trypanosoma*. Each of these factors has an energetic cost for the bird. The combination of black fly feeding and transmission of blood parasites may tip the scale toward mortality in times of stress, decreased food availability as demonstrated in Great Horned Owls in the Yukon (Rohner and Hunter 1996; Hunter et al. 1997), or perhaps during periods of inclement weather. The importance of black flies in reducing "fitness" and influencing other physiological processes deserves further study.

It is also interesting that harassment by black flies and the subsequent avoidance behavior by the host may result in other subtle effects and trade-offs. Great Horned Owls may put themselves at greater risk of predation by choosing roosting locations on the open ground, presumably in an effort to avoid black fly harassment. Perhaps nest site selection is influenced by a similar process. The role of black flies in the natural history of birds remains poorly understood.

TREATMENT, CONTROL, AND MANAGEMENT IMPLICATIONS

Many strategies have been developed for black fly control because of their importance as vectors of *Onchocerca volvulus* in West Africa. The World Health Organization Onchocerciasis Control Program (OCP), established in 1974, was based on the spray of insecticides by helicopters and aircraft over larval habitats (aerial larviciding). With the donation of Mectizan® (ivermectin) by Merck & Co., Inc., in 1987, control operations changed from exclusive use of vector control to a combination of vector control and treatment with ivermectin. In some areas, ivermectin alone was used to control infections (see World Health Organization Web site www.who.int/blindness/partnerships/onchocerciasis_OCP/en/index.html). The OCP was officially closed in December 2002 and considered to have been a highly successful program. Currently, most control programs against black flies are directed toward nuisance pests of humans.

It is unlikely that large-scale black fly control programs would be used or warranted in protecting wild birds. However, there may be circumstances where it should be considered, particularly when endangered species are at risk. Adler et al. (2007) suggested that if management of black flies became necessary to protect the endangered Attwater's Prairie-Chicken on the Attwater Prairie Chicken National Wildlife Refuge, Texas, USA, then control would include a thorough

evaluation of local streams and irrigation systems to first identify black fly breeding sites that could then be treated with an environmentally safe, simuliid-specific biopesticide such as *Bacillus thuringiensis israelensis* (Gray et al. 1996).

Measures to control access of black flies to man-made nest boxes for Eastern Bluebirds and Mangrove Swallows include blocking ventilation openings with cotton and duct tape and creating a darker interior to the nesting cavity to reduce black fly activity (Gaard 2002). Pesticides (pyrethrins) have been used within nesting cavities to reduce black fly numbers. These efforts are targeted toward nestlings that are around 6–10 days of age when they are extremely vulnerable to feeding by black flies.

Vector proofing aviaries in captive or breeding collections using fine mesh netting is another possible prevention method.

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32
Myiasis in Wild Birds

Susan E. Little

INTRODUCTION

Myiasis is the infestation of healthy or necrotic tissue of living vertebrate animals by dipteran larvae and is very common in birds, particularly in nestlings. The larvae of some species of diptera burrow into tissues, creating wounds, while larvae of other species feed on blood but remain on the surface of their avian hosts. The detrimental effects of fly larvae feeding on nestlings are not always readily apparent, and in many cases, may only become clinically evident when compounded by inadequate nutrition or other factors that cause stress. Myiasis is reported across terrestrial vertebrate taxa and may be associated with virtually any dipteran larvae, although cases are usually associated with infestation by blow flies (Calliphoridae), flesh flies (Sarcophagidae), or bot flies (Oestridae) (Catts and Mullen 2002).

Myiasis in wild birds is most frequently associated with infestations of hematophagous dipteran larvae from the families Calliphoridae, Muscidae, and Neottiophilidae (Uhazy and Arendt 1986; Spalding et al. 2002). Cutaneous and subcutaneous infestation of wild birds with dipteran larvae of other families also occurs (Zumpt 1965; Wobeser et al. 1981; Baumgartner 1988; Farkas et al. 2001). Infestation of birds by oestrid bot flies is rare (Artmann 1975; Roberts and Janovy 2005) and will only briefly be mentioned here. Although any warm-blooded animal is susceptible to infestation with larvae of *Cochliomyia hominivorax* and *Chrysomya bezziana*, the causes of obligatory myiasis, screwworm infestations are rarely reported on birds (Roberts and Janovy 2005).

A wealth of terminology is used to characterize the different types of myiases. Larvae may be referred to as obligatory if they require a living host as part of their life cycle, or facultative if they normally develop in decaying organic matter and only occasionally infest necrotic wounds on living animals. A third form, accidental or pseudomyiasis, describes cases where dipteran larvae are mistakenly ingested by an animal and pass through the gastrointestinal tract, but are not associated with any actual infestation (Catts and Mullen 2002).

Myiasis may also be described according to the tissue ingested or the body region affected (Zumpt 1965). The most common forms in wild birds are hematophagous myiasis, which occurs when larvae of obligatory myiatic species ingest blood of the infested host, and subcutaneous myiasis, in which the larvae burrow beneath the skin to feed on host tissues. The distinction is not absolute; the larvae of some hematophagous species remain on the surface of birds, while larvae of other species continue to burrow deeply into the host while still feeding on blood (Sabrosky et al. 1989). Traumatic myiasis is the secondary invasion of wounds by dipteran larvae and has occasionally been reported from wild birds (Zumpt 1965; Baumgartner 1988). Finally, larvae involved in myiasis may be referred to as primary, secondary, or tertiary depending on when they enter the host or the wound. Species that cause primary myiasis are able to initiate feeding on an otherwise healthy animal with intact skin. Species that cause secondary myiasis are facultative parasites that infest preexisting wounds, feeding on living tissue only once the supply of decaying, necrotic tissue on that animal has been depleted. Species that cause tertiary myiasis prefer carcasses and are only rarely seen on living animals very late in the course of disease (Kettle 1995).

SYNONYMS

Synonyms vary according to the type of myiasis being described. Hematophagous species typified by the *Protocalliphora* have been referred to as bird nest flies, bird blow flies, bloodsucking larval flies, parasitic bird flies, nestling "screwworms," and bird nest "screwworms" (Sabrosky et al. 1989). The hematophagous larvae of muscid flies, such as *Philornis* spp., are occasionally referred to as bots, although most authors reserve this term for individual oestrid larvae. Oestrid (=botfly) larvae and their cysts may also be called warbles or

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wolves, but true bot fly infestations are rare on avian hosts. Disease caused by species involved in traumatic or secondary myiasis may be called strike, fly strike, infestation with wound maggots, or blow, and affected animals are sometimes referred to as "blown" or "fly struck" (Catts and Mullen 2002).

HISTORY

Reports of dipteran larvae feeding on Barn Swallow (*Hirundo rustica*) nestlings can be found as early as 1845 in what is regarded as the first description of *Protocalliphora azurea* from France, and in another report of a likely *Protocalliphora* sp. described in 1866 from North America (Dufour 1845 and Walsh 1866 as reported by Sabrosky et al. 1989). Numerous subsequent descriptions of hematophagous and occasionally subcutaneous maggots that parasitize nestling birds have been made, most involving species of the genus *Protocalliphora*, *Philornis*, or *Passeromyia* (Table 32.1). The literature also contains sporadic reports of traumatic myiasis on birds, although this form of myiasis is considered relatively uncommon in wild birds (Wobeser et al. 1981; Baumgartner 1988; Farkas et al. 2001).

Despite the frequent descriptions of affected birds in the literature, myiasis has historically been considered of little consequence to otherwise healthy bird populations. Indeed, in a comprehensive description of North American *Protocalliphora* spp., Sabrosky et al. (1989) notes that most entomology and avian disease books do not mention or emphasize myiasis-inducing diptera on birds. However, the recent discovery that *Philornis downsi* is parasitizing nestlings of Darwin's finches in the Galapagos Islands has reignited interest in the importance of myiasis to the health of wild birds, particularly threatened species (Fessl et al. 2006a, b). Similarly intriguing is the recent finding that myiasis in nestlings increases in disturbed habitats (Gentes et al. 2007). Additional work will likely continue to reveal that in times of limited nutrients, habitat stress, and introduction of new fly species into fragile ecosystems, myiasis can have a significant impact on wild birds.

DISTRIBUTION

Avian myiasis is cosmopolitan in distribution, although the individual species involved may be restricted in their range, and localities often have a predominance of just a handful of important myiasis-inducing diptera (Sabrosky et al. 1989). Flies that produce hematophagous larvae which are obligate feeders on nestling birds include members of the families Calliphoridae (*Protocalliphora* spp.), Muscidae (*Philornis*, *Passeromyia*, *Mydaea*), and Neottiophilidae (*Neottiophilum*, *Actinoptera*) (Uhazy and Arendt 1986; Spalding et al. 2002). The *Protocalliphora* spp. are found in a Holarctic, predominantly northern distribution, extending in North America as far south as Nearctic Mexico, across Palearctic Europe, North Africa, and temperate Asia. Reports in the southern extent of this range are generally from higher altitudes (Sabrosky et al. 1989). These flies appear to be largely absent from the southeastern US (Sabrosky et al. 1989; Spalding et al. 2002). Excellent maps detailing the known range of Nearctic *Protocalliphora* spp. can be found in Sabrosky et al. (1989). The muscid genera that comprise the tropical nest flies also have a broad distribution but are usually found in warmer climates than *Protocalliphora* spp.; *Philornis* is reported from the New World tropics and *Passeromyia* and *Mydaea* occur in Australia and Asia (Pont 1974; Couri 1999). Nest skipper flies (*Neottiphilum* spp.) have a predominantly Palearctic distribution (Catts and Mullen 2002).

Other calliphorids that have been reported associated with wounds on wild birds include *Calliphora* and *Lucilia* spp. in Europe (Baumgartner 1988; Farkas et al. 2001). Reports of sarcophagid flesh flies associated with wound myiasis in birds are mostly confined to *Wohlfahrtia magnifica* in the Old World and *Wohlfahrtia opaca* in the New World (Zumpt 1965; Wobeser et al. 1981).

HOST RANGE

Nestlings of any nidocolous (=nest dwelling) bird may be subject to hematophagous myiasis. However, reports of hematophagous myiasis caused by *Protocalliphora*, *Philornis*, *Passeromyia*, and *Neottiophilum* are by far most common in the passerines (Table 32.1). Myiasis caused by species of *Protocalliphora* is also described from raptors and, occasionally, Piciformes (woodpeckers and allied species). Sporadic reports are found on Columbiformes and Cuculiformes. In a review of known hosts of *Protocalliphora* spp. in North America, 139 different species of birds were reported as potential hosts of *Protocalliphora* spp., but there were no documented reports of natural infestations on water or shore birds, Galliformes, Psittiformes, Caprimulgiformes, Apodiformes, Trogoniformes, or Coraciiformes.

Traumatic myiasis can be reported from any vertebrate, including any species of bird. Confusion regarding *antemortem* and *postmortem* arrival of the fly larvae found on a bird carcass, and the presence of maggots feeding on decaying organic matter in the nests but not the nestlings themselves, can lead to misinterpretation of the involvement of myiasis in a mortality event. Although obligatory and facultative traumatic myiasis has been frequently reported from domestic

Table 32.1. Examples of hematophagous dipteran larvae found on wild birds.

Family	Species	Host order	Example reference
Calliphoridae	*Protocalliphora aenea*	Passeriformes	Halstead (1988)
	Protocalliphora asiovora	Falconiformes	Sabrosky et al. (1989)
		Passeriformes	Whitworth and Bennett (1992)
	Protocalliphora avium	Falconiformes	Bortolotti (1985)
		Passeriformes	Pletsch (1948)
		Strigiformes	Tirrell (1978)
	Protocalliphora azurea	Coraciiformes	Sabrosky et al. (1989)
		Passeriformes	Simon et al. (2004)
	Protocalliphora beameri	Passeriformes	Sabrosky et al. (1989)
	Protocalliphora bennetti	Passeriformes	Whitworth (2003)
	Protocalliphora bicolor	Passeriformes	Sabrosky et al. (1989)
	Protocalliphora braueri	Falconiformes	Sabrosky et al. (1989)
		Passeriformes	Howe (1992)
	Protocalliphora brunneisquama	Passeriformes	Whitworth (2003)
	Protocalliphora chrysorrhoea	Passeriformes	Whitworth and Bennett (1992)
	Protocalliphora cuprina	Columbiformes	Sabrosky et al. (1989)
		Passeriformes	Boland et al. (1989)
	Protocalliphora deceptor	Passeriformes	Revels (1996)
	Protocalliphora falcozi	Passeriformes	Simon et al. (2004)
	Protocalliphora fallisi	Passeriformes	Sabrosky et al. (1989)
		Piciformes	Sabrosky et al. (1989)
	Protocalliphora halli	Passeriformes	Sabrosky et al. (1989)
	Protocalliphora hesperia	Passeriformes	Sabrosky et al. (1989)
	Protocalliphora hesperioides	Passeriformes	Sabrosky et al. (1989)
	Protocalliphora hirundo	Passeriformes	Shields and Crook (1987)
	Protocalliphora interrupta	Passeriformes	Sabrosky et al. (1989)
	Protocalliphora isochroa	Passeriformes	Sabrosky et al. (1989)
	Protocalliphora lata	Falconiformes	Sabrosky et al. (1989)
		Passeriformes	Sabrosky et al. (1989)
		Piciformes	Wiebe and Swift (2001)
	Protocalliphora lindneri	Passeriformes	Sabrosky et al. (1989)
	Protocalliphora metallica	Passeriformes	Revels (1996)
	Protocalliphora occidentalis	Passeriformes	Whitworth (2003)
	Protocalliphora parorum	Passeriformes	Johnson and Albrecht (1993)
	Protocalliphora peusi	Passeriformes	Sabrosky et al. (1989)
	Protocalliphora rugosa	Passeriformes	Whitworth (2003)
	Protocalliphora seminuda	Passeriformes	Sabrosky et al. (1989)
	Protocalliphora shannoni	Passeriformes	Dawson et al. (1999)
	Protocalliphora sialia	Falconiformes	Sargent (1938)
		Passeriformes	Gentes et al. (2007)
		Piciformes	Sabrosky et al. (1989)
		Strigiformes	Proudfoot et al. (2005)
	Protocalliphora spatulata	Passeriformes	Miller and Fair (1997)
	Protocalliphora spenceri	Passeriformes	Matsuoka et al. (1997)
	Protocalliphora tundrae	Passeriformes	Sabrosky et al. (1989)
Muscidae	*Passeromyia heterochaeta*	Falconiformes	Gargett (1975)
		Passeriformes	Lindholm (1998)
	Passeromyia indecora	Passeriformes	Pont (1974)
	Philornis carinatus	Passeriformes	Young (1993)

(*continues*)

Table 32.1. (*Continued*)

Family	Species	Host order	Example reference
	Philornis deceptivus	Passeriformes	Uhazy and Arendt (1986)
	Philornis downsi	Passeriformes	Fessl et al. (2006a)
	Philornis mimicola	Strigiformes	Proudfoot et al. (2006)
	Philornis pici	Passeriformes	Nores (1995)
		Psittaciformes	Snyder et al. (1987)
	Philornis porteri	Passeriformes	Spalding et al. (2002)
	Philornis seguyi	Passeriformes	Couri et al. (2005)
	Philornis sp.	Falconiformes	Hector (1982)
Neottiophilidae	*Neottiophilum praeustum*	Falconiformes	Zumpt (1965)
		Passeriformes	Zumpt (1965)

Note: There are several hundred reports associated with species of wild birds; only a single recent report of each fly species/bird order association is provided in this table of representative examples. Individuals interested in detailed reports from individual species are urged to consult the primary literature.

and wild birds in Europe, reports in Nearctic wild birds are largely lacking (Baumgartner 1988), leading some to propose that the disease may be overlooked due to the relatively short time frame in which there is an opportunity to make a diagnosis prior to decomposition of the carcass (Wobeser 1997).

ETIOLOGY

Hematophagous myiasis of nestling birds is caused by members of three families of dipteran flies: Calliphoridae, Muscidae, and Neottiphilidae (Uhazy and Arendt 1986; Spalding et al. 2002). Examples of individual genera and species reported as parasites of different avian hosts are summarized in Table 32.1. *Protocalliphora* is the most common genus reported; approximately 90 species have been described in this genus worldwide (Catts and Mullen 2002).

Traumatic myiasis in wild birds is caused by flies in the families Calliphoridae and Sarcophagidae (Table 32.2). The human skin bot, *Dermatobia hominis*, may use wild birds as a host for larval development (Roberts and Janovy 2005). Other cases of oestrid bot fly infestation in wild birds are rare, but have been reported (Artmann 1975).

EPIZOOTIOLOGY

The complete life cycle has not been fully delineated for any species of diptera with obligate hematophagous larval stages. However, a general pattern for infestation and development of the members of the calliphorid and muscid genera is evident based on what is known (Sabrosky et al. 1989; Spalding et al. 2002; Fessl et al. 2006a). Nestlings are exposed when adult flies oviposit into nests containing newly hatched birds or directly onto the recently hatched nestlings early in the spring. The mechanisms by which adult female flies locate nests containing newly hatched birds in order to deposit their eggs are unknown (Catts and Mullen 2002). The fly eggs develop rapidly to active first-instar larvae within 1–2 days and then migrate to the nestlings. The larvae of most species feed on the nestlings intermittently for a few hours at a time over a period of 1–2 weeks, moving from the nest material to the birds to feed. Some species (e.g., *Protocalliphora braueri*, *Philornis nielseni*) burrow beneath the skin for several days, breathing through a respiratory pore in the skin through which they later exit (Sabrosky et al. 1989). Other species (e.g., *Philornis downsi*) develop through the first and second instar in the nasal cavities and complete development in the nest materials, intermittently moving to the birds to feed and then returning to the nest material (Fessl et al. 2006a). After several blood meals, the third-instar larvae leave the bird to pupate in the nest material. The prepuparial stage is approximately 1–4 days long, while pupation itself may take 36 days or more to complete. Adult flies then emerge to mate and seek new nestlings on which to oviposit. The eggs and pupa of *Protocalliphora* spp. are not considered cold tolerant, and overwintering of those species in the temperate areas where they occur is thought to be achieved by survival of adults that mate the following spring (Sabrosky et al. 1989).

The prevalence of bird nest flies is high among nidicolous birds. In a comprehensive study of *Protocalliphora* spp. on wild birds in Utah, 48% of the nests of 51 species contained *Protocalliphora* spp. (Whitworth and Bennett 1992). For some host species and in some locations and years, almost all nests examined

Table 32.2. Examples of nonhematophagous dipteran larvae found on wild birds.

Family	Species	Host	Scientific name	Reference
Calliphoridae (bottle flies)	*Calliphora* sp.	Peregrine Falcon	*Falco peregrinus*	Cooper (1978)
		American Kestrel	*Falco sparverius*	Cooper (1978)
	Lucilia sericata	Common Crane	*Grus grus*	Itamies and Merila (1984)
		Kestrel	Not specified	Hinaidy and Frey (1984)
		White Stork	*Ciconia ciconia*	Hinaidy and Frey (1984)
		Harrier	not specified	Hinaidy and Frey (1982)
		European Honey-buzzard	*Pernis apivorus*	Hinaidy and Frey (1982)
		Eurasian Eagle-Owl	*Bubo bubo*	Hinaidy and Frey (1982)
		Tawny Owl	*Strix aluco*	Hinaidy and Frey (1982)
		Short-eared Owl	*Asio flammeus*	Frey and Hinaidy (1978)
		American Kestrel	*Falco sparverius*	Cooper (1978)
		Peregrine Falcon	*Falco peregrinus*	Cooper (1978)
		Greylag Goose	*Anser anser*	Farkas et al. (2001)
Sarcophagidae (flesh flies)	*Wohlfahrtia vigil*	American Robin	*Turdus migratorius*	Eschele and Defoliart (1965)
	Wohlfahrtia opaca	Blue-winged Teal	*Anas discors*	Wobeser et al. (1981)
		Northern Shoveler	*Anas clypeata*	Wobeser et al. (1981)
	Wohlfahrtia magnifica	Greylag Goose	*Anser anser*	Farkas et al. (2001)
Cuterebridae (bot flies)	*Cuterebra buccata*	American Woodcock	*Scolopax minor*	Artmann (1975)
	Dermatobia hominis	Macaw, other wild birds	Various	Roberts and Janovy (2005)

are infested (Arendt 1985b; Hurtrez-Bousses et al. 1997a; Wesolowski 2001), leading some to conclude that virtually every bird species in North America with dry nests and altricial (=requiring care at birth) young is susceptible to infestation with *Protocalliphora* spp. (Sabrosky et al. 1989; Bennett and Whitworth 1991). Indeed, while some species of *Protocalliphora* are host specific, others appear to exhibit habitat preferences, being found in nests in a given geographic region, rather than consistently associated with a single host species (Sabrosky et al. 1989).

Intensity of infestations with *Protocalliphora* spp. are extremely varied, with per nest numbers ranging from 1 to more than 1,000 (Seguy 1955; Whitworth and Bennett 1992). Large numbers of larvae seem to be associated with larger, sturdier nests, presumably because more adequate habitat for development in nest material exists (Sabrosky et al. 1989).

Other calliphorids occasionally associated with wound myiasis in wild birds (e.g. *Calliphora*, *Lucilia*) normally deposit eggs onto decaying carcasses. The larvae then hatch and develop as saprophytes, feeding on decomposing tissue. However, these flies have also been reported as facultative parasites capable of invading necrotic tissue in wounds on living birds (Baumgartner 1988; Farkas et al. 2001).

Unlike the calliphorids, female sarcophagid flesh flies associated with cases of obligatory or facultative traumatic myiasis retain the developing eggs until they hatch. Depending on species, each female fly may deposit 30–200 larvae directly onto the animal (Catts and Mullen 2002). Female flesh flies in the genus *Wohlfahrtia* larviposit directly onto mucous membranes or fresh wounds; first-instar larvae of this genus are also able to penetrate intact skin and create a wound (Gassner and James 1948). After feeding on the host tissues and developing for approximately 1 week, the larvae drop to the ground to pupate and continue their development to adult flies (Gassner and James 1948).

CLINICAL SIGNS

Many infested birds do not exhibit any overt clinical disease associated with blood feeding by the larvae. However, anemia has been reported, particularly when infestations are severe (Dudaniec and Kleindorfer 2006; Fessl et al. 2006a). The blood-feeding larvae of some bird nest flies, including *P. braueri* and some *Philornis* spp., do not remain on the surface of the birds, but rather burrow beneath the skin to invade the subcutaneous space. Such infestations may result

in grossly evident subcutaneous cysts containing one or more larvae.

Protocalliphora avium can cause very distinct clinical signs in raptors. This species has a strong predilection for the aural cavity in species of Falconiformes, and developing larvae of this species can often be found completely occluding the opening to the ear in affected nestlings (Sargent 1938; Tirrell 1978; Bortolotti 1985). Larvae of *P. avium* and *P. downsi* may also be found in the nasal cavity (Hill and Work 1947; Fessl et al. 2006a). An early sign of infestation is the accumulation of dried, reddish-brown excreta from developing, blood-feeding larvae around the opening to infested aural and nasal cavities (Bent 1937; Tirrell 1978).

PATHOGENESIS

All species of diptera with hematophagous larvae are obligatory bloodsucking parasites in the immature form. In order to obtain a blood meal, the larvae of these bird nest flies migrate to the surface of the host and then use hooks on the oral cavity to penetrate the skin. A prothoracic fringe is used to hold the maggot in place and advance the anterior portion more deeply into the skin as the hooks continue to tear at the tissue. Once firmly embedded, the larva begins to feed. Feeding is a relatively slow process, and each larva requires a few hours to ingest an entire blood meal (Sabrosky et al. 1989).

The pathogenic effects of infestations with hematophagous dipteran larvae are difficult to summarize succinctly. Otherwise healthy nestlings often seem to tolerate relatively low numbers of feeding larvae well (Gold and Dahlsten 1983; Eastman et al. 1989). However, when the number of larvae is high, particularly in malnourished nestlings or those with concurrent disease, morbidity and mortality may result (Howe 1992; Whitworth and Bennett 1992; Fessl et al. 2006a). In addition, even subclinical infestations appear to prolong nestling development which itself may increase the risk of loss to predation and thus decrease overall nestling success (Arendt 1985a; Hurtrez-Bousses et al. 1997b). Losses are thought to be particularly likely when severe infestations occur concurrent with inclement weather or nutrient shortages (Sabrosky et al. 1989; Whitworth and Bennett 1992).

Occasionally, some larvae that burrow subcutaneously will penetrate through the thoracic, abdominal, or ocular cavity, causing severe damage as they migrate through the tissues. This damage often results in sepsis and ultimately death of affected birds (Arendt 1985a, b; Uhazy and Arendt 1986). Migrating larvae that invade and damage vital organs (e.g., lung, brain, liver) can also directly cause death of affected birds (Spalding et al. 2002).

Despite the impressive number of larvae found in infestations of species which penetrate the aural or nasal cavities, permanent damage does not apparently occur in most cases (Tirrell 1978). Disfigured aural cavities, damaged tympana, and enlarged nostrils have been reported, however (Bent 1937; Fessl et al. 2006a).

Disease caused by bird nest flies is directly related to blood loss from the feeding larvae and the subsequent iron deficiency anemia that develops. Gold and Dahlsten (1983) calculated that in a heavily infested nest, feeding larvae can consume more than 55% of a chick's blood volume. This chronic blood loss early in life may result in prolonged fledging times and a subsequent increase in the likelihood of predation or disease loss due to other causes (Hurtrez-Bousses et al. 1997a, b). In some cases, parents of nestlings heavily parasitized by *Protocalliphora* spp. are able to somewhat offset the adverse effects of blood loss by nutritional supplementation of the nestlings (Hurtrez-Bousses et al. 1998; Simon et al. 2004), but the energy cost required to increase the feeding has been shown to reduce the subsequent fitness of those parents (Wesolowski 2001).

Among species of diptera with hematophagous larvae that also invade subcutaneously, the larvae continue to advance through the skin until they reach the subcutaneous space. Here, they continue to ingest blood and other host tissue. Detrimental host effects may arise from the location, aggressiveness, or sheer number of the migrating larvae (Arendt 1985a, b; Spalding et al. 2002).

PATHOLOGY

Blood feeding by larvae would be expected to reduce the hematocrit of affected birds. However, perhaps due to rapid production of red blood cells in response to blood loss in the young birds, nestlings with heavy infestations of bird nest flies may have normal or only slightly decreased packed cell volumes (Johnson and Albrecht 1993; O'Brien et al. 2001). Hemoglobin concentrations may be reduced in the face of normal hematocrit levels, which could result in compromised oxygen transport to tissues and subsequent compromised development and survival (O'Brien et al. 2001).

The pathogenic effects of other diptera with hematophagous larvae are more clearly established, particularly for those that migrate through body tissues. For example, parasitism by *Philornis* spp. is responsible for mortalites as high as 97% in nestling Pearly-Eyed Thrashers (*Margarops fuscatus*) in Puerto Rico (Arendt 1985b), and is a significant cause of mortality in several other bird species as well (Fraga 1984; Nores 1995; Spalding et al. 2002; Fessl et al. 2006a). Subcutaneous invasion by other hematophagous dipteran

larvae has also been associated with significant mortality in affected nestlings, presumably due to increased susceptibility to secondary bacterial infections (Young 1993; Warren 1994).

Gross lesions associated with blood-feeding larvae that do not penetrate subcutaneously are limited to the presence of larvae on the birds during feeding and the presence of dried brownish-red larval excreta around feeding sites (Tirrell 1978). The skin surrounding the attachment site of feeding larvae may become swollen and edematous (Tirrell 1978). When larvae penetrate into the subcutaneous space and migrate, lesions are more severe. Uhazy and Arendt (1986) described the lesions associated with subcutaneous *Philornis deceptivus* in nestlings of Pearly-eyed Thrashers as raised and elongate nodules or as open cavities once larvae had exited. Microscopically, the lesions consisted of a cyst lined by mononuclear cells and surrounded by edematous connective tissue. Basophilic necrotic tissue and mononuclear cell infiltrations were associated with the larval mouthparts in active lesions. After larvae exited the cavity, wound contraction occurred and the cyst contained large numbers of mononuclear cells and a fibrinous exudate (Uhazy and Arendt 1986).

Pathology caused by sarcophagid species during traumatic myiasis is similar to that caused by subcutaneously migrating hematophagous dipteran larvae. Maggots may be found within subcutaneous cysts that communicate with the surface of the skin via a small circular opening, or in open cavities (Wobeser et al. 1981). In domestic Greylag Geese (*Anser anser*) infested with *Lucilia sericata* and *W. magnifica*, moderate to severe bleeding is also noted at wound sites and epidermal necrosis seen at the edges of wounds (Farkas et al. 2001).

DIAGNOSIS

Avian myiasis due to hematophagous dipteran larvae is readily diagnosed by observing the presence of blood-fed larvae on or in the nests of live birds, or upon direct examination of affected nestlings. Larvae are found on or around nestlings within the first days to weeks after chicks hatch. Larvae may be seen feeding on the nestlings directly or may be found in the nest material between feedings. However, identification of the exact species of larvae found may be more difficult. Keys are available for identification of dipteran larvae found on nestlings (e.g., Hall 1948; Teskey 1981; Sabrosky et al. 1989; Foote 1991; Whitworth 2002). Use of these keys can be challenging, and guidance from an experienced entomologist familiar with taxonomy and identification of dipteran larvae is critical. Identification of these larvae may also be facilitated by collection of live larvae from the nest and then allowing some specimens to develop to adults. Information on collecting, rearing, and preserving *Protocalliphora* spp. can be found in Sabrosky et al. (1989). Care should also be taken in identifying the agents of traumatic or wound myiasis, particularly when larvae are collected from dead birds. Carcass-feeding larvae are commonly found on vertebrate carcasses in the wild and are unlikely to have played a role in the death of the bird (Baumgartner 1988).

IMMUNITY

Little is known about immune responses of nestling birds to *Protocalliphora* spp. or other hematophagous dipteran larvae. Although occasionally some species of maggots may be found feeding on older birds, a strong preference for newly hatched nestlings has been shown in several individual species and is likely true for most of these diptera (Sabrosky et al. 1989). The relatively low numbers of larvae on adult birds may also be due to removal by preening (Arendt 1985b).

The immune response to traumatic or wound myiasis in birds is similarly understudied, but inferences can be drawn from what is known about the response to infestation in mammalian hosts, particularly livestock. Following infestation by *Lucilia cuprina* in sheep, granulocytes infiltrate the wound surface while lymphocytes aggregate in the dermis. High antibody titers occur upon secondary infestation; however, titers do not appear to differ between resistant and susceptible animals, suggesting any resistance to infestation may be an innate response (Otranto 2001). At present, vaccination for myiasis is not available in humans or domestic animals.

PUBLIC HEALTH AND DOMESTIC ANIMAL CONCERNS

There is no known public health concern associated with hematophagous myiasis flies in birds. Traumatic or wound myiasis does occur in people, but fly infestations on birds are not considered a major source of adult flies which can then deposit their offspring on humans. Similarly, there is no major domestic animal health concern associated with avian myiasis. Traumatic or wound myiasis does occur in domestic animals, including domestic birds such as geese (Farkas et al. 2001), but as with myiasis in people, wild birds are not considered a major source of the adult flies.

WILDLIFE POPULATION IMPACTS

The role of blood-feeding larvae of bird nest flies as pathogens that can impact wild bird populations

remains controversial. Infestations of nestlings with hematophagous dipteran larvae are almost ubiquitous across passerine taxa. The *Protocalliphora* spp. alone appear capable of parasitizing all species of North American birds with altricial young and dry nests (Sabrosky et al. 1989; Bennett and Whitworth 1991), and do not appear to themselves result in wide-scale mortality. However, when coupled with environmental stressors such as habitat reduction, inclement weather, and limited nutrient sources, the presence of the larvae of *Protocalliphora* spp. and resultant increased feeding demands on parents of heavily parasitized nestlings may contribute to both fledgling failure and decreased parental survival in future years (Hurtrez-Bousses et al. 1998; Simon et al. 2004).

While it seems intuitive that chronic blood loss in young, developing birds is compromising, several studies have not shown any adverse effects on the weight or fledging success of the nestlings themselves. Indeed, infestations of *P. braueri* on nestling House Wrens (*Troglodytes aedon*) did not have an effect on either nestling survival or fledgling size as measured by tarsus length at fledging (Eastman et al. 1989). However, in other instances, heavy infestations are associated with delayed development, prolonged time to fledge, and nestling mortality (see summary in Sabrosky et al. 1989). In times of abundant food, increased parental feeding may compensate for the physiologic cost of chronic blood loss associated with the hematophagous larvae (Hurtrez-Bousses et al. 1998; Simon et al. 2004). However, if inclement weather or food shortages coincide with heavy infestations by *Protocalliphora* spp., these diptera may contribute to nestling mortality (Sabrosky et al. 1989; Howe 1992; Whitworth and Bennett 1992). For example, a study of *P. braueri* in nestling Sage Thrashers (*Oreoscoptes montanus*) found no effect on fledgling mortality in a year with consistently mild weather, but the following year, when an 8-day period of severe inclement weather occurred, parasitism by *P. braueri* was positively associated with nestling mortality (Howe 1992).

Habitat disruption can also impact patterns of infestation by *Protocalliphora* spp. on nestlings. In a recent study of Tree Swallows (*Tachycineta bicolor*) in Alberta, Canada, 100% of nests examined were infested with *Protocalliphora* spp., and most of the nests contained more than one species. Interestingly, nests were much more heavily infested on wetlands impacted by oil sands mining than those on an undisturbed site, and nestling growth was adversely affected by the higher parasite load (Gentes et al. 2007).

Muscid larvae, such as those of *Philornis* spp. or *Passeromyia* spp., can also be significant mortality factors for nidicolous birds in tropical areas (Arendt 1985b; Fessl et al. 2006a). These dipteran larvae have been reported as a cause of severe impacts in endangered species of birds when they encounter newly introduced dipteran parasites. Such a problem may have occurred when Pearly-eyed Thrashers invaded the forest reserve of the Puerto Rican Parrot (*Amazona vittata*), bringing with them *Philornis* spp. Fatal parasitism of parrot nestlings with *Philornis* larvae was subsequently reported (Snyder et al. 1987). This situation underscores the potentially catastrophic result of introduction of a new parasite into an already fragile system. Introduction of *P. downsi* into the avifauna of the Galapagos Islands appears to be having similar negative impact on survival of nestling Darwin's finches, including the Mangrove Finch (*Camarhynchus heliobates*) and the Medium-Tree Finch (*Camarhynchus pauper*), of which there are only an estimated 50 and 300 breeding pairs remaining, respectively (Fessl et al. 2006a).

Very little information is available about the degree to which traumatic myiasis affects bird populations, although reports of this condition caused by *Wohlfahrtia*, *Lucilia*, and *Calliphora* spp. and associated with morbidity and mortality in waterfowl, raptors, and passerines can be found in the literature (Eschele and DeFoliart 1965; Cooper 1978; Wobeser et al. 1981; Baumgartner 1988; Farkas et al. 2001) (Table 32.2). Infestations of wounds with fly larvae can cause severe disease in affected individuals, but the impact on populations is considered relatively minor. The larvae of *W. opaca* have been identified as a cause of mortality in ducklings, but only 0.7% of examined ducklings were found infested (Wobeser et al. 1981). Similarly, a recent survey of geese in Hungary found only ~0.1% of domestic geese infested with *W. magnfica* and *L. sericata* (Farkas et al. 2001).

TREATMENT AND CONTROL

Natural mechanisms that result in removal of hematophagous dipteran larvae from nests containing young birds have been shown to increase nestling survival. Adult Bay-winged Cowbirds (*Agelaioides badius*) that removed larvae from their nestlings increased nestling survival (Fraga 1984). Similarly, when various species of oropendola and cacique located nests near parasitic wasp nests, parasitism by *Philornis* spp. declined and fledging success increased (Smith 1968). However, artificial removal of dipteran larvae from the nests of birds is not practical in the wild and may adversely disturb developing nestlings. If parasitism is quite high, infested nests can be removed and destroyed and replaced with artificial ones. This approach would only be effective for controlling larvae that do not burrow subcutaneously or remain on the birds for long periods of time.

Application of insecticides to the nest material has also been proposed, although this approach has inherent limitations in terms of both safety and practicality (Sabrosky et al. 1989). Application of insecticide to nests is perhaps best pursued when severe outbreaks occur that involve threatened or endangered birds. Treatment of nests with insecticides was effective during an outbreak of *P. downsi* on nestlings of Darwin's finches. Application of 1% pyrethrin solution to the nests cleared the infestations and significantly improved fledgling success relative to untreated controls. When treated soon after eggs hatched, the nests remained free of *P. downsi* for the remainder of the nestling period (Fessl et al. 2006b). The safety of pesticide treatments and other similar approaches, such as hanging pest strips near the nest, should be explored further as the potential benefit for nestling survival may be quite significant. In the Galapagos finch study, treated nests had a fledgling rate more than twice that of untreated nests (86.6 and 33.9%, respectively) (Fessl et al. 2006b).

Treatment of wound myiasis requires removing the larvae from the affected tissue, managing the wound to encourage healing, and providing necessary supportive care. Animals recovering from myiasis must be kept indoors and protected from flies to prevent reinfestation of healing wounds with fresh maggots. Because the flies that cause facultative and obligatory myiasis develop rapidly and can continue to develop in carcasses after death, prevention of wounds, such as occurs during feather plucking of geese, and prompt carcass removal may aid control of these flies during a die-off (Farkas et al. 2001).

MANAGEMENT IMPLICATIONS

Although most bird populations appear to tolerate the presence of blood- and tissue-feeding larvae on their young, these parasites may create an added stressor in times of limited nutrients and excess energy requirements. Some ecologists have speculated that increasing edge effects between different habitats will bring previously segregated species closer together, possibliy leading to spread of parasites, including bird nest flies, to new hosts and new populations. Loss of nesting habitat that forces more frequent use of old nest sites may also increase exposure to bird nest flies among nestlings (Loye and Carroll 1995).

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Index

Aatinga leucophthalma (White-eyed Parakeet), 401
Abdominal cavity, 449
Acanthocephala
  clinical signs, 280
  diagnosis, 283
  domestic animal health concerns, 283
  epizootiology, 277–280
  etiology, 277
  history, 277
  host range and distribution, 277
  ova of some common acanthocephalans of birds, 284
  pathogenesis and pathology, 280–283
  proboscides of acanthocephalans of birds, 278
  public health concerns, 283
  synonym, 277
  treatment and control, 285
  wild population impacts, 283–285
Acanthocephalans, 277–279, 280, 282
  eggs, 284
  proboscides of, 278
Acanthocephaliasis, 277
Acanthocephalosis, 277
Acariasis
  diagnosis, 534
  domestic animal health concerns, 534
  epizootiology, 529–530
  etiology, 527–529
  host range and distribution, 527
  immunity, 534
  pathogenesis and pathology, 530–534
  public health concerns, 534
  synonyms, 527
  treatment and control, 535
  wildlife population impacts, 535
Accipiter, 465
Accipiter badius (Shikra), 77, 130
  Accipiter brevipes (Levant Sparrowhawk), 78
  Accipiter cooperii (Cooper's Hawk), 76, 130, 144, 205, 237
  Accipiter gentilis (Northern Goshawk), 76, 114, 130, 143–144, 205, 215, 346, 348, 470
  Accipiter gularis (Japanese Sparrowhawk), 78
  Accipiter minullus (Little Sparrowhawk), 78
  Accipiter nisus (Eurasian Sparrow Hawk), 77, 84, 94, 346, 470
  Accipiter rufiventris (Rufous-chested Sparrowhawk), 130
  Accipiter soloensis (Chinese Goshawk), 78
  Accipiter striatus (Sharp-shinned Hawk), 77, 395
  Accipiter tachiro (African Goshawk), 78, 130
  Accipiter trivirgatus (Crested Goshawk), 395, 471
  Accipiter virgatus (Besra), 78
Accipitridae (Hawks, Eagles, and Kites), 57, 59, 74, 395, 441, 505
acetic acid, 509
Acheta domestica, 378
Acipenser gueldenstaedtii (Sturgeon), 301
Aconoidasida, 15, 36
Acrocephalus schoenobaenus (Sedge Warbler), 345, 479
Acrochordus javanicus (Javan wart snake), 302
Acryllium vulturinum (Vulturine Guinea Fowl), 393, 397, 473
Actitis, 465
  Actitis hypoleucos (Common Sandpiper), 475
  Actitis macularius (Spotted Sandpiper), 332, 475
Actophilornis africanus (African Jacana), 331, 339
Actornithophilus limosae, 521
Acuaria spp.
  Acuaria (Hamannia) uncinata, 335
  Acuaria nasuta, 327
  Acuaria spiralis, 327
Acuariasis, 335
Acuarioid nematodes, 326
Acuariosis, 326
Aechmophorus occidentalis (Western Grebe), 328
Aedeomyia squamipennis, 41
Aegithinidae (Ioras), 61, 444
Aegolius acadicus (Northern Saw-whet Owl), 114, 477
Aegothelidae (Owlet-Nightjars), 443
Aegypius monachus (Cinereous Vulture), 205
Aetheobdella hirudoides, 505
Aethia cristatella (Crested Auklet), 394
Aethia pygmaea (Whiskered Auklet), 394
African Black Duck (*see Anas sparsa*)
African Fish-Eagle (*see Haliaeetus vocifer*)
African Goshawk (*see Accipiter tachiro*)
African Green-Pigeon (*see Treron calvus*)
African Jacana, (*see Actophilornis africanus*)
African Mourning Dove (*see Streptopelia decipiens*)
African Spoonbill (*see Platalea alba*)
African Swift (*see Apus barbatus*)
Agapornis, 465
  Agapornis fischeri (Fischer's Lovebird), 399
  Agapornis personatus (Yellow-collored Lovebird), 401
Agar gel precipitation, 92
Age, as disease determinant of wild birds, 3
Agelaius phoeniceus (Red-winged Blackbird), 42, 209, 479
Ahanta Francolin (*see Francolinus abantensis*)
Aigamo method, of rice farming, 255
air sacculitis, 527
Aix, 465
Aix galericulata (Mandarin Duck), 68, 329, 358, 364
Aix sponsa (Wood Duck), 22, 67, 83, 208, 236, 270, 329, 360, 365, 390, 468, 486
Akiba caulleryi, 54
Alauda arvensis (Eurasian Skylark), 479
Alaudidae (Larks), 60, 444
albendazole, 406, 424, 491
albumen, 234
Alca torda (Razorbill), 227, 332

Parasitic Diseases of Wild Birds Edited by Carter T. Atkinson, Nancy J. Thomas and D. Bruce Hunter
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Alcedinidae (Kingfishers), 60, 443
Alcidae (Auks, Murres and Puffins), 442
Alectoris, 465
Alectoris barbara (Barbary Partridge), 331, 390, 396
Alectoris chukar (Chukar), 114, 155, 169, 391, 396
Alectoris graeca (Rock Partridge), 213, 214, 215, 392, 396
fed with *Toxoplasma gondii* oocysts, 213, 215
Alectoris rufa (Red-legged Partridge), 144, 171, 214, 270, 317, 392, 406
Alectroenas pulcherrima (Seychelles Blue-Pigeon), 125
Alectura lathami (Australian Brush-turkey), 390, 395
Alexandra's Parrot (*see Polytelis alexandrae*)
Alexandrine Parakeet. (*see Psittacula eupatria*)
Alisterus scapularis (Australian King-Parrot), 399
Alle, 465
Alligator mississippiensis (American alligator), 303
Allobilharzia, 246, 247, 251
Allobilharzia visceralis, 251
Allolobophora caliginosa, 401, 464
Allolobophora parva, 464
allozymes, 425
Alopochen aegyptiaca (Egyption Goose), 69, 329
Alpine Accentor (*see Prunella collaris*)
Alpine Swift (*see Tachymarptis melba*)
Amabiliidae, 264
Amadina fasciata, 378
Amaurornis phoenicurus (White-Breasted Waterhens), 488
Amazona spp.
Amazona aestiva (Blue-fronted Parrot), 399
Amazona agilis (Black-billed Parrot), 399
Amazona amazonica (Orange-winged Parrot), 400
Amazona arausiaca (Red-necked Parrot), 400
Amazona autumnalis (Red-lored Parrot), 400
Amazona farinose (Mealy Parrot), 400
Amazona festiva (Festive Parrot), 399
Amazona leucocephala (Cuban Parrot), 399
Amazona ochrocephala (Yellow-crowned Parrot), 401
Amazona vinacea (Vinaceous Parrot), 400
Amazona vittata (Puerto Rican Parrot), 400, 553
American Avocet (*see Recurvirostra americana*)
American Bittern (*see Botaurus lentiginosus*)
American Black Duck (*see Anas rubripes*)
American Coot (*see Fulica americana*)
American Crow (*see Corvus brachyrhynchos*)
American Goldfinch (*see Carduelis tristis*)
American Kestrel (*see Falco sporverius*)
American Redstart (*see Setophaga ruticilla*)
American White Pelican (*see Pelecanus esythroshynchos*)
American Wigeon (*see Anas americana*)
American Woodcock (*see Scolopax minor*)
Amblycera, 516, 517
Amherst (AG) strain, 134
Amicobdella nigra, 505
Amidostomatinae, 366
Amidostomiasis, 355
Amidostomum spp., 355
Amidostomum acutum, 355, 356, 357, 358, 361
Amidostomum anseris, 355, 356, 357, 358, 361, 366
Amidostomum auriculatum, 357, 366
Amidostomum biziurae, 366
Amidostomum cygni, 356, 358, 366
Amidostomum fulicae, 356, 357, 358, 361, 366
Amidostomum henryi, 358, 361, 366
Amidostomum quasifulicae, 361
Amidostomum raillieti, 366
Amidostomum ryzhikovi, 366
Amidostomum skrjabini, 370
Amidostomum spatulatum, 355, 356, 358, 366
Amidostomum species, 355
Amidostomum tribonix, 366
Amidostomum tribonyx, 361, 366
avian hosts infected with, 362–365
clinical signs, 368
diagnosis, 370
domestic animal health concerns, 370
eggs, 367
epizootiology, 367–368
etiology, 366–367
history, 355
host range and distribution, 355–366
immunity, 370
management implications, 371
pathogenesis and pathology, 368–370
prevalence, intensity and abundance, 367–368
public health concerns, 370
synonyms, 355
treatment and control, 371
wildlife population impacts, 371
Ammodramus humeralis (Grassland Sparrow), 114
ammonia, 509
Ammoperdix griseogularis (See-see Partridge), 393
Amoebotaenia, 263
Ampeliceps coronatus (Golden-crested Myna), 111
Amphibiocapillaria, 463
Amphimerus, 231
Amphimerus elongates, 238
Amphimerus elongatus, 233, 235
Amphimerus heterolecithodes, 233, 237
Amphipoda, 279
Amphiuma tridactylum (Three-toed amphiuma), 303
Amplicaecum Baylis, 413
Amprolium, 172, 177, 192
Anas spp., 236, 336, 465, 488
Anas acuta (Northern Pintail), 63, 173, 205, 227, 235–236, 296, 329, 336, 355, 358, 364, 395
Anas americana (American Wigeon), 65, 115, 329, 356, 362, 368
Anas bahamensis (White-cheeked Pintail), 329
Anas capensis (Cape Teal), 329, 362
Anas carolinensis (Green-winged Teal), 227, 235, 355, 357, 363
Anas castanea (Chestnut Teal), 329
Anas clypeata (Northern Shoveler), 63, 115, 173, 235, 296, 329, 359, 364, 550
Anas creccai (Eurasian Teal), 64, 173, 468
Anas cyanoptera (Cinnamon Teal), 329, 356, 362
Anas discors (Blue-winged Teal), 64, 115, 173, 227, 234, 235, 236, 249, 329, 336, 356, 362, 550
Anas erythrorhyncha (Red-billed Duck), 364
Anas falcata (Falcated Duck), 65, 329, 357, 363
Anas flavirostris (Speckled Teal), 390
Anas formosa (Baikal Teal), 65, 329, 356, 362
Anas fulvigula (Mottled Duck), 83, 235, 358, 364, 468
Anas georgica (Yellow-billed Pintail), 329, 360
Anas gracilis (Gray Teal), 357, 363
Anas laysanensis (Laysan Duck), 339
Anas luzonica (Philippine Duck), 330
Anas penelope (Eurasian Wigeon), 65, 329, 357, 363, 389
Anas platyrhynchos and others (Mallard), 346
Anas platyrhynchos, 62, 205, 217
Anas platyrhynchos (Domestic White Pekin), 296, 502
Anas platyrhynchos (Ducks), 163, 488
Anas platyrhynchos (Juvenile Mallards), 367

Anas platyrhynchos (Mallard), 62, 83, 154, 173, 205, 217, 228, 233, 235, 236, 255, 296, 304, 329, 336, 358, 364, 379, 380, 389, 395, 468, 504
Anas platyrhynchos (Mallard ducklings), 250
Anas poecilorhyncha (Spot-billed Duck), 359, 365
Anas querquedula (Garganey), 63, 296, 330, 357, 363
Anas rubripes (American Black Duck), 65, 83, 227, 234, 235, 236, 249, 250, 330, 356, 362, 445, 451
Anas sibilatrix (Chiloe Wigeon), 329, 356
Anas smithii (Cape Shoveler), 362
Anas sparsa (African Black Duck), 330, 356
Anas specularis (Spectacled Duck), 330, 359
Anas strepera (Gadwall), 65, 173, 205, 330, 336, 338, 357, 363, 389
Anas superciliosa (Pacific Black Duck), 330, 359, 502
Anas undulata (Yellow-billed Duck), 330, 360
Anas versicolor (Silver Teal), 329
Anastomus oscitans (Asian Openbill), 235
Anatidae (Ducks, Geese, and Swans), 59
Anatini (Dabbling Ducks), 367
Andersonfilaria, 441–445, 448, 454
Andersonfilaria africanus, morphology of microfilariae of, 453
anemia, 21, 44, 87, 88–89
Anhinga (*see Anhinga anhinga*)
Anhimidae (screamers), 441
Anhinga anhinga (Anhinga), 293, 416, 466
Anhinga melanogaster (Darter), 293
Anhingidae (Anhingas), 59, 441
Anisakiasis, 413
anisakid infections, 424
Anodorhynchus hyacinthinus (Hyacinth Macaw), 400
anonymity, of wild birds, 3
anopheline mosquitoes, 40
Anoplura, 516
anorexia, 21, 87
Anous minutus (Black Noddy), 416, 423
proventriculus of a, 422
anoxia, 44
Anseranas semipalmata (Magpie Goose), 208, 217
Anseriformes, 14, 18, 22, 23, 54, 59, 121, 163, 165, 166, 173–175, 205, 208, 217, 235, 247, 251, 252, 253, 296, 322, 328, 376, 441, 465, 468, 521
Anser spp., 465
Anser albifrons (Greater White-fronted Goose), 69, 328, 357, 363, 389
Anser anser (Graylag Goose), 69, 173, 174, 205, 296, 358, 363, 367, 389, 395, 550, 552
Anser brachyrhynchus (Pink-footed Goose), 359
Anser cygnoides (Swan Goose), 69, 328, 346, 390
Anser erythropus (Lesser White-fronted Goose), 358, 364, 389
Anser fabalis (Taiga Bean-Goose), 69, 356, 362
Anser indicus (Bar-headed Goose), 356, 362
anthelmintics, 322, 339, 352, 371, 386, 406
anthropogenic alterations, impact of, 7
Anthropoides paradiseus (Blue Crane), 182, 397
Anthropoides virgo (Demoiselle Crane), 181, 182, 397
Anthus spp.
Anthus hodgsoni (Olive-backed Pipit), 479
Anthus pratensis (Meadow Pipit), 479
Anthus trivialis (Tree Pipit), 479
anti-*Eimeria* IgM, IgY, and IgA antibodies, 171
anti-erythrocyte factor, 89
Aonchotheca, 463
aorta, 449
Apapane (*see Himatione sanguinea*)
Apatemon, 231
Aphelocoma coerulescens (Florida Scrub Jay), 333, 434
Apicomplexa, 15, 36, 163, 195, 204
Aploparaksis furcigera, 264, 268, 269
Aploparaksis penetrans, 264, 268
Apodidae (Swifts), 443
Apodiformes, 14, 59, 163, 435, 443, 478, 521
Aporrectodea rosea, 486
Aproctella, 441–445, 448, 454
Aproctella alessandroi, 451
Aproctella stoddardi, morphology of microfilariae, 453
Aproctiana, 441–445, 448, 454
Aproctoidea, 440
Aproctoids, 439
Aprosmictus erythropterus (Red-winged Parrot), 400
Apterygidae (Kiwis), 441
Apterygiformes, 14, 175, 441
Apteryx mantelli (North Island Brown Kiwi), 175
Apus spp.
Apus affinis (Little Swift), 446, 451
Apus apus (Common Swift), 478
Apus barbatus (African Swift), 521
Aquila spp
Aquila chrysaetos (Golden Eagle), 75, 76, 131, 215
Aquila clanga (Greater Spotted Eagle), 76, 297
Aquila clanga (Spotted Eagle), 298
Aquila fasciata (Bonelli's Eagle), 131
Aquila pennatus (Booted Eagle), 131
Aquila pomarina (Lesser Spotted Eagle), 76, 297
Aquila rapax (Tawny Eagle), 76
Aquila wahlbergi (Wohlberg's Eagle), 76
Ara spp.
Ara ararauna (Blue-and-yellow Macaw), 233, 399, 427
Ara chloropterus (Red-and-green Macaw), 400
Ara macao (Scarlet Macaw), 400, 427
Aramidae (Limpkins), 360, 442
Aramides saracura (Slaty-breasted Wood-Rail), 114
Aramus guarauna (Limpkin), 360
Aratinga spp.
Aratinga acuticaudata (Blue-crowned Parakeet), 399
Aratinga cactorum (Caatinga Parakeet), 399, 476
Aratinga chloroptera (Hispaniolan Parakeet), 399
Aratinga jandaya (Jandaya Parakeet), 400
Aratinga leucophthalma (White-eyed Parakeet), 401
Aratinga pertinax (Brown-throated Parakeet), 399
Aratinga solstitialis (Sun Parakeet), 400
Arborophila torqueola (Hill Partridge), 391
Arctic Loon (*see Gavia arctica*)
Ardea, 465
Ardea alba (Great Egret), 115, 294, 304, 384, 468
Ardea cinerea (Gray Heron), 293, 346
Ardea cocoi (Cocoi Heron), 294
Ardea goliath (Goliath Heron), 294
Ardea herodias (Great Blue Heron), 115, 235, 293, 304, 308, 468
Ardeidae (Herons, Egrets, and Bitterns), 59, 441, 505
Ardeiformes, 376
Ardeola, 251
Ardeola bacchus (Chinese Pond-heron), 294, 488
Ardeotis kori (Kori Bustard), 269
arecoline hydrobromide, 424
Arhythmorhynchus, 277
Armadillidium vulgare, 327
Artamidae (Wood Swallows), 445
Arthrocystis galli, 23
arthropod ectoparasites, effects of, 3
ascariasis, 388

Ascaridia spp., 402
Ascaridia aegyptiaca, 394, 395
Ascaridia amblimoria, 401
Ascaridia australis, 398, 401
Ascaridia bonasae, 396, 397
Ascaridia borealis, 397
Ascaridia brasiliana, 395
Ascaridia catheturina, 395
Ascaridia circularis, 401
Ascaridia columbae, 394, 396, 398, 399, 401, 405
Ascaridia compar, 395, 396, 397
Ascaridia cordata, 395
Ascaridia cristata, 397
Ascaridia cuculina, 401
Ascaridia dissimilis, 397, 401, 402
Ascaridia dolichocerca, 395
Ascaridia fasciata, 397
Ascaridia francolina, 395, 396
Ascaridia galli, 388, 394, 395, 396, 397, 399, 401, 402, 406
Ascaridia geei, 394, 398
Ascaridia hermaphrodita, 398, 399, 400, 401
Ascaridia hermaphroditia, 394
Ascaridia longecirrata, 398
Ascaridia maculosa, 398
Ascaridia magalhãesi, 398
Ascaridia magnipapilla, 396
Ascaridia nicobarensis, 400
Ascaridia numidae, 396, 397, 401, 404
Ascaridia ornata, 400
Ascaridia orthocerca, 395
Ascaridia perspicillum, 402
Ascaridia pintoi, 395
Ascaridia platyceri, 394, 398, 399, 400, 401
Ascaridia pterophora, 397
Ascaridia razia, 398
Ascaridia sergiomeirai, 395, 399, 400, 401
Ascaridia serrata, 395
Ascaridia skrjabini, 396
Ascaridia stroma, 397
Ascaridia struthionis, 395
Ascaridia styphlocerca, 395, 396
Ascaridia trilabium, 401
avian hosts infected with, 395–401
clinical signs, 404
diagnosis, 405
distribution and host range, 388–394
epizootiology, 401–404
etiology, 394–401
history, 388
immunity, 405
pathology, 404–405
public and domestic animal health concerns, 405
synonyms, 388
treatment and control, 406–407
wildlife population impacts, 405–406
Ascaridoidea, 413, 414
Ascaridoid infections, 424
Ascaridoid parasites, 339
Ascaris spp., 425
Ascarosis, 388
Ascocotyle, 230
Ascometra choriotidis, 264, 268
asexual reproduction
of *Isospora* species, 110
of *Plasmodium gallinaceum*, 39
of *Plasmodium* species, 36
asexual sporogonic cycle, of *Haemoproteus*, 17–18
Ashy-headed Goose (*see Chloephaga poliocephala*)
Asian Openbill (*see Anastomus oscitans*)
Asio flammeus (Short-eared Owl), 550
Asio otus (Northern Long-eared Owl), 114, 477
aspartate aminotransferase, 42
Aspergillus sp., 140, 434
atebrine, 28, 95
Athene noctua (Little Owl), 54, 133, 206, 477
Athesmia, 229
Athesmia heterolecithodes, 226
Athesmia jolliei, 237
Atoxoplasma, 163, 204, 214. *see also* Atoxoplasmosis
distribution, 109
domestic animal health concerns, 117
history, 108–109
public health concerns, 116
treatment and control, 117
wildlife population impacts, 117
Atoxoplasmosis. *see also Atoxoplasma*
clinical signs, 112
diagnosis, 112–113
epizootiology, 110–112
etiology, 109–110
host range, 109
immunity, 113
pathogenesis and pathology, 112
synonym, 108
Atrichornithidae (Scrub-birds), 444
atropine sulfate, 371
Attila, 465
Austrobilharzia terrigalensis, 251
Australian Brush-turkey (*see Alectum lathani*)
Australian Kestrel (*see Falco cenchroides*)
Australian King Parrot (*see Alisterus scapularis*)
Austral Parakeet (*see Enicognathus Ferrugineus*)
Australasian Magpie (*see Gymnoshina tibicen*)
Australian Masked-Owl (*see Tyto novaehollandiae*)
Austrobilharzia spp., 231, 246, 247, 249, 251
Austrobilharzia terrigalensis, 251
Austrobilharzia variglandis, 249, 251, 255
Australian Pelican (*see Pelecanus conspicillatus*)
Austromenopon phaeopodis, 521
Austrosimulium spp.
Austrosimulium australense, 85
Austrosimulium dumbletoni, 85
Austrosimulium ungulatum, 85
avian coccidia, with direct life cycles, 108
avian filarioid genera, in families of birds, 441–445
avian filarioid nematodes, 453
Dirofilariinae, 454
Lemdaninae, 454
Onchocercinae, 454
Splendidofilariinae, 454
avian haemoproteids, 14, 15, 19
host distribution of by avian order, 14
mitochondrial and nuclear gene sequences, 17
role of host migratory behavior in cycle of transmission, 20
avian life history, 3
avian malaria, 13
clinical signs, 42–43
diagnosis, 45–46
distribution, 36
domesticated animal health concerns, 47
epizootiology, 39–42
etiology, 36–39

history, 35–36
host range, 36
immunity, 46–47
intrinsic and extrinsic factors of distribution, 41
management implications, 48–49
mortality, 48
pathology and pathogenesis, 43–45
prevalence of, 41
public health concerns, 47
spatial and seasonal patterns of transmission, 40
stress-mediated changes in the immune system, 40
synonyms, 35
treatment and control, 48
wildlife population impacts, 47–48
avian species, of *Plasmodium*, 37
Avioserpensosis
clinical signs, 385
diagnosis, 385
domestic animal health concerns, 386
epizootiology, 384–385
etiology, 384
history, 384
host range and distribution, 384
immunity, 385
pathology, 385
public health concerns, 385
treatment and control, 386
wildlife population impacts, 386
Avioserpens spp.
in domestic birds, 385
goslings infected with, 385
infections in wild birds, 386
outbreaks of disease, 386
Avioserpens mosgovoyi, 384, 386
Avioserpens taiwana, 384
Aythya, 465
Aythya affinis (Lesser Scaup), 7, 66, 165, 166, 171, 173, 227, 225, 235, 236, 330, 338, 358, 368
Aythya americana (Redhead), 66, 173, 330, 359, 364
Aythya australis (White-eyed Duck), 330
Aythya collaris (Ring-necked Duck), 66, 359, 364, 468
Aythya ferina (Common Pochard), 66, 205, 357, 362, 395
Aythya fuligula (Tufted Duck), 66, 165, 205, 227, 296, 330, 359, 365
Aythya marila (Greater Scaup), 66, 227, 235, 236, 330, 357, 363, 469
Aythya nyroca (Ferruginous Pochard), 330, 357, 363, 469
Aythya valisineria (Canvasback), 66, 173, 227, 235, 330, 356, 362
Aythya valisineria (Canvas Dack), 330
Aythyini (Diving Ducks), 367
Azure-winged Magpie (*see Cyanopica cyanus*)

Bacillus subtilis, 157
Bacillus thuringiensis israelensis (Bti), 94, 544
Baikal Teal (*see Anas formosa*), 65
Balaenicipitidae (Shoebills), 59, 441
Bald Eagle (*see Haliaeetus leucocephalus*)
Balearica, 181
Balearica pavonina (Black Crowned-Crane), 22, 397, 475
Balearica regulorum (Gray Crowned-Crane), 397
Bali Myna (*see Leucopsar rothschildi*)
Band-tailed Pigeon (*see Patagioenas fasciata*)
Bangkok hemorrhagic disease, 54
Barbary Partridge (*see Alectoris barbara*)
Bare-faced Ibis (*see Phimosus infuscatus*)
Barnacle Goose (*see Branta leucopsis*)
Barnardius, 465
Barnardius barnardi (Mallee Ringneck), 400
Barnardius zonarius (Port Lincoln Parrot), 400, 464, 476
Bar-headed Goose (*see Anser indicus*)
Bar-shouldered Dove (*see Geopelia humeralis*)
Bar-tailed Godwit (*see Limosa lapponica*)
Barn Owl (*see* Tyto alba)
Barn Swallow (*see Hirundo rustica*)
Barred Owl (*see Strix varia*)
Barrow's Goldeneye (*see Bucephala islandica*)
Baruscapillaria spp., 463
Baruscapillaria belopolskaiae, 476
Baruscapillaria calliopsis, 482
Baruscapillaria cincli, 479
Baruscapillaria corvorum, 480, 482
Baruscapillaria emberizae, 479, 481, 484
Baruscapillaria falconis, 470, 471, 472, 477, 487–488
Baruscapillaria grallinae, 481
Baruscapillaria inflexa, 479, 482, 485
Baruscapillaria jaenschi, 467, 476
Baruscapillaria mergi, 466, 467, 468, 469, 470
Baruscapillaria montevidensis, 473
Baruscapillaria obsignata, 463, 465, 466, 470, 487, 488, 491
Baruscapillaria ovopunctata, 485
Baruscapillaria resecta, 480, 481, 490
Baruscapillaria rudolphii, 467
Baruscapillaria ryjikovi, 466
Baruscapillaria spilculata, 466, 467
basophilic cytoplasmic inclusions, 191
basophils, 21
baylisascariasis, 424
Baylisascaris spp.
Baylisascaris columnaris, 425
Baylisascaris laevis, 425
Baylisascaris melis, 425
Baylisascaris procyonis, 425, 427
clinical signs, 426
diagnosis, 425–426
domestic animal health concerns, 428
epizootiology, 425
etiology, 424–425
host range and distribution, 425
host range of larvae (larva migrans) of species of, 426
immunity, 427–428
pathology, 427
public health concerns, 428
synonyms, 424
treatment and control, 428
wildlife population impacts, 428
Baylisacaris columnaris larva, cross section of, 427
Baylisacaris procyonis, live specimen (L3 stage) of, 427
Baylisascaris transfuga, 425, 427
Bean Goose (*see Anser fabalis*)
Belted Kingfisher (*see Megaceryle alcyon*)
benign nature, of parasites, 5
Bennettinia, 37, 39
benzimidazole anthelmintics, 352
Besnoitia infections, 26
Besra (*see Accipiter virgatus*)
Bilharziella, 231, 246, 247, 249, 251
Bilharziella polonica, 251
Bilharziella yokogawai, 246
bird–filarioid relationships, 448
bird mange, 527
Bithynia tentaculata (Faucet Snail), 226–227, 240

Biziura lobata (Musk Duck), 358
Black-billed Parrot (*see Amazona agilis*)
Black-billed Wood-Dove (*see Turtur abyssinicus*)
Black Crowned Crane (*see Balearica pavonina*)
Black Eagle (*see Ictinaetus malayensis*)
black flies (*see Simulium spp*)
  clinical signs, 541
  diagnosis, 541–542
  distribution, 537–538
  epizootiology, 539–540
  etiology, 538–539
  history, 537
  host range, 538
  immunity, 542
  pathogenesis, 540–541
  public and domestic animal health concerns, 542
  synonyms, 537
  treatment and control, 543–544
  wildlife population impacts, 542–543
Black Grouse (*see Tetrao tetrix*)
Black Kite (*see Milvus migrans*)
Black Noddy (*see Anous minutus*)
Black Scoter (*see Melanitta nigra*)
Blackhead disease, 154
Black-necked Swan (*see Cygnus melancoryphus*)
Black-shouldered Kite (*see Elanus caeruleus*)
Black-and-white Warbler (*see Mniotilta variai*)
Black-billed Capercaillie (*see Tetrao parvirostris*)
Black-billed Cuckoo-Dove (*see Macropygia nigrirostris*)
Black-billed Magpie (*see Pica hudsonia*)
Black-crowned Night Heron (*see Nycticorax nycticorax*)
Black-fronted Piping-Guan (*see Pipile jacutinga*)
Black-headed Ibis (*see Threskiornis melanocephalus*)
Black-necked Aracari (*see Pteroglossus aracari*)
Black-winged Stilt (*see Himantopus himantopus*)
Black Swan (*see Cygnus atratus*)
Black Vulture (*see Coragyps atratus*)
Black-winged Lorry (*see Eos cyanagenia*)
Blackburnian Warbler (*see Dendroica fusca*)
Blattella germanica (cockroaches), 378
Black Francolin (*see Francolinus froncolmus*)
Black Stork (*see Ciconia nigra*)
Black-billed Whistling Duck (*see Dendrocygna autumalis*)
Black-faced Cormorant (*see Phalacrocorax fuscescens*)
Black-faced Cuckoo-shrike (*see Coracina novaehollandiae*)
Black-headed Gull (*see Larus ridibundus*)
Black-tailed Native-hen (*see Gallinula chloropus*)
blood-feeding ectoparasites, 4, 108, 533, 577
Blood Pheasant (*see Ithaginis cruentas*)
blood-stained diarrhea, 228
Blossom-headed Parakeet (*see Psittacula roseata*)
blow fly (*Protocalliphora braueri*) larvae, 5
Blue-and-yellow Macaw (*see Aca ararauna*)
Bluebonnet (*see Northiella haematogaster*)
Blue-crowned Paraked (*see Aratinga acuticaudata*)
Blue Dacnis (*see Dacnis cayana*)
Blue Crane (*see Arithropodes paradiseus*)
Blue Duck (*see* Hymenolaimus malacorhynchos)
Blue Eared-Pheasant (*see Crossoptilon auritum*)
Blue-and-yellow Macaw (*see Ara arauna*)
Blue-black Grassquit (*see Volatina jacarina*)
Blue-headed Parrot (*see Pionus menstruus*)
Blue-fronted Parrot (*see Amazona aestiva*)
Blue-gray Tanager (*see Thraupis episcopus*)
Blue Grouse (*see Dendragapus obscurus*)
Blue Jay (*see Cyanocitta cristata*)
Blue Rock-thrush (*see Monticola solitorius*)
Blue-spotted Wood-Dove (*see Turtur afer*)
Blue-winged Macaws (*Primolius maracana*)
Blue-winged Teal (*see Anas discors*)
Blythipicus rubiginosus (Maroon Woodpecker), 478
Boat-Tailed Grackle (*see Quicalus major*)
Bombycillidae (Waxwings), 60, 435, 444
Bonasa, 465
  Bonasa bonasia (Hazel Grouse), 360, 391, 396
  Bonasa umbellus (Ruffed Grouse), 20, 155, 167, 226, 327, 331, 388, 393, 397, 529
Bonelli's Eagle (*see Aquila fasciata*)
Boopidae, 516
Booted Eagle (*see Aquila pennatus*)
borrachudo, 537
Botaurus, 465
  Botaurus lentiginosus (American Bittern), 295
  Botaurus pinnatus (Pinnated Bittern), 295
  Botaurus stellaris (Great Bittern), 328
bothridia, 262
Bothrigaster, 229
  Bothrigaster variolaris, 236
  Bothrigaster variolaris (Cyclocoelidae), 233
Bothrops atrox (Fer-de-lance), 302
Bothrops jararaca (Jararaca), 302
Bourke's Parrot (*see Neaphema bourkii*)
Brachylaemidae, 229, 237
Brachylecithum mosquensis, 238
Brachypteraciidae (Ground-Rollers), 443
bradyzoites, 113, 210, 211
brain, 450
Brant (*see Branta bernicla*)
Branta, 465
  Branta canadensis (Canada Goose), 68, 83, 166, 167, 173, 174, 199, 205, 233, 236, 328, 347–348, 348, 355, 356, 362, 389
    annual mortality of, 93
    infection with *Amidostomum* sp. in the gizzard of, 369
  Branta bernicla (Brant), 69, 250, 328, 356, 362
  Branta hutchinsii (Cackling Goose), 69
  Branta leucopsis (Barnacle Goose), 208, 328, 356, 362, 389
  Branta ruficollis (Red-breasted Goose), 328, 346, 359
  Branta sandvicensis (Hawaiian Goose), 217, 328, 358, 389
Brewer's Blackbird (*see Euphagus cyanocephalus*)
Broad-winged Hawk (*see Buteo platypterus*)
bronchitis, 350
Brotogeris chrysoptera (Golden-winged Parakeet), 399
Brotogeris sanctithomae (Tui Parakeet), 400
Brown Dipper (*see Cinclus pallasii*)
Brown Eared-Pheasant (*see Crossoptilon mantchuricum*)
Brown Pelican (*see Pelecanus occidentalis*)
Brown Snake-Eagle (*see Circaetus cinereus*)
Brown-headed Cowbird (*see Molothrus ater*)
Brown-throated Parakeet (*see Aratinga pertinax*)
Bruneria brunnea (grasshoppers), 402
Brucella abortus vaccine, 199
B-2 (sodium 2,5-dichloro-4-bromophenol), 255
Bubo spp.
  Bubo africanus (Spotted Eagle-Owl), 133
  Bubo bubo (Eurasian Eagle-Owl), 133, 401, 477, 550
  Bubo scandiacus (Snowy Owl), 21, 24, 115, 346, 348
  Bubo virginianus (Great Horned Owl), 21, 133, 206, 213, 477, 541
Bubulcus ibis (Cattle Egret), 208, 235, 294
Bucconidae (Puffbirds), 61, 443

Bucephala, 115, 465
Bucephala albeola (Bufflehead), 173, 330, 356
Bucephala clangula (Common Goldeneye), 67, 166, 173, 227, 297, 362, 469
Bucephala islandica (Barrow's Goldeneye), 330, 356
Bucerotidae (Hornbills), 60, 443
Budgerigar (*see Melopsittacus undulates*)
Bufflehead (*see Bucephala albeola*)
buffalo gnat, 537
bumble foot, 527
buparvaquone, 28
Burhinidae (Thick-Knees), 442
Burnished-buff Tanager (*see Tangara cayana*)
bursa of knee, 450
bursate nematodes, 317
Bush birds, 505
Buteo spp., 465
Buteo brachypterus (Modagascar Buzzard), 74
Buteo buteo (Eurasian Buzzard), 74, 114, 131, 205, 208, 346, 471
Buteo galapagoensis (Galapagos Hawk), 516
Buteo jamaicensis (Red-tailed Hawk), 75, 114, 131, 205, 471, 490
Buteo lagopus (Rough-legged Hawk), 75, 346
Buteo lineatus (Red-shouldered Hawk), 75, 131, 205, 237, 471
Buteo nitidus (Gray Hawk), 131
Buteo platypterus (Broad-winged Hawk), 75, 346
Buteo regalis (Ferruginous Hawk), 75
Buteo rufinus (Long-Yegged Buzzgrd), 74
Buteo rufofuscus (Jackal Buzzard), 74
Buteo swainsoni (Swainson's Hawk), 75
Butorides striata (Striated Heron), 115
Butorides virescens (Green Heron), 294

Cacatua spp.
Cacatua ducorpsii (Ducorps' Cockatoo), 398
Cacatua leadbeateri, (Pink Cockatoo), 394
Cacatua sulphurea (Yellow-crested Cockatoo), 398
Cacatuidae (Cockatoos), 59, 442
Cackling Goose (*see Branta hatehinsii*)
Cairina, 465
Cairina moschata (Muscovy Duck), 23, 163, 233, 249, 250, 329, 338, 390, 395
Cairina scutulata (White-winged Duck), 329
calcaneal region, 450
Calidris, 465
Calidris alpina (Dunlin), 476
Calidris canutus (Red Knot), 238, 475
Calidris minutilla (Least Sandpiper), 384
Calidris maritima (Purple Sandpiper), 345
California Quail (*see Callipepla californica*)
Callaeidae (Wattlebirds), 445
Callipepla, 465
Callipepla californica (California Quail), 331, 390, 451
Callipepla squamata (Scaled Quail), 395
Calliphora spp., 550, 553
Calliphoridae (bottle flies), 548, 550
Calliphoridae (*Protocalliphora* spp.), 547
Callonetta leucophrys (Ringed Teal), 329
Calodium, 463
cambendazole, 371
Camnula pellucida (grasshoppers), 402
Campanulotes compar, 519
Campephagidae (Cuckoo-shrikes), 61, 444
Canada Goose (*see Branta canadensis*)
Canadian Sandhill Crane (*see Grus canadensis rowani*)
canaries (*see Serinus canaria*)
Candida sp., 140
canker, 120
Capella, 465
Cape Shoueter (*see Anas smithii*)
Cape Teal (*see Anas capensis*)
Canvasback (*see Aythya volisineria*)
Cape Barren Goose (*see Cereopsis novaehollandiae*)
capillariasis, 463, 491
Capillaria spp., 140, 463, 464
Capillaria anatis, 370, 463, 465, 466, 482
Capillaria avellari, 468
Capillaria avicola, 481
Capillaria brasiliana, 468
Capillaria cecumitis, 475, 476
Capillaria contorta, 471, 472
Capillaria corvorum, 480
Capillaria droummondi, 470
Capillaria ellisi, 470
Capillaria freitasi, 482
Capillaria fulicae, 475
Capillaria gigantoteca, 470
Capillaria graucalina, 480
Capillaria gymnorhinae, 481
Capillaria herodiae, 468
Capillaria longistriata, 478
Capillaria newzealandica, 477
Capillaria nyrocinarum, 468, 469, 470
Capillaria ornate, 479
Capillaria parvumspinosa, 466
Capillaria phasianiana, 474, 489
Capillaria phasianina, 473
Capillaria pirangae, 483
Capillaria plagiaticia, 476
Capillaria pomatostomi, 483
Capillaria pudendotecta, 470
Capillaria recurvirostrae, 476
Capillaria resectum, 482
Capillaria rigidula, 482, 483
Capillaria similis, 484, 485
Capillaria spinulosa, 468, 469
Capillaria strigis, 477
Capillaria tenuissima, 470, 471, 472, 477, 487–488
Capillaria uropapillata, 474
Capillaria vasi, 476
Capillaria venusta, 478, 479
capillarid nematodes
in avian hosts, 466–485
clinical signs, 488
diagnosis, 489–490
domestic animal health concerns, 491
eggs of, 464
epizootiology, 464–488
etiology, 464
female sex bias of, 464
genera of birds infected with cosmopolitan, 465
history, 463–464
host range and distribution, 464
immunity, 490–491
larvae, 464
pathology, 488–489
public health concerns, 491
synonyms, 463
taxonomy and genera, 463
treatment and control, 491
wildlife population impacts, 491
Capillostrongyloides, 463

Capitonidae (Barbets), 60, 443
Caprimulgidae (Nightjars), 59, 443
Caprimulgiformes, 14, 59, 163, 443, 478
Caprimulgus macrurus (Large-tailed Nightjar), 478
Carcharhinus perezi, 466
Cardinalidae (Saltators and Cardinals), 60
Cardinalis cardinalis (Northern Cardinal), 112, 116, 327, 333, 378
Cardiofilaria spp., 440, 441–445, 448, 454
  Cardiofilaria nilesi, 446, 451, 455
  Cardiofilaria pavlovskyi, 455
Carduelis chloris (European Greenfinch), 111, 121, 207, 479
Carduelis tristis (American Goldfinch), 112
Carduiceps clayae, 521
Cariama cristata (Red-legged Seriema), 397, 406
Cariamidae (Seriemas), 442
carnidazole, 144
carotenoid-based coloration, by tits, 5
Carrion Crow (*see Corvus corone*), 207
Caryospora, 163
Caryothraustes poliogaster (Black-faced Grosbeak), 333
Caspian Snowcock (*see Tetroogallus caspius*)
Casuariidae (Cassowaries), 441, 505
Casuariiformes, 163, 441
Catatropis, 231
Catatropis verrucosa, 226
Cathaemasia, 229
Cathaemasia hians, 235
Cathaemasiidae, 229, 235
Cathartes aura (Turkey Vulture), 79, 208
Cathartidae (New World Vultures), 59, 441
Catharus minimus (Gray-cheeked Thrush), 479
Catreus wallichi (Cheer Pheasant), 390
Cattle Egret (*see Bubulcus ibis*), 208
cecal coccidiosis (*Eimeria colchici*), 172
cecal nematode (*Trichostrongylus tenuis*), 5
cecal strongyle, 316
cecal threadworm, 316
cell-mediated immunity (CMI), 199
cell necrosis, in liver, 21
centrid, 419
Centrocercus urophasianus (Greater Sage-Grouse), 6, 167, 169, 391
Centropus sinensis (Greater Coucal), 401
Cepphus columba (Pigeon Guillemot), 332
Ceratopogonidae, 451
ceratopogonid (*Culicoides*) flies, 13, 15, 18–19
ceratopogonid vectors, 14
cercariae, 248
Cercomonas gallinae, 121
Cercomonas hepaticum, 134
cerebrospinal (or cerebellar or cerebral) nematodiasis, 424
Cereopsis novaehollandiae (Cape Barren Goose), 356, 389
Ceriodaphnia spp., 355
Certhia familiaris (Eurasian Treecreeper), 207
Certhiidae (Creepers), 60, 444
Cestodes, 4, 261
  causing pathology and mortality in wild birds, 264–265
  clinical signs, 267
  diagnosis, 270
  domestic animal health concerns, 271
  epizootiology
    cyclophyllidean eggs, 265–266
    Diphyllobothriid life cycles, 267
    Tetrabothriidea, 266
  etiology, 262–263
  geographic distribution of, 263–265
  history, 262
  host range, 263
  immunity to infections, 270
  infections in abnormal sites, 269
  life cycles, 265–266
  management implications, 272
  pathology of
    Cyclophyllidea, 267–269, 269–270
    Diphyllobothriidae, 269, 270
    larval cestodes, 269–270
    Tetrabothriidea, 269
  public health concerns, 270–271
  reproductive systems, 263
  synonyms, 262
  transmission with aquatic life cycles, 263
  treatment and control, 272
  wildlife population impacts, 271–272
Cestodiasis, 262
Ceylon Junglefowl (*see Gallus lafayetii*)
Chaffinch (*see Fringilla coelebs*)
Chaimarrornis leucocephalus (White-capped Redstart), 480
Chandlerella spp., 440, 441–445, 445, 446, 448, 452, 454
  Chandlerella alii, 449
  Chandlerella apusi, 449
  Chandlerella bosei, 449
  Chandlerella buckleyi, 449
  Chandlerella bushi, 449, 457
  Chandlerella chitwoodae, 446, 449, 451
  Chandlerella columbae, 449
  Chandlerella columbigallinae, 449
  Chandlerella hepatica, 449
  Chandlerella himalayansis, 449
  Chandlerella inversa, 449
  Chandlerella lerouxi, 449
  Chandlerella lienalis, 449
  Chandlerella longicaudata, 449
  Chandlerella pelecani, 449
  Chandlerella petrowi, 450
  Chandlerella quiscali, 446, 450, 451, 458
  Chandlerella robinsoni, 450
  Chandlerella shaldybini, 449
  Chandlerella sinensis, 449
  Chandlerella singhi, 449
  Chandlerella skrjabini, 449
  Chandlerella stantchinski, 449
  Chandlerella striatospicula, 445, 446, 449, 451
  Chandlerella sultana, 449
  Chandlerella thapari, 449
Changeable Hawk-Eagle (*see Spizaetus cirrhatus*)
Channel-billed Toucan (*see Ramphastos vitellinus*)
Charadriidae (Plovers and Lapwings), 59, 361, 442, 505
Charadriiformes, 14, 59, 121, 163, 175, 205, 208, 238, 247, 251, 253, 263, 291, 376, 377, 435, 465, 475, 521
Charadrius, 465
  Charadrius alexandrinus (Snowy Plover), 331
  Charadrius hiaticula (Common Ringed Plover), 475
  Charadrius vociferus (Killdeer), 476
Chaunocephalus, 230
Chaunocephalus ferox, 235
Cheer Pheasant (*see Catreus wallichi*)
Cheilospirura nasuta, 327
chemotherapy, 285
Chen spp., 465
  Chen caerulescens (Snow Goose), 69, 167, 173, 174, 348, 355, 359, 365, 390

Chen caesulescens caerulescens (Lesser Snow Goose), 167, 174
Chen canagica (Emperor Goose), 69, 328, 363
Chen rossii (Ross' Goose), 173, 355, 359, 365, 390
Chenonetta jubata (Maned Duck), 390
Chestnut-cheeked Starling (*see Sturnia philippensis*)
Chestnut Teal (*see Anas castanea*)
chewing lice
clinical signs, 518–520
diagnosis, 520
epizootiology, 518
etiology, 516–518
history, 515
host defense and immunity, 520–522
host range and distribution, 515–516
pathogenesis and pathology, 518–520
public and domestic health concerns, 522
synonyms, 515
treatment and control, 522
wildlife population impacts, 522–523
Chinese Francolin (*see Francolinus pintadeanus*)
chicken, domestic, infections, 54
chickens, with infections of *Eimeria*, 163
Chilean Flamingo (*see Phoenicopterus chilensis*), 328, 338
Chiloe Wigeon (*see Anas sibilatrix*)
Chimango Caracara (*see milvago chimango*)
Chinese Goshawk, 78
Chinese Pond-heron (*Ardeola bacchus*)
Chionididae, 442
Chlamydotis undulate (Houbara Bustard), 261, 393
Chlidonias, 465
Chlidonias hybrida (Whiskered Tern), 476
Chloebia gouldiae (Gouldian Finch), 195
Chloephaga picta (Upland Goose), 360, 390
Chloephaga poliocephala (Ashy-headed Goose), 356, 389
Chloropseidae (Leafbirds), 60
chloroquine phosphate, 48
chloroquine sulfate, 28
Choanotaenia, 264
Choanotaenia infundibulum, 264, 268, 269, 271
Chrysolophus, 465
Chrysolophus amherstiae (Lady Amherst's Pheasant), 392
Chrysolophus pictus (Golden Pheasant), 331, 391, 473
Chtonobdella bilineata, 505
Chtonobdella limbata, 505
Chukar (*see Alectoris chuckar*)
Cicinnurus magnificus (Magnificent Bird-of-paradise), 345
Ciconia spp.
Ciconia ciconia (white short), 235, 550
Ciconia maguari (Maguori Stork), 295, 389
Ciconia nigra (Black Stork), 235
Ciconiidae (Storks), 59, 441
Ciconiiformes, 14, 35–36, 59, 163, 208, 235, 247, 251, 253, 293, 306, 328, 441, 465, 468
Cinclidae (Dippers), 61, 444
Cinclus pallasii (Brown Dipper), 479
Cinereous Vulture (*see Aegypius monachus*)
Cinnamon Teal (*see Anas cyanoptera*)
cintrin, 371
Circaetus cinereus (Brown Snake-Eagle), 75
Circaetus gallicus (Short-toed Eagle), 74, 471
Circulatory system, 449
Circus spp.
Circus aeruginosus (Western Marsh-Harrier), 79, 130, 237, 346, 471
Circus cyaneus (Northern Harrier), 78, 237, 346
Circus macrourus (Pallid Harrier), 78, 205
Circus pygargus (Montagu's Harrier), 78, 471
Circus spilonotus (Eastern Marsh-Harrier), 395
Cladotaenia, 265
Clangula, 465
Clangula hyemalis (Long-tailed Duck), 66, 66 173, 227, 227, 267, 297, 330, 358, 364, 469
Clay-colored Robin (*see Turdus grayi*), 208
Clelia clelia (Mussurana), 303
Climactreridae (Australian Treecreepers), 61, 444
Clinostomidae, 229
Clinostominae, 235
Clinostomum, 229
Clinostomum attentuatum, 235
Clinostomum complanatum, 226
cloaca (*Leucochloridium*), 226
Cloacitrema, 231
Cloacotaenia megalops, 264, 269
clopidol, 177
clorsulon, 424
Clostridium perfringens, 157, 170
Cnephia ornithophilia, 83, 85, 543
Coal Tit (*see Periparus ater*), 376
Coccidea, 57
Coatinga Parakeet (*see Aratinga cactorum*)
Coccidia, 162, 164
renal, 173, 175, 176
coccidian parasite, 204
coccidiasis, 162
coccidiosis, 3, 108, 111, 116, 165, 181
control of, 172
diagnosis, 170
renal, 175
coccidiostat, 192–193
Coccidium truncatum, 162
Coccothraustes vespertinus (Evening Grosbeak) 109, 527
Cockatiel (*see Nymphicus hollandicus*)
Coco, Heron (*see Ardea cocoi*)
Cochlearius, 465
cocoons of Glossiphoniidae, 503
Coerulea coerulea ("purple sugarbird"), 376
Colaptes auratus (Northern Flicker), 332, 478
Colaptes maximus (*see Piculus rubiginosus*)
Coliidae (Mousebirds), 59, 443
Coliiformes, 14, 59, 163, 443
Colinus, 465
Colinus virginianus (Northern Bobwhite), 13, 21, 23, 36, 155, 156, 163, 167, 169, 195, 213, 280, 316, 320–321, 327, 331, 378, 388, 390, 395, 426, 487
Colius erythromelon (Red-faced Mouse Bird), 114
Collyriclum, 231
Collyriculum faba, 226, 238
Colpocephalum holzenthali, 517
Colpocephalum turbinatum, 516, 521
Coluber constrictor (Blue Racer), 302
Columba spp., 168, 465
Columba arquatrix (Rameron Pigeon), 70, 398
Columba eversmanni (Pale-backed Pigeon), 70
Columba guinea (Speckled Pigeon), 70
Columba larvata (Lemon Dove), 70
Columba livia (Rock Pigeon), 13, 22, 70, 113, 120, 122, 139, 168, 172, 204, 205, 208, 327, 332, 381, 398, 402, 464, 488, 501, 519, 520, 534
Columba oenas (Stock Dove), 70
Columba palumbus (Common Wood Pigeon), 20, 22, 70, 124, 205, 398
Columba vitiensis (Metallic Pigeon), 70

Columbicola columbae, 517, 519
Columbidae (Pigeons and Doves), 57, 59, 361, 435, 442
Columbiformes, 14, 18, 22, 23, 59, 163, 168, 169, 205, 208, 217, 238, 253, 435, 442, 465, 476, 548
Columbiformes, 114
Columbina spp.
Columbina picui (Picui Ground-Dove), 398
Columbina inca (Inca Dove), 129, 144
Columbina passerina (Common Ground-Dove), 129
Columbina talpacoti (Ruddy Ground Dove), 71, 206, 208, 238, 398
Comb Duck (*see Sarkidiornis melanotas*)
Common Crane (*see Grus grus*)
Common Cuckoo (*see Cuculus canorus*)
Common Eider (*see Somateria mollissima*)
Common Goldeneye (*see Bucephala clangula*)
Common Grackle (*see Quiscalus quiscula*)
Common Greenshark (*see Tringa nebularia*)
Common Ground-Dove (*see Columbina passerina*)
Common Hill Myna (*see Gracub religiosa*)
Common Loon (*see Gavia immer*)
Common Merganser (*see Mergus merganser*)
Common Moorhen (*see Gallinula chlosopus*)
Common Murre (*see Uria aalge*)
Common Nightingale (*see Luscinia megarhynchos*)
Common Pochard (*see Aythya ferina*)
Common Quail (*see Coturnix coturnix*)
Common Raven (*see Corvus corax*)
Common Redshark (*see Tringa totonus*)
Common Ringed Plover (*see Charadrius hiaticula*)
Common Sandpiper (*see Actitis hypoleucos*)
Common Shelduck (*see Tadorna tadorna*)
Common Snipe (*see Gallmago gallinago*)
Common Swift (*see Apus apus*)
Common Tern (*see Sterna hirundo*)
Common Wood Pigeon (*see Columba palumbus*)
connective tissues, 449
Conopophagidae (Gnateaters), 443
contracaeciasis, 413
Contracaecum spp., 339, 420
caudal features of diagnostic interest in adult male, 420
clinical signs, 421
Contracaecum bioccai, 420
Contracaecum magnipaillatum, 416
Contracaecum magnipapillatum, 416, 419, 420
Contracaecum microcephphalum, 420
Contracaecum micropapillatum, 420
Contracaecum multipapillatum, 416, 418, 420, 423, 424
Contracaecum multipapillatum sensu lato, 423
Contracaecum ogmorhini, 415
Contracaecum ogmorhini sensu lato, 420
Contracaecum osculatum, 413
Contracaecum pelagicum, 416, 420
Contracaecum radiatum, 420
Contracaecum rudolphii, 415, 416, 418
Contracaecum rudolphii sensu lato, 416, 418, 420, 421
Contracaecum septentrionale, 416, 420
Contracaecum spiculigerum, 424
Contracaecum tricuspe, 420
Contracaecum variegatum, 414, 416
Contracaecum variegatum sensu lato, 420
diagnosis, 419–421
domestic animal health concerns, 423
epizootiology, 416–419
etiology, 413–416
host range and distribution, 416
host range of species of, 417
immunity, 421–423
lesions caused by, 421
pathogenesis and pathology, 421
public health concerns, 423
synonyms, 413
treatment and control, 424
wildlife population impacts, 424
Cooper's Hawk (*see Accipites cooperii*)
copepods, 385
Copperhead (*Agkistrodon contortrix*), 302
Copper Pheasant (*see Syrmaticus soemmerringii*)
copper sulfate, 255, 340
Coracias garrulus (European Roller), 332
Coraciidae (Rollers), 60, 443
Coraciiformes (Kingfishers), 14, 60, 163, 238, 252, 332, 377, 443, 548
Coracina novaehollandiae (Black-faced Cuckoo-shrike), 480
Coragyps atratus (Block Vulture), 79
Corcoracidae (Choughs and Apostlebirds), 61
cormorants (*see Phalacrocorax* spp.)
Cortrema, 229
Cortrematidae, 229
Corvidae (Crows, Jays, and Magpies), 60, 435, 445, 505
Corvus spp., 263, 465
Corvus brachyrhynchos (American Crow), 207, 209, 327, 333, 450, 451, 454, 480
Corvus corax (Common Raven), 480
Corvus cornix (Hooded Crow), 501
Corvus corone (Carrion Crow), 207, 226, 345, 480
Corvus frugilegus (Rook), 207, 345, 346, 347, 480
Corvus hawaiiensis (Hawaiian Crow), 42–43, 204, 218
Corvus monedula (Eurasian Jackdaw), 207, 345, 346, 480, 490
Corvus nasicus (Cuban Crow), 333
Corvus ossifragus (Fish Crow), 110
Corynosoma constrictum, life cycle of, *279*, 280
Corynosoma species, 277, 279
costs to host, against parasitism, 4, 6
Cotingidae (Cotingas), 443
Cotton Pygmy-goose (*see Nettapus coromandelianus*)
Coturnix, 465
Coturnix coturnix (Common Quail), 331, 391, 396
Coturnix japonica (Japanese Quail), 155, 156, 169, 196, 213
Cotylurus, 231
coughing, 197
counter-immunoelectrophoresis, 92
Cracidae (Guans, Chachalacas, and Curassows), 59, 442
Cracticidae (Bellmagpies), 61, 445, 505
Crax blumenbachii (Red-billed Curassow), 390
Creatophora cinerea (Wattled starling), 111
Crested Auklet, (*see Aethia cristatella*)
Crested Fireback (*see Lophura ignita*)
Crested Flycatcher (*Myiarchus crinitus*), 528
Crested Goshawk (*see Accipiter trivirgatus*)
Crested Guineafowl (*see Guttera pucherani*)
Crested Pigeon (*Geophaps lophotes*), 70
Crested Partridge (*see Rollulus rouloul*)
Crickets (*Acheta domestica*), 378
Crimson-backed Tanager (*see Rampholcelus dimidiatus*), 209
Crimson Rosella (*see Platycercus elegans*)
crisis period, 40
Crocodylocapillaria, 463
cropworm, 463

Crossoptilon, 465
Crossoptilon auritum (Blue Eared-Pheasant), 390
Crossoptilon mantchuricum (Brown Eared-Pheasant), 390
Crotophaga ani (Smooth-billed Ani), 332
crows (*see Corvus* spp)
crustaceans, 340
Cryptocotyle, 230
Cryptocotyle lingua, 237
cryptosporidiosis, 197, 198, 200
Cryptosporidium spp., 163
clinical signs, 197–198
Cryptosporidium andersoni, 195
Cryptosporidium anserinum, 195
Cryptosporidium baileyi, 195
in chickens, 199
infections, in humans, 200
life cycles of, 196
Cryptosporidium blagburni, 195, 199
Cryptosporidium galli, 195
Cryptosporidium meleagridis, 195, 199
infections, in humans, 200
life cycles of, 196
Cryptosporidium muris, 195
Cryptosporidium parvum, 195
Cryptosporidium tyzzeri, 195
diagnosis, 198–199
domestic animal health concerns, 200
epizootiology, 196–197
etiology and host range, 195–196
history, 195
immunity, 199
pathology, 198
public health concerns, 199–200
synonym, 195
treatment and control, 200
wildlife population implications, 200
cryptozoites, 39
Crypturellus spp.
Crypturellus noctivagus (Yellow-legged Tinamou), 389
Crypturellus parvirostris (Small-billed Tinamou), 389
Crypturellus tataupa (Totaupa Tinamou), 389
Crypturellus undulates (Undulated Tinamou), 389
Crypturellus variegates (Variegated Tinamou), 389
Crytocotyle lingua, 239
Ctenodactylus gundi, 204
Cuban Parrot (*see Amazona leucacephala*)
Cuclotogaster heterographus, 518
Cuculidae (Cuckoos), 59, 442
Cuculiformes, 14, 59, 114, 163, 332, 442
Cuculus canorus (Common Cuckoo), 401
Culex spp.
Culex annulus, 41
Culex nigripalpus, 40, 41
Culex pipiens, 41
Culex quinquefasciatus, 40, 41
Culex restuans, 41
Culex saltanensis, 41
Culex sitiens, 41
Culex stigmatasoma, 40
Culex stimatosoma, 41
Culex tarsalis, 40, 41
Culicidae, 451
culicine species, 40
Culicoides spp.
Culicoides arakawae, 85
Culicoides arboricola, 18
Culicoides bottimeri, 17, 18
Culicoides circumscriptus, 85
Culicoides crepuscularis, 18
Culicoides downesi, 18
Culicoides edeni, 18
Culicoides haematopotus, 18
Culicoides hinmani, 18
Culicoides impunctatus, 18
Culicoides knowltoni, 18
Culicoides nubeculosus, 18
Culicoides odibilis, 85
Culicoides-proof facilities, 28
Culicoides schultzei, 85
Culicoides species, 17, 82
Culicoides sphagnumensis, 18
Culicoides stilobezziodes, 18
Culiseta morsitans, 41
Curatrem, 424
Curonian Spit, 86
Cuterebra buccata, 550
Cuterebridae (bot flies), 550
Cyanerpes caeruleus (Purple Honeycreeper), 111, 377
Cyanerpes cyaneus (Red-legged Horeycreeper), 111
Cyanocitta, 465
Cyanocitta cristata (Blue Jay), 19, 21, 22, 327, 333, 454, 489
Cyanocorax yncas (Green Jay), 24
Cyanopica cyanus (Azure-winged Magpie), 474, 481
Cyanoramphus auriceps (Yellow-fronted Parakeet), 218, 401
Cyanoramphus novaezelandiae (Red-fronted Parakeet), 218
Cyathocotyle, 229
Cyathocotyle bushiensis (Cyclocoelidae), 225, 226, 227, 234, 235, 240
Cyathocotylidae, 229, 235
Cyathostoma spp., 351
Cyathostoma americanum, 351
Cyathostoma bronchialis, 344, 347, 349, 350, 351
Cyathostoma (cf. *Syngamus* spp.), 343, 346
Cyathostoma (*Cyathostoma*) spp., 343
Cyathostoma (*Hovorkonema*) spp., 343
Cyathostoma (Hovorkonema) variegatum, 343, 351
Cyathostoma infections, 348
Cyathostoma lari, 343
cyathostome infections, 352
cyathostomes, 344, 349–351
cyathostomiasis, 343
Cyclocoelidae, 229, 235, 236, 238
Cyclocoelum, 229
Cyclocoelum mutabile, 225, 238
Cyclophyllidea, 262, 269–270
cyclophyllidean and diphyllobothriid cestodes, life cycles of, 264
cyclophyllideans, 265–266
cyclophyllidean scolices, 262
Cyclophyllidean sp., 264
cyclophyllidean species, 262
Cyclops sternuus, 384
Cygnus spp.
Cygnus atratus (Black Swan), 328, 356, 389, 470
Cygnus buccinator (Trumpeter Swan), 68, 328, 336, 359, 368
Cygnus columbianus (Tundra Swan), 68, 227, 236, 328, 360, 365, 451, 452, 470, 521
Cygnus cygnus (Whooper Swan), 227, 236, 328, 365, 360, 390

Cygnus spp. (*Cont.*)
Cygnus melancoryphus (Black-necked Swan), 328, 362, 470
Cygnus olor (Mute Swan), 68, 227, 233, 236, 282, 328, 358, 364, 470, 521
Cypsiurus parvus (Palm-Swift), 528
cystacanth, 279
cystic and nodular mite disease, 527
cytochrome *c* oxidase I gene, 239
cytostome, 39

Dabbling Ducks (*Anas* spp.), 115, 236, 367
Dacnis cayana (Blue Dacnis), 111
Daphnia species, 335–336
Dark Chanting Goshawk (*see Meliera metabates*)
Dark-throoted Thrush (*see Turdus rificollis*)
Darter (*see Anhinga melanogaster*)
Davaineidae, 262, 264
decapods, 279
Deegeriella regalis, 516, 521
defense cost, to the host against parasites, 4
Delichon urbicum (House Martin), 7
Demoiselle Crane (*see Anthropoides virgo*)
Dendragapus fuliginosus (Sooty Grouse), 393
Dendragapus obscurus (Dusky Grouse), 331, 390, 396
Dendritobilharzia, 246, 247, 251, 255
Dendritobilharzia larvae, 247
Dendritobilharzia pulverrulenta, 247, 249, 250, 251
Dendrobaena adaiensis, 486
Dendrobaena octaedra, 486
Dendrocolaptidae (woodcreepers), 435, 443
Dendrocopos major (Great Spotted Woodpecker), 478
Dendrocygna spp.
Dendrocygna arborea (West Indian Whistling-Duck), 328, 390
Dendrocygna autumnalis (Black-bellied Whistling-Duck), 356, 362
Dendrocygna bicolor (Fulvous Whistling-Duck), 83, 328, 363
Dendrocygna viduata (White-faced Whistling-Duck), 328
Dendrodrilus rubidus, 486
Dendroica spp.
Dendroica dominica (Yellow-throated Warbler), 481
Dendroica discolor (Prairie Warbler), 333
Dendroica fusca (Blackburnian Warbler), 333, 481
Dennyus hirundinis, 521
dermanyssid mites, 534
Dermatobia hominis, 550
Dessetfilaria, 441–445, 448, 454
Dessetfilaria guianensis, 456
diarrhea, 87, 198
Dicaeidae (Flowerpickers), 61, 444
Dicheilonematinae, 435
diclazuril, 200, 219
Dicranotaenia coronula, 264
Dicrocoeliidae, 226, 229, 237, 238
Dicruridae (Drongos), 61, 445
digenetic trematode, life cycle of, 228
Dilepididae, 262, 264
dimetridazole, 144–145
Dinobdella ferox, 505
Dioctophyma renale, 308
Diomedeidae (Albatrosses), 441
Diphyllobothriidae, 262, 264, 270
Diphyllobothriid cestodes, 271
Diphyllobothriid life cycles, 267
Diphyllobothrium species, 267, 271
Diplostomatidae, 229
Diplostomum, 229
Diplotriaena, 435
clinical signs, 436
diagnosis, 437
Diplotriaena ozouzi, 435
epizootiology, 435–436
etiology, 435
history, 434
host range and distribution, 434–435
pathology, 436
public and domestic animal health concerns, 437
treatment and control, 437
wildlife population impacts, 437
Diplotriaenidae, 435
Diplotriaenoidea, 440
diplotriaenoids, 439
Dirofilariinae, 448
dispharagosis, 326
Dispharagus spp.
Dispharagus crassicauda, 337
Dispharagus nasutus, 327
Dispharagus spiralis, 327
Dispharagus spiralis columbae, 327
Dispharagus tentaculatus, 327
Dispharagus uncinatus, 335
dispharyngosis, 326
Dispharynx, 326
clinical signs, 327–334
diagnosis, 334–335
Dispharynx nasuta, 326, 327–328, 334–335, 339, 380
Dispharynx spiralis, 326
Dispharynx stonae, 327
domestic animal health concerns, 329
epizootiology, 327
etiology, 327
history, 326–327
host range and distribution, 326, 328–333
immunity, 335
pathogenesis and pathology, 334
synonym, 326
treatment and control, 339–340
dispharynxiasis, 326, 339
Disseminated visceral coccidiosis (DVC)
clinical signs, 190
diagnosis, 191–192
domestic animal health concerns, 192
epizootiology, 187–190
etiology, 181–187
history, 181
host range, 181
immunity, 192
management impacts, 193
pathogenesis and pathology, 190–191
public health concerns, 192
species of *Eimeria* associated with, 181
synonyms, 181
treatment and control, 192–193
wildlife population impacts, 192
diving ducks (Aythyini), 367
DNA vaccines, based on the circumsporozoite protein, 48
domestic goose (*see Anser anser*)
doves, infected with *Leucocytozoon* marchouxi, 87, 89, 93
Double-crested Cormosant (*see Phalacrocosax auritus*)
Double-spurred Francolin (*see Francolinus bicalcoratus*)
Dracunculus, 384

Drepanididae (Hawaiian honeycreepers), 444
drepanidotaeniasis, 262
Dromadidae (Crab Plovers), 442
Dromaiidae (Emus), 441
Dromaius novaehollandiae (Emu), 427, 459, 452
droppings, of birds, post malarial infection, 43
Dryadophis bifossatus (Cazadora brown-lined snake), 303
Drymarchon corais (Indigo snake), 302
Dryocopus pileatus (Pileated Woodpecker), 478
Ducorp's Cockatoo (*see Cacatua ducorpsii*)
Ducula spp.
  Ducula bicolor (Pied Imperial-Pigeon), 217
  Ducula carola (Spotted Imperial-Pigeon), 71
  Ducula spilorrhoa (Torresian Imperial-Pigeon), 217
dulfaquinoxaline, 177
Dulidae (Palmchats), 444
Dumetella carolinensis (Gray Catbird), 332
Dunlin (*see Calidris alpina*)
Dunnock (*see Prunella modularis*)
duokvin, 200
Duplicaecum, 413
Dusky Parrot (*see Pionus fuscus*)
Dusky Turtle-Dove (*see Streptopelia lugens*)
dusting, of birds, 28
dwarf mallards, 252
dyspnea, 137, 197, 349

Eared Dove (*see Zenaida auriculata*)
Eared Grebe (*see Podiceps nigricollis*)
earthworms, 352
Eastern Bluebird (*see Sialia sialis*)
Eastern equine encephalitis, 521
Eastern Marsh-Harrier (*see Circus spilonotus*)
Eastern Meadowlark (*see Sturnella magna*)
Eastern mosquitofish (*Gambusia holbrooki*), 290
Eastern Rosella (*see Platycercus eximius*)
Eastern Screech-Owl (*Megascops asio*), 213
Eastern Towhee (*see Pipilo esythrophthalmus*)
Echinochasmus, 230
Echinocoleus, 463
Echinocoleus cyanopicae, 474, 481
Echinoparyphium, 230
Echinoparyphium recurvatum, 236
Echinosotoma spp., 230, 234, 236
  Echinosotoma trivolvis, 236
  Echinostoma revolutum, 226
  Echinostoma trivolvis, 234
Echinostomatidae, 230, 235, 236
Echinostoma trivolvis, 234
echinostomiasis (*Echinostoma* spp.), 239
echinuriasis, 337
Echinuria spp.
  clinical signs, 336
  diagnosis, 337
  domestic animal health concerns, 339
  Echinuria jugadornata, 335
  Echinuria uncinata, 326, 327, 328, 335, 336, 337
  epizootiology, 335–336
  etiology, 335
  history, 335
  host range and distribution, 326, 328–333
  pathogenesis and pathology, 336–337
  synonyms, 335
  treatment and control, 339–340
ectoparasitic hippoboscid flies, 13, 17
ectoparasitic mites, 534

Egretta spp.
  Egretta tricolor (Tricolored Heron), 294, 305
  Egretta caerulea (Little Blue Heron), 115, 468
  Egretta garzetta (Little Egret), 114, 294, 395
  Egretta thula (Snowy Egret), 294, 304, 305, 384
Egyptian Goose (*Alapochen aegyptiaca*)
Egyptian Vulture (*see Neaphron percnoptesus*)
Eiberg strains, 135
Eimeria spp., 108, 166, 168, 174
  common pathogenic intestinal species of, 166–168
  in cranes, life cycle of, 187
  developmental stages of, 164
  Eimeria acervulina, 169
  Eimeria adenoeides, 167, 169, 171
  Eimeria angusta, 167, 169
  Eimeria anseris, 171
  Eimeria auritusi, 173, 174
  Eimeria avium, 162
  Eimeria aythyae, 165, 166
  Eimeria boschadis, 173
  Eimeria bosquei, 187
  Eimeria brunetti, 169
  Eimeria bucephalae, 166
  Eimeria colchici, 168, 169, 170
  Eimeria columbarum, 168
  Eimeria dispersa, 163, 167, 171
  Eimeria duodenalis, 168, 169
  Eimeria dusingi, 168
  Eimeria fraterculae, 175
  Eimeria gallopavonis, 167, 169, 171
  Eimeria goelandi, 175
  Eimeria gruis, 162, 181, 186
  Eimeria gruis, oocysts of, 186
  Eimeria innocua, 169
  Eimeria kotlani, 171
  Eimeria labbeana, 168
  Eimeria lettyae, 167, 169
  Eimeria maxima, 169
  Eimeria meleagridis, 169
  Eimeria meleagrimitis, 167, 169, 171
  Eimeria mitis, 169
  Eimeria mivati, 169
  Eimeria mulardi, 163
  Eimeria necatrix, 169
  Eimeria nocens, 171
  Eimeria phasiani, 168, 169, 170
  Eimeria praecox, 169
  Eimeria procera, 168
  Eimeria reichenowi, 162, 181, 186
  Eimeria reichenowi, oocysts of, 186
  Eimeria saitamae (Domestic Ducks), 171
  Eimeria serventyi, 174
  Eimeria somateriae, 165, 173, 174
  Eimeria stigmosa, 167
  Eimeria subrotunda, 169
  Eimeria taldykurganica, 169
  Eimeria tenella, 169
  Eimeria tropicalis, 187
  Eimeria truncata, 174, 175, 176, 177
  Eimeria tsunodai, 169
  Eimeria uzura, 169
  Eimeria wobeseri, 175
  eported from avian hosts, 163
  hepatic, 177
  infection development, 162
  intestinal
    clinical signs of, 170

Eimeria spp. (*Cont.*)
diagnosis of, 170–171
distribution and host range of, 162–163
domestic animal concerns of, 171–172
epizootiology of, 164–170
etiology of, 163–164
immunity of, 170–171
management implications, 172
pathology of, 170
public health concerns of, 171
treatment and control, 172
wildlife population impacts, 172
renal
clinical signs and pathology, 175
diagnosis, 176
domestic animal health concerns, 177
epizootology, 173
etiology of, 172
host range and prevalence, 173–175
immunity, 176
management implications, 177
public health concerns, 176
treatment and control, 177
wildlife population impacts, 177
sporulated oocyst of, 164
Eimeriidae, 163
Eisenia foetida, 344, 401, 486
Elanus caeruleus (Black-shouldered Kite), 74
Elegant Parrot (*see Neophema elegans*)
Eleonora's Falcon (*see Falco eleonorae*)
Elimia virginica, 227
ELISA test, 26, 46, 92
Emberiza spp.
Emberiza citrinella (Yellowhammer), 206, 401, 481
Emberiza schoeniclus (Reed Bunting), 481
Emberiza sulphurata (Yellow Bunting), 481
Emberiza variabilis (Gray Bunting), 481
Emberizidae (Buntings and Sparrows), 60, 444
embryonation, 401
Emerald-spotted Wood-Dove (*see Turtur chalcospilos*), 73
Emperor Goose (*see Chen canagica*)
Empidonax minimus (Least Flycatcher), 332
Emu (*see Dranaius nevaehoilandiae*), 459
Emys blandingi (Blandings turtle), 303
Encephalitis, 521
endopolygony, 113
energy, as a parameter to parasitism, 4
Enicognathus ferrugineus (Austral Parakeet), 399
enrofloxacin, 200
Eomenacanthus stramineus, 521
Eos bornea (Red Lorry), 217
Eos cyanogenia (Black-winged Lorry), 218
eosinophils, 21
epithelial hyperplasia, 21
epizootics, 27
from avian malaria, 35
of eustrongylidosis, 289
at lower elevations, 48
of trematodes, 225
epomidiostomiasis, 355
Epomidiostomum spp., 369
avian hosts infected with, 362–365
clinical signs, 368
diagnosis, 370
domestic animal health concerns, 370
eggs, 367
epizootiology, 367–368
Epomidiostomum alii, 364, 366
Epomidiostomum asymmetricum, 362, 366
Epomidiostomum crami, 355, 362, 363, 364, 365, 366, 368, 369
Epomidiostomum cygni, 364, 366
Epomidiostomum orispinum, 362, 363, 364, 365, 366
Epomidiostomum penelopi, 362, 366
Epomidiostomum petalum, 364, 365, 366
Epomidiostomum querquedulae, 363, 364, 365, 366
Epomidiostomum sarkidiorni, 362, 366
Epomidiostomum serratum, 363, 364, 365, 366
Epomidiostomum skrjabini, 362, 363, 364
Epomidiostomum subquadratum, 363, 365, 366
Epomidiostomum sultanai, 362, 363, 366
Epomidiostomum uncinatum, 362, 363, 364, 365, 366, 368
Epomidiostomum vogelsangi, 362, 366
etiology, 366–367
history, 355
host range and distribution, 355–366
immunity, 370
management implications, 371
pathogenesis and pathology, 368–370
prevalence, intensity and abundance, 367–368
public health concerns, 370
treatment and control, 371
wildlife population impacts, 371
Erckel's Francolin (*see Francolinus erckelii*)
Erithacus, 465
Erithacus rubecula (European Robin), 22, 207
erythrocytes invasion, 15, 42
erythrocytic meronts, 46
erythrocytic parasitemias, 21
erythrocytic stages, of the parasites, 21
Escherichia coli, 157, 521
Escherichia intermedia, 157
Estrildidae (Waxbills), 60, 444
Eucoccidiorida, 163
Eucoleus spp., 463
Eucoleus annulatus, 463, 464, 465, 466, 487, 488
Eucoleus contortus, 463, 464, 465, 466, 472, 486–487, 487–488, 489
eggs of, 487
stichosome, 486
Eucoleus crypturi, 466
Eucoleus dispar, 470, 471, 472, 477, 479, 480, 482, 484, 487–488, 489
Eucoleus frugilegi, 464
Eucoleus obtusiuscula, 475, 476
Eucoleus penidoi, 466
Eucoleus perforans, 464, 468, 473, 474, 488
Eucoleus suppereri, 472
Eucotyle, 230
Eucotyle nephritica (Eucotylidae), 226
Eucotylidae, 230, 237, 238
Eucyclops serratulus, 384, 385
Eudocimus, 465
Eudocimus albus (White Ibis), 115, 285, 290, 296
Eudyptula minor (Little Penguin), 174, 175, 217, 235, 267
Eufilaria spp., 441–445, 448, 454
Eufilaria bartlettae, 451
Eufilaria kalifai, 451
Eufilaria longicaudata, 214, 445, 446, 456
Eufilari longicaudata, 451
Eulimdana spp., 441–445, 448, 454
Eulimdana bainae, 448, 451, 521

Eulimdana clava, 450, 452
Eulimdana cypseli, 446, 451
Eulimdana metcalforum, 456
Eulimdana wongae, 448, 451, 521
Eumegacetes, 230
Eumegacetidae, 230
Eunectes murinus (Green anaconda), 302
Euphagus cyanocephalus (Brewer's Blackbird), 209
Euphonia violacea (Violaceous Euphonia), 111
Eupodotis ruficrista (Red-crested Bustard), 269
Eurasian Bullfinch (*see Pyrrhula pyrrhula*)
Eurasian Buzzard (*see Buteo buteo*)
Eurasian Capercaillie (*see Tetrao urogallus*)
Eurasian Collared Dove (*see Streptopelia decaocto*)
Eurasian Coots (*see Fulica atra*)
Eurasian Eagle-Owl (*see Bubo bubo*)
Eurasian Hobby (*see Falco subbuteo*)
Eurasian Jackdaw (*see Corvus monedula*)
Eurasian Jay (*see Garrulus glandarius*)
Eurasian Kestrel (*see Falco tinnunculus*)
Eurasian Magpie (*see Pica pica pica*)
Eurasian Nutcracker (*see Nucifraga caryocatactes*)
Eurasian Nuthatch (*see Sitta europaea*)
Eurasian Oystercatcher (*see Haematopotus ostralegus*)
Eurasian Pygmy-Owl (*see Glaucidium passerinum*)
Eurasian Skylark, (*see Alauda arvensis*)
Eurasian Sparrowhawk (*see Accipiter nisus*)
Eurasian Spoonbill (*see Platalea leucorodia*)
Eurasian Teal (*see Anas creccai*)
Eurasian Treecreeper (*see Certhia familioris*)
Eurasian Tree Sparrow (*see Passer montanus*)
Eurasian Turtle-Dove (*see Streptopelia turtur*)
Eurasian Wigeon (*see Anas penelope*)
European Greenfinch (*see Carduelis chloris*)
European Herring Gull (*see Larus argentatus*)
European Honey-Buzzard (*see Pernis apivorus*)
European Robin (*see Erithacus rubecula*)
European Roller (*see Coracias garrulus*)
European Scops-Owl (*see Otus scops*)
European Shag (*see Phalacrocorax aristotelis*)
European Starling (*see Sturais vulgaris*)
Eurylaimidae (Broadbills), 61, 443
Eurypygidae (Sunbittern), 442
Eusimulium geneculare, 85
Eustrongylides spp., 289, 296, 297
avian hosts of, 291–297
diagnostic features of the caudal end of male, 299
diagnostic features of the cranial end of, 299
distribution of, 290
eggs of, 304
environmental factors affecting transmission of infections, 301–304
Eustrongylides acrochordi, 298
Eustrongylides africanus, 298
Eustrongylides excisus, 289, 292, 296, 298, 300, 301, 302, 304, 306, 309
Eustrongylides gadopsis, 298
Eustrongylides ignotus, 289–290, 292–295, 296, 298, 300, 302, 303, 304, 309
egg of, 309
with oligochaete, 301
without oligochaete, 301
Eustrongylides indicus, 298
Eustrongylides mergorum, 298
Eustrongylides papillosus, 298
Eustrongylides phalacrocoracis, 298
Eustrongylides plotinus, 298
Eustrongylides rubrum, 298
Eustrongylides sinicus, 298
Eustrongylides spinispiculum, 298
Eustrongylides tricolor, 298
Eustrongylides tubifex, 289–290, 291–293, 297, 298, 300, 301, 303, 306, 307, 309
Eustrongylides wenrichi, 298
larval of, 301
lesions of infections, 298, 305–307
life cycle, 300
life cycle of, 300
life history of, 298–301
paratenic and accidental hosts of, 302–303
prevalence in infected fish, 304, 308
range of development times in days, 301
range of fish intermediate hosts of, 304
Eustrongylidiasis, 289
Eustrongylidosis, 289, 308
clinical signs, 304–305
diagnosis, 306–308
domestic animal health concerns, 308
epizootiology, 298–304
etiology, 298
history, 289
host range and distribution, 289–298
management implications, 309–310
pathology, 305–306
public health concerns, 308
synonyms, 289
treatment and control, 309
wildlife population impacts, 308–309
Evening Grosbeak (*see Coccothraustes vespertinus*)
evolutionary trap, 7
exflagellation process, 40
exoerythrocytic merogony, of *Plasmodium elongatum*, 35
exoerythrocytic meronts, 39, 40
exoerythrocytic parasites, 42
exsheathment process, 320
extrinsic factors, of parasites, 21
eye infection, with *Philophthalmus* sp., 239

Fabricii (*Prosthogonimus*), 226
Falcated Duck (*see Anas falcata*)
Falco spp.
Falco biarmicus (Lanner Falcon), 132
Falco cenchroides (Australian Kestrel), 80
Falco cherrug (Saker Falcon), 80, 132, 139
Falco chicquera (Red-necked Falcon), 132, 488
Falco columbarius (Merlin), 79, 132, 148
Falco eleonorae (Eleonora's Falcon), 80
Falco mexicanus (Prairie Falcon), 236, 434
Falco naumanni (Lesser Kestrel), 80, 131
Falco peregrinus (Peregrine Falcon), 79, 132, 143, 472, 488, 550
Falco rupicoloides (Greater Kestrel), 80
Falco rusticolus (Gyrfalcon), 132, 237, 488
Falco sparverius (American Kestrel), 28, 80, 205, 472, 550
Falco subbuteo (Eurasian Hobby), 80, 472
Falco tinnunculus (Eurasian Kestrel), 28, 79, 114, 131, 205, 346, 424, 434, 472
Falconidae (Falcons and Caracaras), 57, 59, 441
Falconiformes, 14, 59, 130, 163, 205, 208, 236, 297, 441, 465, 470, 548, 549
Fallisia neotropicalis, 36
Fasciola, 225
Fasciola trachea, 343

faucet snail, 226
feather lice, 517
febantel, 491
febrile paroxysms, in birds, 43
fecal flotation visualization and measurement method, 112
Feckled Duck (*see Stictonetta noevosa*)
female gametocyte. *see* macrogametocytes
fenbendazole, 272, 322, 340, 352, 437, 459, 491
Ferruginous Hawk (*see Buteo regalis*)
Ferruginous Pochard (*see Aythya nyroca*)
Ferruginous Pygmy-Owl (*see Glaucidium brasilianum*)
Festive Parrot (*see Amazovi feativa*)
fertilization, of *Haemoproteus* species, 17
fibrosis, 370, 421
Fieldfare (*see Turdus pilaris*)
Filaria spp.
  Filaria cypseli, 521
  Filaria nasuta, 327
  Filaria uncinata, 335
Filarioidea, 434, 439, 440
filarioid nematodes
  clinical signs, 450
  diagnosis, 454–455
  epizootiology, 445–450
  etiology, 440
  history, 439–440
  host range and distribution, 440–445
  immunity, 455–458
  pathology, 450–454
  public and domestic animal health concerns, 458
  sites in the avian host occupied by adult avian filarioids, by subfamily and genus, 448–450
  treatment and control, 459
  wildlife population impacts, 458–459
filarioids, 439
Filicollis anatis, 285
Fimbriaria fasciolaris, 263, 264, 269
fimbriariasis, 262
finches, 111, 121, 204
Fish Crow (*see Corvus ossifragus*)
Fisher's Lovebird (*see Agapornis fisheri*)
fish parasites (*see Eustrongylides* spp.)
flamingo (*see Phoenicopterus* spp.)
Florida Sandhill Crane (*see Grus canadensis pratensis*)
Florida Scrub Jay (*see Aphelocoma coerulescens*)
Florida Snail Kite (*see Rostrhamus sociabilis plumbeus*)
flubendazole, 352, 371
Fluminicola virens, 227
formalin-killed vaccine, 94
Formicariidae (Antthrushes and Antpittas), 443
Forpus passerinus (Green-rumped Parrotlet), 114, 399
Fox Sparrow (*see Passerella iliaca*)
Francolinus spp.
  Francolinus afer (Red-necked Francolin), 392
  Francolinus ahantensis (Ahanta Francolin), 395
  Francolinus bicalcaratus (Double-spurred Francolin), 391, 396
  Francolinus erckelii (Erekel's Froncolin), 217
  Francolinus francolinus (Black Francolin), 155
  Francolinus gularis (Swamp Francolin), 393
  Francolinus pintadeanus (Chinese Francolin), 390
Fratercula corniculata (Horned Puffin), 332
free-ranging species, challenges in studying, 3
Fregatidae (Frigatebirds), 441
Freitascapillaria, 463
Fringilla coelebs (Chaffinch), 121, 206, 527
Fringillidae (Siskins and Crossbills), 60, 108, 444
Fuadias madagascariensis, 435
Fulica spp., 465
  Fulica americana (American Coot), 208, 226, 227, 233, 238, 336, 361, 378, 445, 446, 451, 475
  Fulica atra (Eurasian Coot), 205, 226, 227, 297, 331, 361, 365, 385, 331
Fulvous Whistling-Duck (*see Dendrocygna bicolor*)
Furnariidae (Ovenbirds), 61, 435, 443

Gabar Goshawk (*see Micronisus gabar*)
Gadwall (*see Anas strepera*)
Galapagos Dove (*see Zenaida galapagoensis*)
Galapagos hawk (*see Buteo galapagoensis*)
Galbulidae (Jacamars), 443
Gallicolumba luzonica (Luzon Bleeding-heart), 19, 23, 217, 332, 398
Galliformes, 14, 18, 23, 59, 121, 136, 163, 165–169, 167, 205, 208, 217, 252, 322, 360, 376, 435, 441, 465, 473, 521
Gallinago, 465
Gallinago gallinago (Common Snipe), 361, 476
Gallinula spp., 465
  Gallinula chloropus (Common Moorhen), 225, 227, 361, 378, 475, 488
  Gallinula ventralis (Black-tailed Native-hen), 361
Gallomec Plus®, 352
Galloperdix spadicea (Red Spurfowl), 392
Gallus spp., 465
  Gallus gallus (domestic chicken), 23, 113, 114–115, 169, 195, 327, 455, 473
  Gallus gallus (Red Jungle Fowl), 155, 331, 392, 396, 521
  Gallus lafayetii (Ceylon Junglefowl), 390
  Gallus sonneratii (Gray Junglefowl), 391
Gambusia holbrooki (Eastern mosquitofish), 310
game-farm pheasants, 134
gametes, 110
gametocytes, 19
  circulating, 36
  development in species of *Leucocytozoon*, 57–58
  of *Haemoproteus meleagridis*, 15
  of *Leucocytozoon simondi*, 83
  mature, morphological forms of the, 16
  morphology of, 15
  of *Plasmodium* species, 39–40
gametogenesis, of *Haemoproteus* species, 17
gametogony, 110, 165, 188
Gammarus lacustris, 335, 337, 378
Ganguleterakis, 394
gapes, 343
gapeworm infection, 343, 347–348
gapeworms, 344, 349–350, 351
Garganey (*see Anas querquedula*)
Garniidae (genus *Fallisia*), 36
Garrulus, 465
Garrulus glandarius (Eurasian Jay), 206, 481
Garter snake (*Thamnophis sirtalis*), 302
Gastrotaenia spp., 261
  Gastrotaenia cygni, 262, 264
  Gastrotaenia dogieli, 262, 264
gastrotaeniasis, 262
Gavia spp.
  Gavia arctica (Arctic Loon), 261, 291, 328
  Gavia immer, 56
  Gavia immer (Common Loon), 228, 291, 466, 538, 542
  Gavia stellata (Red-throated Loon), 291, 328, 360, 466
Gaviidae (Loons), 59, 360, 441, 505
Gaviiformes, 14, 59, 163, 291, 328, 360, 441, 466

Gelochelidon, 465
Geopelia humeralis (Bar-shoulderdd Dove), 73
Geopelia striata (Zebra Dove), 73, 126
Geopetitia aspiculata, 376, 377, 378, 380, 381
Geophaps lophotes (*see* Crested Pigeon)
Geothlypis poliocephala (Gray-crowned Yellowthroat), 333
Geotrygon montana (Ruddy Quail-Dove), 398
giemsa stain, 13, 39, 45, 112, 458
Gigantobilharzia spp., 231, 246, 247, 249, 251
  acotylea, 251
  adami, 251
  ardeola, 251
  gyrauli, 251
  huronensis, 251
  huttoni, 251
  lawayi, 251
  nettapi, 251
  plectropteri, 251
  sturniae, 251
  tantali, 251
Giovannolaia, 37, 39
gizzard infections, 267
gizzard nematodes, 367
gizzard worm, 326, 355, 371
Glareolidae (Pratincdes and Coursers), 442
Glaucidium brasilianum (Ferruginous Pygmy-Owl), 206
Glaucidium passerinum (Eurasian Pygmy-Owl), 394
α_2-globulin, 42
γ_I-and γ_2-globulin, 42
Glossiphoniidae, 505, 506
glossiphoniid leeches, 508
glossiphoniids, 506
Glossy Ibis (see *Plegadis falcinellus*)
glutamate dehydrogenase, 42
γ-glutamyltransferase, 42
gobies (*Neogobius kessleri* and *Neogobius melanostomus*), 298
Golden-crested Myna (*see Ampeliceps coronatus*)
Golden Eagle (*see Aquila chrysaetos*)
Golden-olive Woodpecker (*see Piculus rubiginosus*)
Golden Pheasant (*see Chrysolophus pictus*)
Golden-winged Parakeet (*see Brotogeris chrysoptera*)
Goldeneye (*see Bucephala* spp)
Goliath Heron (*see Ardea goliath*)
Gouldian Finch (*see Chloebia gouldiae*)
Goura spp.
  Goura cristata (Western Crowned-Pigeon), 217
  Goura scheepmakeri (Southern Crowned-Pigeon), 217
  Goura victoria (Victoria Crowned-Pigeon), 217
Gracula religiosa (Common Hill Myna), 114, 170
Grallina cyanoleuca (Magpie-lark), 162, 481
Grallinidae (Mudnest Builders), 61, 445
granulomas, 191
grasshoppers (*Bruneria brunnea*, *Camnula pellucida*, and *Melanoplus* spp.), 402
Grass snake (*Natrix tessellata*), 302
Gray Bunting (*see Embesiza variabilis*)
Gray-cheeked Thrush (*see Catharus minimus*)
Gray Crowned Crane (*see Balearica regulorum*)
Gray-faced Woodpecker (*see Picus canus*)
Gray Hawk (*see Buteo nitidus*)
Gray Heron (*see Ardeo cinerea*)
Gray Junglefowl (*see Gallus Sonneratii*)
Gray partridge (*see Perdix perdix*)
Gray Peacock-Pheasant (*see Polyplectron bicalcaratum*)
Gray Teal (*see Anas gracilis*)
Gray Tinamou (*see Tinamus tao*)
Graylag Goose (*see Anser anser*)
Great Bittern (*see* Botaurus stellaris)
Great Black-headed Gull (*see Larus ichthyaetus*)
Great Blue Heron (*see Ardea hercdias*)
Great Bustard (*see* Otistarda)
Great Crested Grebe (*see Podiceps cristatus*)
Great Egret (*see Ardeo alba*)
Great Horned Owl (*see Bubo virginianus*)
Great Painted-Snipe (*see Rostratula benghalensis*)
Great Spotted Woodpecker (*see Dendrocopos major*)
Great-tailed Grackle (*see Quiscalus mexicanus*)
Great Tit (*see Parus major*)
Greater Coucal (*see Centropus sinensis*)
Greater Kestrel (*see Falco rupicoloides*)
Greater Prairie-Chicken (*see Tympanuchus cupido*)
Greater Rhea (*see Rhea americana*)
Greater Sage-Grouse (*see Centrocercus urophasianus*)
Greater Sandhill Crane (*see Grus canadensis tabida*)
Greater Scaup (*see Aythya marila*)
Greater Spotted Eagle (*see Aquila clonga*)
Greater white-fronted Goose (*see Anser albifrons*)
Greater Yellow legs (*see Tringa melanoleuca*)
Green-and-gold Tanager (*see Tangara schrankii*)
Green-backed Tit (*see Pasus monticolus*)
Green-cheeked Parakeet (*see Pyrrhula molinae*)
Green Heron (*see Butorides virescens*)
Green Jay (*see Cyanocorax yncas*)
Green Peafowl (*see Pavo muticus*)
Green Pheasant (*see Phasianus versicolor*)
Green-rumped Parrotlet (*see* Forpus passerinus)
Green-winged Teal (*see Anas carolinensis*)
Green-Woodpecker (*see Picus viridis*)
grouse disease, 316, 326
Gruidae (Cranes), 59, 442
Gruiformes, 14, 22, 59, 163, 169, 205, 208, 238, 297, 360, 361, 376, 465, 475
gruiforms, 121
Grus spp.
  Grus americana (Whooping Crane), 181, 185, 188, 285, 331, 339, 346, 399, 406, 475
  Grus canadensis canadensis (Lesser Sandhill Crane), 183
  Grus canadensis pratensis (Florida Sandhill Crane), 184
  Grus canadensis pulla (Mississippi Sandhill Crane), 184
  Grus canadensis rowani (Canadian Sandhill Crane), 183
  Grus canadensis (Sandhill Crane), 182, 331, 475
  Grus canadensis tabida (Greater Sandhill Crane), 183, 191
  Grus grus (Common Crane), 346, 397, 475, 550
  Grus japonicus (Red-crowned Crane), 181, 185
  Grus monacha (Hooded Crane), 185, 188
  Grus antigone (Sarus Crane), 184, 397
  Grus vipio (white-naped Crane), 181, 182, 184
Gryporhynchidae, 264, 266
Guanay Cormorant (*see Phalacrocorax bougainvillii*)
Guttera pucherani (Crested Guineafowl), 396
Gymnophallus (Microphallidae), 227, 230
Gymnorhina tibicen (Australasian Magpie), 481
Gymnostinops montezuma (Montezuma Oropendola), 24
Gynaecophila, 381
Gyps africanus (White-backed Vulture), 74, 208
Gyrfalcon (*see Falco rusticolus*)
Gyropidae, 516

Haemadipsa zeylanica, 505
Haemadipsidae, 502, 505, 506, 508
Haemamoeba, 37, 39

Haematomyzidae, 516
Haematopodidae (Oystercatchers), 442
Haematopus ostralegus (Eurasian Oystercatcher), 331, 516
Haementeria depressa, 505
Haementeria medicinalis, 502
Haemogregarina, 108, 109
haemoproteid infections, among the avian orders, 14
Haemoproteids, 14
haemoproteosis, 35
Haemoproteus spp., 6, 23, 24, 35, 40, 46, 54
  clinical signs, 21
  complex life cycle of, 17
  diagnosis, 25–26
  distribution, 14
  domesticated animal health concerns, 27
  epizootiology, 17–21
  etiology, 15–17
  gametocytes of, 16
  Haemoproteus attenuatus, 22
  Haemoproteus balearicae, 22
  Haemoproteus balmorali, 18
  Haemoproteus belopolskyi, 18
    of Blackcaps (*Sylvia atricapilla*), 20
  Haemoproteus coatneyi, 20, 22
  Haemoproteus columbae, 13, 18–19, 22, 23
    of pigeons and doves, 13
    of Rock Pigeons, 26
    transmission by hippoboscid flies, 20
  Haemoproteus danilewskii, 18–19, 22
    Blue Jays with infection of, 21
    of ducks, 20
  Haemoproteus dolniki, 18
  Haemoproteus fringillae, 18
  Haemoproteus halcyonis, 24
  Haemoproteus handai, 18, 23
  Haemoproteus-infected mates, 28
  Haemoproteus lanii, 18
  Haemoproteus lophortyx, 17, 18, 21, 23
    bobwhites with natural infections of, 21
  Haemoproteus maccallumi, 18, 22
  Haemoproteus majoris, 27
  Haemoproteus mansoni, 18
    of Ruffed Grouse (*Bonasa umbellus*), 20
  Haemoproteus meleagridis, 15, 18–19, 21, 23, 25, 26, 27
    gametocytes of, 15
    megalomeront of, 19
    of wild turkeys, 27
  Haemoproteus nettionis, 18, 22
    of ducks, 20
  Haemoproteus noctuae, 21
  Haemoproteus palumbis, 18, 20, 22
  Haemoproteus passeris, 22, 24
  Haemoproteus prognei, 7, 28
  Haemoproteus ptilotis, 22
  Haemoproteus sacharovi, 17, 18, 22, 23
  Haemoproteus syrnii, 21, 24
  Haemoproteus tartakovskyi, 18
  Haemoproteus tinnunculi, 28
  Haemoproteus turtur, 18
  Haemoproteus velans, 18
    of woodpeckers, 20
  history, 13–14
  host range, 14–15
  immunity, 26–27
  infection in pigeons and doves, 27
  infections, subclinical impacts of, 28
  management implications, 28
  as models for testing evolutionary theories, 13
  morphological forms of the mature gametocytes, 16
  pathogenesis and pathology, 21–25
  preerythrocytic tissue stages of, 13
  public health concerns, 27
  sexual and asexual reproduction, 17
  sexual stages of, 13
  synonyms, 13
  and temperate climates, 20
  treatment and control, 28
  wildlife population impacts, 27–28
haemosporidian disease, 54
Haemosporidiosis, 13
Haemospororida order, 36
hairworms, 316, 463
Haliaeetus spp., 465
  Haliaeetus albicilla (White-tailed Eagle), 75, 237, 239, 346, 472
  Haliaeetus leucocephalus (Bald Eagle), 75, 215, 233, 237, 297, 298, 472
  Haliaeetus vocifer (African Fish Eagle), 75
halofuginone, 48
Harrier, 550
Harlequin Duck (*see Histrionicus histrionicus*)
Hartlaub's Duck (*see Pteronetta hartlaubii*)
Hawaii Amakihi (*see Hemignathus virens*)
Hawaiian Crow (*see Corvus hawaiiensis*)
Hawaiian Goose (*see Branta sandvicensis*)
Hawks (Falconiformes), 277
Hazel Grouse (*see Bonasa bonasia*)
Heliornithidae (Finfoots), 442
Helmeted Guineafowl (*see Numida meleagris*)
Helodrilus caliginosus, 486
hematazoa, 7
hematocrits, 42, 43
hematophagous dipteran larvae, 548–549
hematozoan disease, 54 infections, in birds, 35
hematozoans, 21
hematozoin, 39
Hemignathus virens (Hawaii Amakihi), 46, 48, 111
Hemiprocnidae (Treeswifts), 443
hemorrhagic streaks, 21
hemosiderin, 89
heparinized Mallard blood (*Anas platyrhynchos domesticus*), 502
hepatic megalomeronts, 26
Hepaticola, 463
hepatic trematodiasis, 233
Hepatozoon, 108, 109
Heptapsogaster sp., 517
Heraldakis, 394
heterakiasis, 388
heterakid eggs, 156
Heterakis spp., 394, 402
  avian hosts infected with, 389–394
  clinical signs, 404
  diagnosis, 405
  distribution and host range, 388–394
  epizootiology, 401–404
  etiology, 394–401
  Heterakis beramporia, 391, 392, 393
  Heterakis bosia, 393
  Heterakis brevispiculum, 391, 392
  Heterakis dispar, 389, 390, 392, 393, 394
  Heterakis gallinarum, 6, 156, 388, 389, 390, 391, 392, 393, 394, 401, 402, 403–404, 404, 405, 407, 413, 491

Heterakis hamulus, 391, 392
Heterakis indica, 392
Heterakis inglisi, 389
Heterakis interlabiata, 392
Heterakis isolonche, 388, 390, 391, 392, 393, 401, 405
Heterakis kumaoni, 392
Heterakis kurilensis, 394
Heterakis macroura, 390, 392
Heterakis meleagris, 393
Heterakis nainitalensis, 392
Heterakis nattereri, 389, 390
Heterakis oscari, 390
Heterakis parva, 392, 393
Heterakis pavonis, 389, 391, 392, 393, 394
Heterakis pedioecetes, 393
Heterakis psophiae, 393
Heterakis pusilla, 390, 392
Heterakis silindae, 391, 392
Heterakis skrjabini, 393
Heterakis spiculata, 389
Heterakis tenuicauda, 390, 392, 393
Heterakis tragopanis, 393
Heterakis valdemucronata, 389
Heterakis vulvolabiata, 391
history, 388
immunity, 405
pathology, 404–405
public and domestic animal health concerns, 405
synonyms, 388
treatment and control, 406–407
wildlife population impacts, 405–406
heterakosis, 388
Heterocypris incongruens, 335
heteromorphic deutonymphs (hypopi), 529
heterophils, 21
Heterophyidae, 230, 235, 237
Hexametra sp., 425
Hill Partridge (*see Arborophila torqueda*)
Himalayan Monal (*see Lophophorus impejanus*)
Himalayan Snowcock (*see Tetraogallus himalayensis*)
Himantopus leucocephalus (Pied Stilt), 361
Himantopus, 465
Himantopus himantopus (Black-winged Stilt), 361
Himasthla, 230
Himatione sanguinea (Apapane), 111
hippoboscid flies, 15, 18
columbiform parasites from, 15
as vectors, 15
hippoboscid transmission, of *H. lophortyx*, 17
hippoboscid vectors, 13–14
Hirudinidae, 502, 505, 506, 508
hirudinids, 502, 507
Hirudobdella antipodum, 505
Hirudo medicinalis, 505
Hirudo nipponia, 505
Hirundinidae (Swallows), 60, 435, 444
Hirundoecus, 519
Hirundo rustica (Barn Swallow), 481, 518
Hispaniolan Parakeet (*see Aratinga chloroptera*)
Hispaniolepis falcata, 264, 268
histomonads, 156, 157
Histomonas spp.
Histomonas bonasae, 156
Histomonas isolonche, 156
Histomonas meleagridis, 140, 154, 158, 405
epizootiology of, 156
infected liver, gross lesions of, 157
within lesions, 158
species of the order galliformes reported to be susceptible to infection, 155
histomoniasis
clinical signs, 156–157
diagnosis, 157–158
distribution, 154
domestic animal and public health concerns, 158
epizootiology of, 156
etiology, 154
from junglefowl, 159
history, 154
host range, 154
immunity, 158
pathogenesis and pathology, 157
prevention, control, and management implications, 158–159
synonyms, 154
wildlife population impacts, 158
Histrionicus histrionicus (Harlequin Duck), 330, 358
holostomes, 226
Homo sapiens (Human), 302
honeycreepers (Drepanidinae), 48
Hooded Crane (*see Grus monacha*)
Hooded Crow (*see Corvus cornix*)
Hooded Merganser (*see Lophodytes cucullatus*)
Hooded Vulture (*see Necrosyrtes monachus*)
Hooded Warbler (*see Wilsonia citrina*)
Horned Grebe (*see Podiceps auritus*)
Horned Puffin (*see Fratercula corniculata*)
host foraging or nesting behavior, 21
host–parasite relationship, 3, 13, 256
host responses, from avian hosts, 22–24
host specificity, of the parasites, 21
Houbara Bustard (*see Chlamydotis undulata*)
House Martin (*see Delichon urbicum*)
House Sparrow (*see Passer domesticus*)
House Wren (*see Troglodytes aedon*)
Houttuynia struthionis, 272
Huffia, 37, 39
Huffmanela, 463
Humboldt Penguin (*see Spheniscus humboldti*)
Hyacinth Macaw (*see Anodorhynchus hyacinthinus*)
Hyalella, 337
Hyalella azteca, 337
Hydrobatidae (Storm Petrels), 441
Hylocichla mustelina (Wood Thrush), 238
Hymenolaimus malacorhynchos (Blue Duck), 329
Hymenolepididae, 262
hymenolepididiasis, 262
Hymenolepis falsata, 269
Hymenolepis microps, 271
hyperplasia, of white pulp arteriolar endothelium, 21
hyperplasia of the proventriculus, 237
Hypodectes, 529
Hypodectes nudus, dorsal and ventral views of adult female, 532
Hypoderaeum, 230
Hypoderaeum conoideum, 226
hypoderatid mites (Hypoderatidae), 529
Hyptiasmus, 229
Hysterothylacium, 413, 414, 423

Ibidorhynchidae, 442
Icteridae (Troupials), 60, 435, 444
Ictinaetus malayensis (Black Eagle), 74
IgA and IgG1 pathogen-specific antibodies, 142

Iiwi (*see Vestiaria coccinea*)
immunoblot analysis, 92
immunocompromised condition, of the bird, 56–57
immunofluorescence, 92
Inca Dove (*see Columbina inca*)
Indian Peafowl (*see Pavo cristatus*)
Indicatoridae (Honeyguides), 61, 443
indirect hemagglutination test (IHAT), 214
Indocapillaria, 463
inflammatory infiltrates, 25
inflammatory myopathy, 44
InPouch culture kits, 136, 139
insecticides, treatment with, 554
intestinal cryptosporidiosis, 198
intestinal flagellates, 3
intestinal infection, in cranes, 187
Intestine, 449
intraerthrocytic development
  of *Haemoproteus* species, 15
  of *Plasmodium* species, 36
intraerythrocytic gametocytes, morphology of, 25
intraerythrocytic hemosporidian parasites, 13
intraerythrocytic parasites, of wild birds, 35
intravascular agglutinations, of erythrocytes, 44
intrinsic factors, of parasites, 21
Irenidae (Fairy-bluebirds), 61, 444
Ischnocera, 516, 517
Island Canary (*see Serinus canaria*)
Island Collared-Dove (*see Streptopelia bitorquota*)
Isopoda, 279
isopod intermediate hosts, elimination of the, 339
Isospora spp., 163
  clinical signs, 112
  diagnosis, 112–113
  distribution, 109
  domestic animal health concerns, 117
  epizootiology, 110–112
  etiology that cause atoxoplasmosis, 109–110
  history, 108–109
  host range, 109
  immunity, 113
  Isospora buteonis, 424
  Isospora canaria, 110
  Isospora rothschildi, 109
  oocyst of, 109–110
  pathogenesis and pathology, 112
  public health concerns, 116
  synonym, 108
  that cause atoxoplasmosis, 108
  treatment and control, 117
  wildlife population impacts, 117
Israeli House Sparrow (*see Passer domesticus biblicus*)
Ithaginis cruentus (Blood Pheasant), 390, 396
ivermectin, 340, 352, 381, 406, 437, 459, 491, 535
Ixobrychus sinensis (Yellow Bittern), 488
Ixoreus, 465
Ixoreus naevius (Varied Thrush), 481

Jacanidae (Jacanas), 59, 442
Jackal Buzzard (*see Butea rufofuscus*)
Jackass Penguin (*see Spheniscus demersus*)
Japanese Quail (*see Coturnix japonica*)
Japanese Sparrowhawk (*see Accipiter gularis*)
Japanese White-eye (*see Zosterops japonicus*)
Jararaca (*see Bothrops jararaca*)
Jardaya Parakeet (*see Aratinga jandaya*)
Java Sparrow (*see Padda oryzivora*)
Jilinobilharzia, 246, 247, 251
Jilinobilharzia crecci, 251
Jones' Barn (JB) strain, 134–135

Kalij Pheasant (*see Lophura leucomelanos*)
Kaplan–Meier survivorship curves, 144
Kaupifalco monogrammicus (Lizard Buzzard), 74, 78
Kea (*see Nestor notabilis*)
Keel-billed Toucan (*see Ramphastos sulfuratus*)
Killdeer (*see Charadrius vociferus*)
killifish (*Fundulus heteroclitus*), 290
King Eider (*see sanaena spectabilis*)
kingfishers (*Megaceryle* spp.), 226
King Vulture, (*see Sarcoramphus papa*)
Kinkajou (*Potos flavus*), 425
Kitodites, 529
Klebsiella spp., 434
Knemidokoptes spp.
  Knemidokoptes derooi, 528
  Knemidokoptes jamaicensis, 528
  Knemidokoptes pilae, 528
knemidokoptic podoacariasis, 535
knemidokoptids, 531
koilin lining, 369
Kori Bustard (*Ardeotis kori*)
Kowalewskiella, 263
Kupffer cells, 89
*Kytodites nudus, d*orsal and ventral views of adult female, 531

labored breathing, 87
lactic acid, 509
Lady Amherst's Pheasant (*see Chrysolophus amherstiae*)
Laemobothriidae, 516
Lagochilascaris, 425
Lagopus, 465
  Lagopus lagopus scotica (Red Grouse), 5, 6, 7, 316, 322
  Lagopus lagopus (Willow Ptarmigan), 261, 268, 271, 320, 345, 393, 397, 403, 405, 459, 464
  Lagopus leucura (White-tailed Ptarmigan), 397
  Lagopus muta (Rock Ptarmigan), 360, 393, 397
Laminosioptes cysticola (Laminosioptidae), 528, 532
laminosioptids, 534
Lamprotornis superbus (Superb Starling), 111
Laniidae (Shrikes), 60, 444
Lanius excubitor (Northern Shrike), 206
Lanius schach (Long-tailed Shrike), 482
Lankesterella, 108, 109
Lanner Falcon (*see Falco biarmicus*)
Lappet-faced Vulture (*see Torgos tracheliotus*)
Large-tailed Nightjar (*see Caprimulgus macrurus*)
Laridae (Gulls), 442, 505
Larus spp.
  Larus, 465
  Larus argentatus (European Herring Gull), 332, 238, 346
  Larus atricilla (Laughing Gull), 208, 238
  Larus canus (Mew Gull), 291, 332
  Larus delawarensis (Ring-billed Gull), 208, 255
  Larus ichthyaetus (Great Black-headed Gull), 332
  Larus novaehollandiae (Silver Gull), 46, 476
  Larus ridibundus (Black-headed Gull), 205, 291, 343, 346
  Larus thayeri (Thayer's Gull), 332
larval *Austrobilharzia*, 246
larval *Bilharziella*, 247
larval diphyllobothriids, 263
larval *Gigantobilharzia*, 247

larval *Mesocestoides* (Cyclophyllidea), 263
larval *Ornithobilharzia*, 246
larval *Trichobilharzia*, 247
larva migrans, 424
Lateriporus sp., 267
Laterotrema, 231
latex agglutination test (LAT), 92, 214
Laughing Dove (*see Streptopelia senegalensis*)
Laughing Gull (*see Larus atricilla*)
Laysan Albatross (*see Phoebastria immutabilis*)
Laysan Duck (*see Anas laysanensis*)
Least Sandpiper (*see Calidris minutilla*)
Lecithodendriidae, 230, 238
leech parasites, of birds
  clinical signs, 506
  diagnosis, 506–508
  epizootiology, 503–506
  etiology, 502–503
  freshwater, 501
  history, 501
  host range and distribution, 501–502, 505
  immunity, 508–509
  life cycle of, 503
  pathogenesis and pathology, 506
  public and domestic animal health concerns, 509
  sanguivorous, 503–504
Leipoa ocellata (Malleefowl), 517
Lemdana, 441–445, 448, 454
Lemdana wernaarti, 457
Lemdaninae, 448
Lemon Dove (*see Columba larvata*)
Leptoptilos crumeniferus (Macabou Stork), 269, 295, 389, 395, 454
Leptosomatidae (Cuckoo Rollers), 443
Leptotila verreauxi (White-tipped Dove), 129
lesions
  by acute infections with *Plasmodium*, 44–45
  associated with atoxoplasmosis, 116
  associated with blood-feeding larvae, 552
  associated with *Trichomonas gallinae*, 137–138
  Baylisascaris species, 427
  caused by *Contracaecum*, 421
  caused by fungi and nematods, 140
  of *Cryptosporidium*, 198
  from *Dispharynx nasuta*, 334
  in ducks infected with *Cloacotoenia megalops*, 269
  due to trematodes, 233
  in DVC, 181, 190–191
  of *Eimeria* species, 182–185
  Eustrongylides spp. infections, 298, 305–307
  of *Histomonas meleagridis*, 157
  of renal *Eimeria*, 175
  from schistosomes, 246, 249, 255
  Serratospiculum, 436
  of *Toxoplasma*, 212–213
  in waterfowl with fatal leucocytozoonosis, 89, 91
Lesser Flamingo (*see Phoenicopterus minor*)
Lesser Kestrel (*see Falco naumanni*)
Lesser Scaup (*see Aythya affinis*)
Lesser Spotted Eagle (*see Aquila pomarina*)
Lesser Snow Goose (*see Chen caerulescens caerulescens*)
Lesser White-fronted Goose (*see Anser erythropus*)
Lesser Sandhill Crane (*see Grus canadensis canadensis*)
lethargy, 87
Leucochloridiidae, 230
Leucochloridiomorpha, 229
Leucochloridium, 230
Leucocytoon, by orders and families of birds, 59–61
Leucocytozoidae, 54, 57
leucocytozoids, 54
Leucocytozoon spp., 13, 17, 27, 35, 46, 54, 59, 60, 61, 82–87
  clinical signs, 87
  diagnosis, 91–92
  disease, 54
  distribution, 56
  domesticated animal health concerns, 93
  epizootiology, 82–87
  etiology, 57–82
  in a gaviiform, 56
  geographic distribution of eight species of, 57, 58
  history, 54–55
  host range, 56–57
  immunity, 92–93
  infections, 25
  Leucocytozoon balmorali, 60, 61, 85
  Leucocytozoon bennetti, 60, 85
  Leucocytozoon berestneffi, 60, 81, 85
  Leucocytozoon bonasae, 58
  Leucocytozoon caprimulgi, 59, 85
  Leucocytozoon caulleryi, 54, 55, 57, 59, 81, 82, 85, 89, 92, 93
  Leucocytozoon centropi, 59, 85
  Leucocytozoon cheissini, 59, 85
  Leucocytozoon colius, 59, 85
  Leucocytozoon communis, 60, 85
  Leucocytozoon danilewskyi, 59, 81, 84, 85, 88
  Leucocytozoon dizini, 59, 85
  Leucocytozoon dubreuili, 60, 61, 81, 82, 84, 85
  Leucocytozoon eurystomi, 60, 85
  Leucocytozoon fringillinarum, 60, 61, 81, 85
  Leucocytozoon grusi, 59, 85
  Leucocytozoon hamiltoni, 61, 85
  Leucocytozoon leboeufi, 59, 85
  Leucocytozoon legeri, 59, 85
  Leucocytozoon lovati, 59, 81, 84, 85
  Leucocytozoon maccluri, 85
  Leucocytozoon macleani, 57, 59, 85, 93
  Leucocytozoon majoris, 27, 60, 61, 81, 85
  Leucocytozoon marchouxi, 55, 56, 57, 59, 70–73, 85, 87, 89, 91, 93
  Leucocytozoon neavei, 58, 59, 85
  Leucocytozoon nycticoraxi, 59, 85
  Leucocytozoon nyctyornis, 60, 85
  Leucocytozoon polyoon, 226, 234
  Leucocytozoon sakharoffi, 60, 85
  Leucocytozoon sakharoffi, of crows, 81
  Leucocytozoon schoutedeni, 57, 58, 59, 85, 93
  Leucocytozoon simondi, 55, 57, 58, 59, 82, 83, 84, 85, 88, 89, 90, 537
    gametocytes of, 83
    infections in waterfowl, 92
    infections in wood ducks, 84
    life cycle of, 82
    megalomeronts of, 90
    in waterfowl, 81, 86, 87, 93
    in wild waterfowl, 56, 62–69
  Leucocytozoon smithi, 54, 55, 57, 59, 84, 85, 86, 87, 537
    from turkeys, 81
    in Wild Turkeys (*Meleagris gallopavo*), 86, 87
  Leucocytozoon sousadiasi, 59, 85
  Leucocytozoon squamatus, 60, 85
  Leucocytozoon struthionis, 57, 59, 85
  Leucocytozoon tawaki, 85

Leucocytozoon spp. (*Cont.*)
Leucocytozoon toddi, 55, 57, 59, 84, 85, 91
in diurnal raptors, 81, 87
in raptors, 56, 89, 93
in wild falconiforms, 74–80
Leucocytozoon vandenbrandeni, 59, 85
management implications, 95
by orders and families of birds, 59–61
pathogenesis, 87–89
pathology, 89–91
prevalence of species of *Leucocytozoon* in birds in various zoogeographic regions, 56
prevention, treatment and control, 94–95
public health concerns, 93
synonym, 54
wildlife population impacts, 93–94
Leucopsar rothschildi (Bali Myna), 109, 116
merozoite in monocyte of, 110
Leucosarcia melanoleuca (Wonga Pigeon), 217
Leukocytozoon (sic) *danilewskyi*, 54, 87
levamisole, 339, 340, 381, 406, 459, 491
levamisole hydrochloride, 322, 407, 459
Levant Sparrowhawk (*see Accipiter brevipes*)
Levinseniella, 230
Leyogonimus, 230
Leyogonimus polyoon, 225, 227, 238
life history traits, of a bird, 4
Ligula, 267, 271
Ligula intestinalis, 264, 267, 269, 270
Limnodrilus hoffmeisteri, 300–301
Limosa fedoa (Marbled Godwit), 451, 521
Limosa lapponica (Bar-tailed Godwit), 271–272
Limpkin (*see Aranus guarauna*)
Little Black Cormorant (*see Phalacrocorax sulcirostris*)
Little Blue Heron (*see Egretta caesulea*)
Little Bustard (*see Tetrox tetrax*)
Little Cuckoo-Dove (*see Macropyg's ruficeps*)
Little Egret (*see Egretta garzetta*)
Little Grebe (*see Tachybaptus ruficollis*)
Little Owl (*see Athene noctua*)
Little Penguin (*see Eudyptula minor*)
Little Pied Cormorant (*see Phalacrocorax melanoleucos*)
Little Sparrowhawk (*see Accipiter minullus*)
Little Swift (*see Apus affinis*)
livebearers (Poeciliidae), 290
liver enlargement, 116
Lizard Buzzard (*see Kupifalco monogrammicus*)
Locusta migratoria, 378
Lofortyx, 465
Lonchura punctulata (Nutmeg Mannikin), 111
Long-billed Partridge (*see Rhizothera longirostris*)
Long-crested Eagle (*see Lophaetus occipitalis*)
Long-legged Buzzard (*see Butea rufinus*)
Long-tailed Cormorant (*see Phalacrocorax africanus*)
Long-tailed Duck (*see Clangula hyemalis*)
Long-tailed Parakeet (*see Psittacula longicaudata*)
Long-tailed Shrike (*see Lonias schach*)
Lophaetus occipitalis (Long-crested Eagle), 76
Lophodytes, 465
Lophodytes cucullatus (Hooded Merganser), 67, 297, 330
Lophophorus impejanus (Himalayan Monal), 391
Lophura spp.
Lophura diardi (Siamese Fireback), 114
Lophura ignita (Crested Fireback), 391
Lophura leucomelanos (Kalij Pheasant), 392
Lophusa nycthemera (Silver Pheasant), 393
Loriidae (Lories and Lorikeets), 442
Lorinus sp. (Lory), 115
Lucilia sericata, 550, 552
Lucilia spp., 553
Lueheia spp., 277
Lueheia inscripta, 281
Lueheia inscripta, transmission of, 281
Lumbricus terrestris, 486
lumen of blood vessels, 449
Lunaceps numenii, 521
lungs, 449
Luscinia calliopei (Siberian Rubythroat), 482
Luscinia megarhynchos (Common Nightingale), 482
Luzon Bleeding-heart (*see Gallicoumba luzonica*)
lymphatics, 449
lymphocytes, 21
lymphocytic infiltrates, 21
lymphoid hyperplasia, 369
lymphoid–macrophage system capillary endothelium cells, 19
Lynceus brachyurus, 335
Lynchia hirsuta, 17, 18
Lyperosomum, 229
Lyperosomum longicauda, 226
Lyrurus, 465

Macaw, 550
Macrobilharzia, 246, 247, 251
Macrobilharzia pulverulenta, 251
Macrocephalon maleo (Maleo), 390
Macrocyclops fuscus, 385
macrogametocytes, 17, 20, 39–40, 165
fertilization of, 40
Macropygia nigrirostris (Black-billed Cuckoo-Dove), 398
Macropygia ruficeps (Little Cuckoo-Dove), 73
Macyella, 230
Madagascar Buzzard (*see Buteo brachypterus*)
Madagascar Green-Pigeon (*see Treron australis*)
Madagascar Turtle-Dove (*see Streptopelia picturata*)
Magellanic Penguin (*see Spheniscus magellanicus*)
Magnificent Bird-of-paradise (*Cicinnurus magnificus*)
Magpie Goose (*see Anseranas semipalmata*)
Magpie-lark (*see Grallina cyanoleuca*)
magpies, 347
Maguari Stork (*see Ciconia maguari*)
Malaconotidae (Bushshrikes), 60
malaria, human, 35
malarial infection, in avian hosts. *see* avian malaria
malarial parasites, 13
malarial pigment. *see* hematozoin
male gametocyte. *see* macrogametocytes
male microgametocytes, 17
Maleo (*see Macrocephalon maleo*)
Mallard (*see Anas platyrhynchos*)
Malleefowl (*see Leipoa ocellata*)
Mallee Ringneck (*see Barnardius barnardi*)
Mammomanogamus, 351
Mandarin Duck (*see Aix galericulata*)
Maned Duck (*see Chenonetta jubata*)
Mangrove Swallow (*see Tachycineta albilinea*)
Manorina melanocephala (Noisy Miner), 22
Mansonia crassipes, 41
Marabou Stork (*see Leptoptilos crumeniferus*), 295, 389, 395
Marbled Godwit (*see Limosa fedoa*)
Marbled Teal (*see Marmaronetta angustirostris*)

Maritrema, 230
Maritrema acadiae, 236, 239
Maritrema bonaerensis n. sp., 240
Maritrema sp., 236
Marmaronetta angustirostris (Marbled Teal), 358, 364
Maroon Woodpecker (*see Blythipicus rubiginosus*)
mass-capture methods, 3
mature gametocytes, 19
Mawsonotrema eudyptulae, 235
Meadow Pipit (*see Anthus pratensis*)
Mealy Parrot (*see Amazona farinose*)
mebendazole, 339, 340, 352, 371, 437, 459
Mediorhynchus spp., 277
Mediorhynchus centurorum, 282
Mediorhynchus centurorum in a Red-bellied Woodpecker, 282
Mediorhynchus gallinarum, 282, 283
Mediorhynchus orientalis, 283
mefloquine, 28, 48
megalomeronts, 19
with associated muscle pathology, 25
of *Haemoproteus*, 25
hepatic, 26
inflammatory infiltrates affecting, 25
in leucocytozoonosis, 88–89
from pectoral muscle of a domestic turkey with an experimental infection, 27
ruptured, from pectoral muscle of a domestic turkey, 26
Megaceryle alcyon (Belted Kingfisher), 238, 346
Megaceryle torquatus (Ringed Kingfisher), 238
megaloschizonts, 89
Megapodiidae (Megapodes), 441
Megascops asio (Eastern Screech-Owl), 133, 213
Melanerpes spp.
Melanerpes carolinus (Red-bellied Woodpecker), 218, 282, 478
Melanerpes erythrocephalus (Red-headed Woodpecker), 332
Melanerpes flavifrons (Yellow-fronted Woodpecker), 376
Melanitta spp.
Melanitta fusca (White-winged Scoter), 115, 285, 296, 330, 360, 365, 469
Melanitta nigra (Black Scoter), 67, 356, 362, 395, 469
Melanitta perspicillata (Surf Scoter), 67, 330, 359, 469
Melanopus spp., 378
melarsomine, 95
melatonin, 40
Meleagrididae (Turkeys), 59
Meleagris spp., 465
Meleagris gallopavo (Domestic Turkey, Wild Turkey), 13, 23, 154, 155, 167, 169, 195, 205, 208, 214, 215, 217, 233, 234, 237, 268, 327, 345, 388, 393, 397, 464, 473, 528
with infections of *Eimeria*, 163
with infections of *Plasmodium durae*, 42
Meleagris ocellata (Ocellated Turkey), 392, 396
Melierax metabates (Dark Chanting Goshawk), 78
Meliphagidae (Honeyeaters), 61, 444
Melopsittacus, 465
Melopsittacus undulatus (Budgerigar), 113, 137, 168, 206, 210, 213, 399
meningoencephalitis, 424
Menopon gallinae, 521
Menoponidae, 516
Menuridae (Lyrebirds), 444
Mergellus albellus (Smew), 67, 297, 330, 359
Mergus merganser (Common Merganser), 66, 227, 255, 297, 306, 331, 469
Mergus serrator (Red-breasted Merganser), 67, 227, 297, 304, 305, 470
Merlin (*see Falco columbarius*)
merogony, 13, 36, 39
meronts, 19, 21, 36, 57, 91, 116
morphology in species of *Leucocytozoon*, 58
Meropidae (Bee-eaters), 60, 443
merozoites, 82, 165
of *Eimeria gruis* and *Eimeria reichenowi*, 187–188
haemoproteids transmitted by hippoboscid flies, 20
of *Isospora* species, 112
in leucocytozoonosis, 88
released from exoerythrocytic meronts, 39
species of *Cryptosporidium*, 196–197
of species of *Isospora*, 110–111
transformation, 15, 19
Mesamidostomum, 366
Mesitornithidae (mesites), 442
Mesocestoides, 271
Mesocestoides sp., 264
Mesocestoididae, 264, 266
Mesocyclops leuckarti, 384, 385
Mesostephanus appendiculatoides, 235
Me-49 strain, 213
metacryptozoites, 39
Metallic Pigeon (*see Columba Vitiensis*)
methanol, 112
Metorchis, 231
Metorchis bilis, 233, 237, 239
Metroliasthes lucida, 264, 268
metronidazole, 144
Mew Gull (*see Larus canus*)
Mhc alleles, 42
mice (*Mus musculus*), 195
microcrustaceans, 279
microfilariae, 440
of avian filarioids, 453
morphology, 456–458
microfilariae, morphological traits of, 454–458
microfilaria (MF), 439
microgametocytes, 20, 39
Microlynchia pusilla, 18
Micronisus gabar (Gobar Goshawk), 79
Microphallidae, 230, 236
micropyle, 112
Microscaphidiidae, 230
Microsomacanthus collaris, 264, 267
Microsomacanthus parvula, 264, 268, 269
Microtetrameres spp., 376, 377, 381
Microtetrameres centuri, 379
Microtetrameres corax, 378, 379
Microtetrameres egretes, 376
Microtetrameres inermis, 378
Microtetrameres nestoris, 380
Microtetrameres spiralis, 376
Micrurus sp. (Coral snake), 302
midgut basal lamina, 17
midgut epithelium, 40
midgut wall, 17
Milvago chimango (Chimango Caracara), 79, 472
Milvus migrans (Black Kite), 76, 114, 130
Milvus milvus (Red Kite), 76, 472
Mimidae (Mockingbirds and Thrashers), 60, 435, 444
Mimus polyglottos (Northern Mockingbird), 208, 333
miracidium, 226, 248

Mississippi Sandhill Crane (*see Grus canadensis pulla*)
Mistle Thrush (*see Turdus viscivorus*)
mitochondrial gene sequences, of avian haemoproteids, 17
Mitochondrial genes (mtDNA-cox2), 425
Mniotilta variai (Black-and-white Warbler), 482
modified agglutination test (MAT), 214
moist heather plants (*Calluna vulgaris*), 317
molecular methods, 25, 39
molluscicides, 240
Molothrus ater (Brown-headed Cowbird), 333, 482
Momotidae (Motmots), 60, 443
monensin, 172, 192
Monk Parakeet (*see Myiopsitta monachus*)
Monocercomonadidae, 154
monocytes, 21
Montagu's Harrier (*see Circus pygargus*)
Montezuma Oropendola (*see Gymnostinops montezuma*)
Monticola solitarius (Blue Rock-thrush), 482
mortality
  from acanthocephalan infections, 280
  among leeches, 509
  associated with *Haemoproteus*, 27
  from atoxoplasmosis, 111
  in breeding Willow Ptarmigan, 271
  in California sea otters (*Enhydra lutris*), 285
  caused by *Eimeria truncata*, 175
  cestodes, 264–265
  due to acanthocepalan-induced peritonitis, 285
  due to dispharynxiasis, 339
  from DVC, 192
  of *Eimeria* spp., 182–185
  Eustrongylides spp. infection, 309
  leeches, 509
  leucocytozoonosis, 94
  myiasis, 552
  from *Plasmodium* species, 48
  from trematodes, 240
Mosquito
  Aedeomyia squomipennis, 41
  Culex annulus, 41
  Culex morsitans, 41
  Culex nigripalpus, 41
  Culex pipiens, 41
  Culex quinque fasciatus, 41
  Culex restuans, 41
  Culex saltenensis, 41
  Culex Sitiens, 41
  Culex stimatosoma, 41
  Culex tarsalis, 41
  Mansonia crassipes, 451
mosquito vectors, 41, 451
Motacilla alba (White Wagtail), 345
Motacillidae (Wagtails and Pipits), 60, 444
motile zygote, 17
Mott cells, 21
Mottled Duck (*see Anas fulvigula*), 83, 235, 364
Mountain Chickadee (*see Poecile gambeli*)
Mourning Dove (*see Zenaida macroura*)
Mudpuppy (*Necturus maculosus*), 303
Murre (*Uria* sp.), 291
Muscicapidae (Old World Flycatchers), 60, 435, 444
Muscicapinae, 505
Muscidae, 547
Muscovy Duck (*see Cairina moschata*)
Musk Duck (*see Biziura lobata*)
Musophagidae (Turacos), 59, 442
Musophagiformes, 59
Mussurana (*see Clelia clelia*)
Mute Swans (*see Cygnus olor*)
Mycteria americana (Wood Stork), 295, 468
Myialges nudus, 527, 530
Myiarchus crinitus (Crested Flycatcher), 529
myiasis
  clinical signs, 550–551
  diagnosis, 552
  distribution, 547
  epizootiology, 549–550
  etiology, 549
  history, 547
  host range, 547–549
  immunity, 552
  management implications, 554
  pathogenesis and pathology, 551–552
  public and domestic health concerns, 552
  synonyms, 546–547
  treatment and control, 553–554
  wildlife population impacts, 552–553
Myiopsitta monachus (Monk Parakeet), 23, 116
myofibroblasts, 19
myositis, 21
  in avian hosts, 13
Myrsidea ptilinorhynchi, 518
Myxobdella annandalei, 505

Nacunda Nighthawk (*see Podager nacunda*)
Namaqua Dove (*see Oena capensis*)
narasin, 177
nasal and oral exudation, 137
Nashville Warbler (*see Vermivora ruficapilla*)
Nearctic ecozone, 20
Nearctic region, *Leucocytozoon* species in, 56
neck, 450
necrosis, 157, 175, 212
Necrosyrtes monachus (Hooded Vulture), 74
Nectariniidae (Sunbirds and Spiderhunters), 60, 444
neguvon, 371
nematodiasis, 368, 413
Neochen jubata (Orinoco Goose), 329
Neochmia modesta (Plum-headed Finch), 195
Neodiplostomum, 229
Neoknemidokoptes laevi, 532
Neoknemidokoptes laevis, 528
Neophema spp.
  Neophema bourkii (Bourke's Parrot), 399
  Neophema elegans (Elegant Parrot), 399
  Neophema pulchella (Turquoise Parrot), 400
  Neophema splendida (Scarlet-chested Parrot), 400
Neophron percnopterus (Egyptian Vulture), 130
Neotropic Cormorant (*see Phalacrocorax brasilianus*)
Neottialges, 529
Neottiophilidae (*Neottiophilum*, *Actinoptera*), 547, 549
Neottiophilum praeustum, 549
Nerodia sipedon (Northern water snake), 302
Nerodia taxispilota (Brown water snake), 302
Nesoenas mayeri (Pink Pigeon), 70, 125, 134
nestling birds, cost of parasitism, 6
Nestor meridionalis (New Zealand Kaka), 380
Nestor notabilis (Kea), 476
nest site fidelity, 4

Netta, 465
Netta peposaca (Rosy-Billed Pochard), 330
Nettapus auritus (African Pygmy-Goose), 329
Nettapus coromandelianus (Cotton Pygmy-goose), 329, 357
Netta rufina (Red-crested Pochard), 66, 330, 359, 364
neural larva migrans (NLM), 425
New Britain Hawk-Owl (*see Ninox odiosa*)
New Zealand Kaka (*see Nestor meridionalis*)
New Zealand white rabbit (*Oryctolagus cuniculus*), 302
nicarbazin, 177
niclosamide, 255, 272, 424
Ninox odiosa (New Britain Hawk-Owl), 401
nodular typhlitis, 404
Noisy Friarbird (*see Philemon corniculatus*), 22
Noisy Miner (*see Manorina melanocephala*), 22
nonhematophagous dipteran larvae, 550
Northern Bobwhite (*see Colinus virginianus*)
Northern Cardinal (*see Cardinalis cardinalis*)
Northern Flicker (*see Colaptes auratus*)
Northern fowl mite (*Ornithonyssus sylviarum*), 534
Northern Goshawk (*see Accipiter gentilis*)
Northern Harrier (*see Circus cyaneus*)
Northern Lapwing (*see Vanellus vanellus*)
Northern leopard frog (*see Rana pipiens*), 303
Northern Long-eared Owl (*see Asio otus*)
Northern Mockingbird (*see Mimus polyglottos*)
Northern Pintail (*see Anas acuta*)
Northern Saw-whet Owl (*see Aegolius acadicus*)
Northern Shoveler (*see Anas clypeata*)
Northern Shrike (*see Lanius excubitor*)
Northern water snake (*Nerodia sipedon*), 302
Northiella haematogaster (Bluebonnet), 399
North Island Brown Kiwi (*see Apteryx mantelli*)
Nothoprocta, 465
Nothura maculosa (Spotted Nothura), 389, 395, 466
Notocotylidae, 231, 236
Notocotylus, 231
Notocotylus attenuatus, 226, 236, 240
Novyella, 37, 39
Nucifraga caryocatactes (Eurasian Nutcracker), 297, 345, 480
nuclear gene sequences, of avian haemoproteids, 17
Numenius phaeopus (Whimbrel), 451, 521
Numida, 465
Numida meleagris (Helmeted Guineafowl), 113, 155, 168, 331, 388, 391, 396, 473
Numididae (Guineafowl), 59
Nutmeg Mannikin (*see Lonchura punctulata*)
nutrient shortage in early development, effect, 6
Nyctanassa violacea (Yellow-crowned Night-Heron), 115, 238, 295
Nyctibiidae (Potoos), 443
Nycticorax caledonicus (Rufous Night Heron), 424
Nycticorax nycticorax (Black-crowned Night-Heron), 295, 389, 468
Nymphicus hollandicus (Cockatiel), 398

Ocellated Turkey (*see Meleagris ocellata*)
ocular larva migrans (OLM), 425
Odontophorus capueira (Spot-winged Wood-Quail), 237, 476
Odontoterakis, 394
Oena capensis (Nomaqua Dove), 73
Oligacanthorhynchus species, 279
oligochaetes, 301
oligochaete worm (*Limnodrilus hoffmeisteri*), 300
Olive-backed Pipit (*see Anthus hodgsoni*)
Onchocerca volvulus, 542
Onchocercidae, 440
Onchocercinae, 448
Oncicola canis, 280
Oncicola oncicola, 280
Oniscus species, 327
oocysts, 40, 108
  of *Cryptosporidium hominis*, 199
  of Caryospora, *Isospora*, and *Eimeria* in feces, 198
  contamination of environment, 212
  distribution and prevalence of shedding of *Eimeria* spp., 182–185
  of *Eimeria reichenowi* and *Eimeria gruis*, 182, 187–189
  Eimeria species, 165
  of *Eimeria* species, 176–177
  environmental contamination with, 192
  from Fish Crow (*Corvus ossifragus*), 110
  of *Isospora canaria*, 110
  of *Isospora rothschildi*, 109
  of *Isospora* species, 112
  species of *Cryptosporidium*, 196–200
  of species of *Sarcocystis*, 163
  sporulated, 212
  Toxoplasma, 211–213
  Toxoplasma gondii, 211–212
ookinetes, 17
Oosthuizobdella garoui, 505
operculum, 226
Ophidascaris, 425
Ophryocotyle, 263
Ophthalmophagus, 229
Opisthobrachia, 247
Opisthocomidae (Hoatzin), 442
Opisthocomiformes, 59
Opisthorchiidae, 226, 231, 235, 237, 238
Opisthorchis, 231
Opisthorchis felineus, 239
Opisthorchis sp., 233, 237
Opossums (*Didelphis* spp.), 114
opthaine, 509
oral suckers, 226
Orange Weaver (*see Ploceus aurantius*)
Orange-winged Parrot (*see Amazona amazonica*)
Orchipedidae, 231
Orchipedum, 231
Order Diptera, 451
Order Phthiraptera, 451
Order Tetrabothriidea, 264
Oreoscoptes montanus (Sage Thrasher), 5
Oriolidae (Old World Orioles), 60, 444
Oreortyx, 465
Oriental Turtle-Dove (*see Streptopelia orientalis*)
Orientia tsutsugamushi, 534
Orinoco Goose (*see Neochen jubata*)
ormetoprim-sulfa, 192
Ornithobdella edentula, 505
Ornithobdellidae, 502, 505, 506, 507, 508
Ornithobilharzia spp.
  Ornithobilharzia, 246, 247, 249, 251
  Ornithobilharzia baeri, 251
  Ornithobilharzia canalicula, 251
  Ornithobilhorzia emberizae, 251

Ornithocapillaria spp., 463
Ornithocapillaria appendiculata, 467
Ornithocapillaria carbonis, 466, 467
Ornithocapillaria cylindrica, 472
Ornithocapillaria ovopunctata, 479, 480, 482, 483, 484
Ornithocapillaria phalacrocoraxi, 467
Ornithocapillaria picorum, 478
Ornithocapillaria quiscali, 481, 483, 485
Ornithomyia aviculria, 18
ornithophilic black flies, bifid claws of, 538
Ornithosis bedsoniae, 521
Ortalis vetula (Plain Chachalaca), 331
Oryctolagus cuniculus (New Zealand white rabbit), 302
Oscelloscia floridana, 327
Osprey (*see Pandion haliaetus*)
Ostrich (*see Struthio camelus*)
Oswaldoia, 229
Otididae (Bustards), 59, 442
Otiditaenia spp., 268
Otiditaenia conoideis, 264, 269
Otiditaenia macqueeni, 264, 269
Otis, 465
Otis tarda (Great Bustard), 393
Otus scops (European Scops-Owl), 133
oviduct ruptures, 234
ovoid merozoites, 39
oxfendazole, 406
oxyclozanide, 240
Oxyruncidae (Cotingas), 444
Oxytrema silicula, 227
Oxyura jamaicensis (Ruddy Duck), 68, 227, 236, 331, 336, 359, 365
Oxyura leucocephala (White-headed Duck), 360, 365

Pachytrema, 231
Pacific Black Duck (*see Anas superciliosa*)
Padda oryzivora (Java Sparrow), 24
Painted turtle (*Chrysemys picta*), 303
Palearctic ecozone, 20
Pale-backed Pigeon (*see Columba eversmanni*)
Pallid Harrier (*see Circus macrourus*)
Palm Tanager (*see Thrgupis palmarum*)
Palm-Swift (*see Cypsiurus parvus*)
panacur, 437
Pandion haliaetus (Osprey), 80, 236, 472
Pandionidae (Ospreys), 59, 441
Parabasalia, phylum, 154
Paracapillaria, 463
Paracapillaroides, 463
Paradilepis delachauxi, 264
Paradilepis scolecina, 264, 268
Paradilepis sp., 264
Paradisaeidae (Birds of Paradise), 61, 445
Paradise Tanager (*see Tangara chilensis*)
Paradoxornithidae (Parrotbills), 60
Paraguayan caiman (*Caiman yacare*), 303
Parahaemoproteus, 15
Parakeet (species not reported), 23
Paramonostomum, 231
Paramphistomatidae, 231, 236
Parapronocephalum, 231
parasitism
cost of, 4, 6
and distribution of effects of parasites, 6
energy as a cost parameter against, 4
and predation, 5–6
study of, in wild birds, 3
in terms of widlife diseases, 3
Paraspidodera, 394
Parastrigea, 231
Parastrigea tulipoides, 237
Paratanaisia, 230
Paratanaisia bragai, 228, 237, 238
Paratrichosoma, 463
Paridae (Chickadees and Tits), 61, 444
paromomycin, 200
Paronchocerca spp., 440, 441–445, 446, 448, 452, 454
Paronchocerca bambusicolae, 449
Paronchocerca bumpae, 449
Paronchocerca ciconarium, 454
Paronchocerca ciconiarum, 449, 455
Paronchocerca francolina, 449
Paronchocerca helicina, 450
Paronchocerca ibanezi, 449
Paronchocerca limboonkengi, 449
Paronchocerca mansoni, 449
Paronchocerca mirzai, 449
Paronchocerca rousseloti, 449, 450
Paronchocerca schelupovi, 449
Paronchocerca sonini, 449
Paronchocerca straeleni, 449
Paronchocerca struthionus, 456
Paronchocerca tonkinensis, 449
Parorchis, 230, 231
Parorchites zederi, 264, 268
Partridge (*Perdix spp*), 168
Parulidae (New World Warblers), 60, 435, 444
Parus major (Great Tit), 5, 47, 207, 482
Parus monticolus (Green-backed Tit), 482
Paruterina, 265
Paruterinidae, 264
Passer, 465
Passer spp.
Passer domesticus biblicus (Israeli House Sparrow), 24
Passer domesticus (House Sparrow), 22, 206, 209, 333, 345, 401, 452, 530
Passer hispaniolensis (Spanish Sparrow), 482
Passer montanus (Eurasian Tree Sparrow), 111, 206, 209
Passerella, 465
Passerella iliaca (Fox Sparrow), 482
Passeridae (Old World Sparrows), 60
Passeriformes (perching birds), 14, 22, 24, 60, 123, 136, 163, 170, 206, 208, 218, 238, 247, 252, 253, 277, 297, 332, 376, 377, 378, 435, 443, 465, 479, 548, 549
Passerini's Tangara (*see Ramphocelus passerinii*)
Passeromyia heterochaeta, 548
Passeromyia indecora, 548
Pasteurella multocida, 521
Pastor roseus (Rosy Starling), 484
Patagioenas spp.
Patagioenas fasciata (Band-tailed Pigeon), 70, 125, 143, 398
Patagioenas leucocephala (White-crowed Pigeon), 125
Patogioenas speciosa (Scaled Pigeon), 398
Pavo cristatus (Indian Peafowl), 155, 331, 392, 396, 402
Pavo muticus (Green Peafowl), 391
Pavoncella, 465
Pearsonema, 463
pediculosis, 515
Pedionomidae (Plains-wanderer), 442

Pegosomum sp., 235
Pelecanidae (Pelicans), 441
Pelecaniformes, 14, 59, 163, 173, 174, 217, 235, 247, 251, 263, 292, 328, 441, 465, 466
Pelecanoididae (Diving-Petrels), 441
Pelecanus spp., 465
Pelecanus conspicillatus (Australian Pelican), 467
Pelecanus erythrorhynchos (American White Pelican), 293, 467
Pelecanus occidentalis (Brown Pelican), 235, 270, 293, 467
Pelecanus rufescens (Pink-backed Pelican), 270, 293
Pelecitus spp., 440, 441–445, 446, 448, 450–452, 454
Pelecitus ceylonensis, 451
Pelecitus fulicaeatrae, 445, 446, 451, 458, 521
Pelecitus tubercauda, 458
penguins (*see* Spheniscidae)
Perch (*Perca fluviatilis*), 298
Perdix, 465
Perdix perdix (Gray Partridge), 6, 35, 155, 156, 205, 217, 331, 391, 396, 405, 473
Perdix spp., 168
Peregrine Falcon (*see Falco peregrinus*)
Periparus ater (Coal Tit), 376
permethrin spray, 535
Pernis apivorus (European Honey-Buzzard), 79, 550
Phaenicopteridae (Flamingos), 441
Phaethontidae (Tropicbirds), 441
Phagicola, 230
Phagicola longa, 235
phagocytosis, of trichomonads, 141
Phalacrocoracidae (Cormorants), 59, 441
Phalacrocorax spp., 292, 416, 465
Phalacrocorax africanus (Long-tailed Cormorant), 292
Phalacrocorax aristotelis (European Shag), 227
Phalacrocorax auritus (Double-crested Cormorant), 173, 174, 176, 233, 235, 270, 292, 421, 466
Phalacrocorax bougainvillii (Guanay Cormorant), 270
Phalacrocorax brasilianus (Neotropic Cormorant), 467
Phalacrocorax carbo (Great Cormorants), 264, 292, 300, 328, 467
Phalacrocorax fuscescens (Black-faced Cormorant), 292, 467
Phalacrocorax melanoleucos brevirostris (Pied Cormorant), 290
Phalacrocorax melanoleucos (Little Pied Cormorant), 292, 467
Phalacrocorax pygmaeus (Pygmy Cormorant), 292, 304, 467
Phalacrocorax sulcirostris (Little Black Cormorant), 467
Phalacrodectes, 529
phanerozoites, 39
Phapitreron leucotis (White-eared Dove), 71
Phasianidae (Pheasants and Partridges), 59, 360, 442, 505
Phasianus spp., 154, 465
Phasianus colchicus (Ring-necked Pheasant), 6, 114, 154, 155, 163, 168, 169, 205, 213, 214, 233, 237, 331, 345, 347–350, 388, 392, 396, 405, 474, 488
Phasianus versicolor (Green Pheasant), 270, 391
Pheucticus ludovicianus (Rose-breasted Grosbeak), 333
Philenon corniculatus (Noisy Friarbird), 22
Philepittidae (Asities), 61, 444
Philippine Duck (*see Anas luzonica*)
Philomachus, 465
Philomachus pugnax (Ruff), 332
Philophthalmidae, 231, 236, 238
Philophthalmus spp., 231, 234, 236
Philophthalmus gralli, 234, 238, 240
Philophthalmus hegeneri, 238, 239
Philophthalmus megalurus, 239
Philopteridae, 516
Philopterus sp., 517
Philornis spp., 546, 553
Philornis carinatus, 548
Philornis deceptivus, 549
Philornis downsi, 549
Philornis mimicola, 549
Philornis pici, 549
Philornis porteri, 549
Philornis seguyi, 549
Phimosus infuscatus (Bare-faced Ibis), 296
Phoca caspica (Caspian seal), 302
Phocascaris, 415
Phoebastria immutabilis (Laysan Albatross), 527, 530, 535
Phoenicopteriformes (Flamingos), 23, 56, 59, 163, 247, 251, 328
Phoenicopterus chilensis (Chilean Flamingo), 328
Phoenicopterus minor (Lesser Flamingo), 23, 114
Phoeniculidae (Woodhoopoes), 443
Phoenicurus, 465
photoperiod, 7
physiological trade-offs, in relation to parasitism, 5
Phytotomidae (Plantcutters), 444
Pica, 465
Pica hudsonia (Black-billed Magpie), 451, 482
Pica pica pica (Eurasian Magpie), 345, 451, 482
Picathartidae (Rockfowl), 61
Picidae (Woodpeckers), 60, 435, 443
Piciformes (Woodpeckers), 14, 60, 163, 169–170, 218, 332, 435, 443, 465, 478, 548
Picui Ground-Dove (*see Columbina picui*)
Piculus rubiginosus (*see* Golden-olive Woodpecker)
Picus canus (*see* Gray-faced Woodpecker)
Picus viridis (*see* Green Woodpecker)
Pied Avocet (*see Recurvirostra avosetta*)
Pied-billed Grebe (*see Podilymbus podiceps*)
Pied Cormorant (*see Phalacrocorax melanoleucos brevirostris*)
Pied Currawong (*see Strepera graculina*)
Pied Imperial-Pigeon (*see Duchula bicolor*)
Pied Stilt (*see Himantopus leucocephalus*)
Pig frog (*Rana grylio*), 303
Pigeon Guillemot (*see Cepphus columba*)
Pileated Woodpecker (*see Dryocopus pileatus*)
pillbugs, 327
Pine snake (*Pituophis melanoleucus*), 302
Pink-backed Pelican (*see Pelecanus rufescens*)
Pink Cockatoo (*see Cacatua leadbeateri*)
Pink-footed Goose (*see Anser brachyrhynchus*)
Pink-necked Pigeon (*see Treron vernans*)
Pink Pigeon (*see Nesoenas mayeri*)
Pinnated Bittern (*see Botaurus pinnatus*)
Pionus spp.
Pionus fuscus (Dusky Parrot), 399
Pionus maximiliani (Scoly-headed Parrot), 400
Pionus menstruus (Blue-headed Parrot), 399
piperazine, 491
piperazine dihydrochloride, 424
piperazine sulfate, 371
piperonyl butoxide, 521–522
Pipile jacutinga (Black-fronted Piping-Guan), 395
Pipilo erythrophthalmus (Eastern Towhee), 483
Pipridae (Manakins), 61, 444

Piranga olivaceai (Scarlet Tanager), 483
Piranga rubra (Summer Tanager), 483
piranhas (*Serrasalmus nattereri*), 290
Piscicapillaria, 463
Pittidae (Pittas), 60, 444
Placobdella spp.
Placobdella costata, 505
Placobdella novabritanniae, 505
Placobdella ornata, 502, 505
Placobdella papillifera, 502, 505
Placobdelloides jaegerskioeldi, 501
Placobdelloides maorica, 502, 505
Plagiorchiidae, 231, 237
Plagiorchis, 231
Plagiorchis elegans, 237
Plagiorhynchus cylindraceus, 280, 281, 282, 283
from an American Robin (*Turdus migratorius*), 279
in an American Robin (*Turdus migratorius*), 282
Plagiorhynchus species, 277
Plain Chachalaca (*see Ortalis vetula*)
Plain Wren (*see Thryothorus modestus*)
plasmochin, 28
Plasmodiidae, 36
Plasmodioides, 36
Plasmodium spp., 15, 17, 27, 35, 54
from the Australian region, 36
clinical signs of infection, 42–43
domesticated animal health concerns, 47
gold standard for diagnosis of infection, 45–46
immunity, 46–47
infection relapse, 20
infection through blood inoculation, 46
intrinsic and extrinsic factors of distribution, 41
management implications, 48–49
mortality from, 48
natural vectors of, 41
Plasmodium anasum, 37
Plasmodium ashfordi, 37
Plasmodium bertii, 37
Plasmodium cathemerium, 36, 37
infections with, 44
Plasmodium circumflexum, 36, 37, 38, 41
erythrocytic stages of, 38
Plasmodium columbae, 37
Plasmodium coturnixi, 37
Plasmodium dissanaikei, 37
Plasmodium durae, 37
Plasmodium durae (domestic turkeys), 37, 42, 45, 48
Plasmodium elongatum, 36, 37, 40, 41–42
Plasmodium fallax, 37
Plasmodium formosanum, 37
Plasmodium forresteri, 37
Plasmodium gabaldoni, 37
Plasmodium gallinaceum, 37, 39, 40, 41–43, 43, 48
Plasmodium garnhami, 37
Plasmodium giovannolai, 37
Plasmodium (*Giovannolaia*) sp., 41
Plasmodium griffithsi, 37
Plasmodium gundersi, 37
Plasmodium hegneri, 37
Plasmodium hermani, 37, 40, 41, 47
Plasmodium hexamerium, 37
Plasmodium huffi, 37
Plasmodium juxtanucleare, 37, 41–42
Plasmodium kempi, 36, 37
Plasmodium leanucleus, 37
Plasmodium lophurae, 37
Plasmodium lutzi, 37
Plasmodium matutinum, 37
Plasmodium (*Novyella*) sp., 41
Plasmodium nucleophilum, 36, 37
Plasmodium octamerium, 37
Plasmodium paranucleophilum, 37
Plasmodium parvulum, 37
Plasmodium pedioecetae, 37
Plasmodium pinottii, 37
Plasmodium polare, 37
Plasmodium relictum, 7, 35, 36, 37, 40, 41–42, 44, 45, 46, 48
Plasmodium rouxi, 36, 37, 41
Plasmodium subpraecox, 37
Plasmodium tejerai, 37
Plasmodium vaughani, 36, 37
prevalence of, 41–42
public health concerns, 47
spatial and seasonal patterns of transmission, 40
and stress-mediated changes in the immune system, 40
subgenera and species of avian, 37
treatment and control, 48
wildlife population impacts, 47–48
Platalea, 465
Platalea ajaja (Roseate Spoonbill), 290, 296, 468
Platalea alba (African Spoonbill), 296
Platalea leucorodia (Eurasian Spoonbill), 296
Platycercus elegans (Crimson Rosella), 218, 399
Platycercus eximius (Eastern Rosella), 399
Platyhelminthes, 262
Platynosomum, 229
Plegadis, 465
Plegadis falcinellus (Glossy Ibis), 296
Ploceidae (Weavers), 60, 444
Ploceus qurantius (Orange Weaver), 378
plumage coloration, 21
Plum-headed Finch (*see Neochmia modesta*)
Pluvialis, 465
pneumonitis, 116
Podager nacunda (Nacunda Nighthawk), 401
Podargidae (Frogmouths), 59, 443
Podiceps, 465
Podiceps auritus (Horned Grebe), 262, 292, 328
Podiceps cristatus (Great Crested Grebe), 291, 328
Podiceps grisegena (Red-necked Grebe), 328, 466, 521
Podiceps nigricollis (Eared grebe), 466
Podicipedidae (Grebes), 360, 441, 505
Podicipediformes (Grebes), 14, 56, 59, 163, 252, 263, 291, 328, 360, 441, 465, 466, 521
Podilymbus podiceps (Pied-billed Grebe), 262, 466
podoacariasis, 527
Poecile gambeli (Mountain Chickadee), 4
polar bodies, 112
Polyangium, 230
Polydelphis, 425
polymerase chain reaction (PCR), 25, 41, 46, 109, 140, 425
for *Eimeria* species, 190
Polymorphus spp., 277, 279
Polymorphus botulus, 285
Polymorphus magnus, 283
Polymorphus mathevossianae, 285
Polymorphus minutus, 280, 282, 283, 285
Polyplectron bicalcaratum (Gray Peacock-Pheasant), 391
Polytelis spp.
Polytelis alexandrae (Alexandra's Parrot), 332, 398
Polytelis anthopeplus (Regent Parrot), 217, 400
Polytelis swainsonii (Superb Parrot), 217

Pomatostomus superciliosus (White-browed Babbler), 483
Porcellionides pruinosus, 327
Porcellio scaber, 327
Porphyrio martinica (Purple Gallinule), 475
Porrocaecum, 339, 413, 415–416, 419, 423, 425
  Porrocaecum, host range of species of, 418
  Porrocaecum angusticolle, 416, 419
  Porrocaecum crassum, 416, 419
  Porrocaecum depressum, 415, 416, 419, 421
  Porrocaecum ensicaudatum, 416, 419, 421
Port Lincoln Parrot (*Barnardius zonarius*)
Porzana porzana (Spotted Crake), 361
Postharmostomum gallinum, 237
Posthodiplostomum, 229
Prairie Chicken (*see Tympanuchus* spp.)
Prairie Falcon (*see Falco mexicanus*)
praziquantel, 255, 352
predation, 5
preerythrocytic merogony, 19
preerythrocytic meronts, 15
preerythrocytic meronts, from avian hosts, 22–24
preerythrocytic stages, of the parasite, 21
premunition, 46
prepatent period ranges
  haemoproteids transmitted by *Culicoides*, 19–20
  haemoproteids transmitted by hippoboscid flies, 20
primaquine, 28
primaquine phosphate, 48
Primolius maracana (Blue-winged Macaw), 233
Prionopidae (Helmetshrikes), 60
Procellariidae (Sheawaters and Petrels), 441, 505
Procellariiformes (Albatrosses and Petrels), 14, 56, 59, 163, 173, 174, 441
Profilicollis altmani, 285
Profilicollis botulus, 282
Progne subis (Purple Martin), 28, 543
Pronocephalidae, 231
Prosimulium spp., 543
  Prosimulium decemarticulatum, 85
  Prosimulium hirtipes, 85
Prosobrachia, 247
prospective case-control studies, 4
Prosthogonimidae, 231, 235, 237
Prosthogonimus, 231
  Prosthogonimus cuneatus, 226
  Prosthogonimus macrorchis, 237
  Prosthogonimus ovatus, 226
Protocalliphora spp., 547, 549, 550, 553
  Protocalliphora aenea, 548
  Protocalliphora asiovora, 548
  Protocalliphora avium, 548, 551
  Protocalliphora azurea, 548
  Protocalliphora beameri, 548
  Protocalliphora bennetti, 548
  Protocalliphora bicolor, 548
  Protocalliphora braueri, 548
  Protocalliphora braueri (Blow Fly) larvae, 5
  Protocalliphora brunneisquama, 548
  Protocalliphora chrysorrhoea, 548
  Protocalliphora cuprina, 548
  Protocalliphora deceptor, 548
  Protocalliphora downsi, 551
  Protocalliphora falcozi, 548
  Protocalliphora fallisi, 548
  Protocalliphora halli, 548
  Protocalliphora hesperia, 548
  Protocalliphora hesperioides, 548
  Protocalliphora hirundo, 548
  Protocalliphora interrupta, 548
  Protocalliphora isochroa, 548
  Protocalliphora lata, 548
  Protocalliphora lindneri, 548
  Protocalliphora metallica, 548
  Protocalliphora occidentalis, 548
  Protocalliphora parorum, 548
  Protocalliphora peusi, 548
  Protocalliphora rugosa, 548
  Protocalliphora seminuda, 548
  Protocalliphora shannoni, 548
  Protocalliphora sialia, 548
  Protocalliphora spatulata, 548
  Protocalliphora spenceri, 548
  Protocalliphora tundrae, 548
proventricular worms, 326
Prunella collaris (Alpine Accentor), 483
Prunella modularis (Dunnock), 483
Prunellidae (Accentors), 60, 444
Psephotus haematonotus (Red-rumped Parrot), 400
Pseudamphimerus, 231
Pseudlemdana, 441–445, 448, 454
Pseudocapillaria, 463
Pseudocapillarioides, 463
Pseudolynchia brunnea, 18
Pseudolynchia canariensis, 18
Pseudomenopon pilosum, 521
Pseudomenopon sp., 521
Pseudomonas aeruginosa, 139
Pseudomonas spp., 434
Pseudophyllidea, 262
Pseudoterranova, 423
Psilochasmus, 231
Psilochasmus oxyurus, 239
Psilostomatidae, 231, 236, 237, 238
Psilostomum, 231
Psittacidae (Parrots), 59, 442
Psittaciformes, 14, 18, 23, 59, 121, 136, 139, 163, 168, 169, 206, 217, 332, 442, 459, 465, 476, 549
Psittacula spp.
  Psittacula eupatria (Alexandrine Parakeet), 459
  Psittacula krameri (Rose-ringed Parakeet), 400
  Psittacula longicauda (Long-tailed Parakeet), 400
  Psittacula roseata (Blossom-headed Parakeet), 13, 23
Psophiidae (Trumpeters), 442
Pteroclididae (Sandgrouse), 442
Pterocliformes, 59
Pterocliformes (Sandgrouse), 56
Pteroglossus aracari (Black-necked Aracari), 479
Pteronetta hartlaubii (Hartlaub's Duck), 329
Pterothominx spp., 463
  Pterothominx alpina, 475
  Pterothominx blomei, 473, 474
  Pterothominx bursata, 463, 465, 466, 486
  Pterothominx caudinflata, 463, 464, 465, 466, 473, 474, 486, 488, 491
  Pterothominx exilis, 468, 479, 480, 481, 483, 484, 485
  Pterothominx longifilla, 479, 483
  Pterothominx meleagridis, 473, 474
  Pterothominx moraveci, 464, 476
  Pterothominx philippinensis, 464, 488, 490–491
  Pterothominx totani, 475, 476
Ptilogonatidae (Silky-flycatchers), 61, 435
Ptilonorhynchidae (Bowerbirds), 61, 445

Ptilonorhynchus violaceus (Satin Bowerbird), 218, 518
Puerto Rican Parrot (*see Amazona vittata*)
Puffinus tenuirostris (Short-tailed Shearwater), 173, 174, 267
Pulchrosoma pulchrosoma, 238
pulmonary arteries, 449
pulmonary congestion, 116
pulmonary edema, 116
Pulmonata, 247
Pumpkin-seed (*Lepomis gibbosus*), 298
Purple Gallinule (*see Porphyrio martinica*)
Purple Honeycreeper (*see Cyanerpes caeruleus*)
Purple Martin (*see Progne subis*)
Purple Sandpiper (*see Colidris maritima*)
Pycnonotidae, 444
Pycnonotus jocosus (Red-whiskered Bulbul), 218
Pygmy Cormorant (*see Phalacrocorax pygmaeus*)
Pynonotidae (Bulbuls), 61
pyrantel, 371
pyrethrin solution, 544, 554
pyrethrum, 521
pyrimethamine, 28
pyrimethamine (Daraprim), 216, 219
pyrimethamine–sulfadoxine combinations, 28, 48
Pyrrhocorax graculus (Yellow-billed Chough), 483
Pyrrhula spp.
  Pyrrhula pyrrhula (Eurasian Bullfinch), 116
  Pyrrhura leucotis (White-eared Parakeet), 233, 400
  Pyrrhura molinae (Green-cheeked Parakeet), 399
Pytilia hypogrammica (Red-Faced Pytilia), 195

Quadraceps ridgwayi, 516
quinine derivatives, 95
Quiscalus spp.
  Quiscalus major (Boat-Tailed Grackle), 113, 327, 333
  Quiscalus mexicanus (Great-tailed Grackle), 209
  Quiscalus niger (Greater Antillean Grackle), 333
  Quiscalus quiscula (Common Grackle), 209, 451, 454, 483

Raccoon Dog (*Nyctereutes procyonoides*), 302
Raccoon (*Procyon lotor*), 425
raccoon roundworm encephalitis, 424
Radjah Shelduck (*see Tadorna radjah*)
Raillietina spp., 263, 264, 268
  Raillietina echinobothrida, 271
  Raillietina laticanalis, 270
Rainbow Lorikeet (*see Trichoglossus haematodus moluccanus*)
Rallidae (Rails, Gallinules, and Coots), 59, 361, 442, 505
Rameron Pigeon (*see Columba arquatrix*)
Ramphastidae (Toucons), 443
Ramphastos, 465, 516
  Ramphastos sulfuratus (Keel-billed Toucan), 114–115
  Ramphastos toco (Toco Toucan), 169, 479
  Ramphastos vitellinus (Channel-billed Toucan), 479
Ramphocaenus melanurus (Long-billed Gnatwren), 332
Ramphocelus spp.
  Ramphocelus dimidiatus (Crimson-backed Tanager), 209
  Ramphocelus passerinii (Passerini's Tangara), 111
  Ramphocelus carbo (Silver-beaked Tanager), 111
Rana spp.
  Rana catesbeiana (Bullfrog), 303
  Rana megapoda (Bigfoot leopard frog), 303
  Rana ridibunda (Lake frog), 303
Raphidascaris, 413
Razorbill (*see Alca torda*)
Recurvirostra, 465
  Recurvirostra americana (American Avocet), 476
  Recurvirostra avosetta (Pied Avocet), 361
Recurvirostridae (Avocets and Stilts), 59, 361, 442, 505
Red-and-green Macaw (*see Ara chloropterus*)
Red-and-yellow Barbet (*see Trachyphous erythrocephalus*)
Red-bellied Woodpecker (*see Melanerpes carolinus*)
Red-billed Curassow (*see Crax blumenbachii*)
Red-billed Duck (*see Anas erythrorhyncha*)
Red-breasted Goose (*see Branta ruficollis*)
Red-breasted Merganser (*see Mergus serrator*)
Red Collared-Dove (*see Streptopelia tranquebarica*)
Red-crested Bustard (*see Eupodotis ruficrista*)
Red-crested Pochard (*see Netta rufina*)
Red-crowned Crane (*see Grus japonensis*)
Red-eyed Dove (*see Streptopelia semitorquata*)
Red-faced Mouse Bird (*see Colius erythromelon*)
Red-Faced Pytilia (*see Pytilia hypogrammica*)
Red-footed Booby (*see Sula sula*)
Red-fronted Parakeet (*see Cyanoramphus novaezelandiae*)
Red Grouse (*see Lagopus lagopus scotica*)
Redhead (*see Aythya americana*)
Red-headed Woodpecker (*see Melanerpes erythrocephalus*)
Red Junglefowl (*see Gallus gallus*)
Red Kite (*see Milvus milvus*)
Red Knot (*see Calidris canutus*)
Red-legged Honeycreeper (*see Cyanerpes cyaneus*)
Red-legged Partridge (*see Alectoris rufa*)
Red-legged Seriema (*see Cariama cristata*)
Red-lored Parrot (*see Amazona autumnalis*)
Red Lorry (*see Eos bornea*)
red mite (*Dermanyssus gallinae*), 109
Red-necked Falcon (*see Falco chicquera*)
Red-necked Francolin (*see Francolinus afer*)
Red-necked Grebe (*see Podiceps grisegena*)
Red-necked Parrot (*see Amazona arausiaca*)
Red-rumped Parrot (*see Psephotus haematonotus*)
Redshank (*see Tringa totanus*)
Red-shouldered Hawk (*see Buteo lineatus*)
Red Spurfowl (*see Galloperdix spadicea*)
Red-tailed Hawk (*see Buteo jamaicensis*)
Red-throated Loon (*see Gavia stellata*)
Red-whiskered Bulbul (*see Pycnonotus jocosus*)
Redwing (*see Turdus iliacus*)
Red-winged Blackbird (*see Agelaius phoeniceus*)
Red-winged Parrot (*see Aprosmictus erythropterus*)
Red-winged Tinamou (*see Rhynchotus rufescens*)
Reed Bunting (*see Embesiza schoeniclus*)
Reeves' Pheasant (*see Syrmaticus reevesii*)
Regent Bowerbird (*see Sericulus chrysocephalus*)
Regent Parrot (*see Polytelis anthopeplus*)
Regulidae (Kinglets), 61
Remizidae (Penduline Tits), 444
renal cryptosporidiosis, 198
Renicola lari, 236
Renicola pinguis (Troglotrematidae), 226
Renicolidae, 236
Renicolla, 231
repair or replace costs, to the host, 4
residuum, 112
resource trade-offs, against parasitism, 5
respiratory cryptosporidiosis, 198
reticuloendothelial cells, in bone marrow, 35
retrospective case–control studies, 4
Rhabdornithidae, 444
Rhadinea merremii (Graceful brown snake), 303
Rhea americana (Greater Rhea), 345, 389, 395, 466

Rheidae (Rheas), 441
Rheiformes, 163, 441
Rhinocryptidae (Topaculos), 443
Rhinonyssus rhinolethrum, 528, 533
Rhizothera longirostris (Long-billed Partridge), 392
Rhynchophthirina, 516
Rhynchotus rufescens (Red-winged Tinamou) 389, 395
Rhynochetidae (Kagu), 442
Ribeiroia ondatrae, 236, 237
Ribeiroia sp., 231, 239
Richardsonianus howensis, 505
Ricinidae, 516
Ricinus sp., 517
Ring-billed Gull (*see Larus delawarensis*)
Ringed Kingfisher (*see Megaceryle torquatus*)
Ring-necked Dove (*see Streptopelia capricela*)
Ring-necked Duck (*Aythya collaris*)
Ring-necked Pheasant (*see Phasianus colchicus*)
Ring Turtle-Dove (*see Streptopelia* risoria)
Ringed Teal (*see Collonetta leucophrys*)
Rissa, 465
River otter (*Lutra canadensis*), 302
robenidin, 177
Rock Partridge (*see Alectoris graeca*)
Rock pigeon (*see Columba livia*)
Rock Ptarmigan (*see Lagopus muta*)
rodents, 425
Rollulus rouloul (Crested Partridge)
ronidazole, 144
Rook (*see Corvus frugilegus*)
Roseate Spoonbill (*see Platalea ajaja*)
Rose-ringed Parakeet (*see Psittacula krameri*)
Ross' Goose (*see Chen rossii*)
Rostratula benghalansis (Greater Painted-Snipe), 488
Rostratulidae (Painted Snipes), 59, 442
Rostrhamus sociabilis plumbeus (Florida Snail Kite), 236
Rostrhamus sociabilis (Snail Kite), 233
Rosy-Billed Pochord (*see Netta peposoca*)
Rosy Starling (*see Pastor roseus*)
Rouen ducks, 249
Rough-legged Hawk (*see Buteo lagopus*)
Royal Tern (*see Thalasseus maximus*)
Ruddy Duck (*see Oxyura jamaicensis*)
Ruddy Ground-Dove (*see Columbina talpacoti*)
Ruddy Quail-Dove (*see Geotrygon montana*)
Ruddy Shelduck (*see Tadorna ferruginea*)
Rufescent Tiger-Heron (*see Tigrisoma lineatum*)
Ruff (*see Philomachus pugnax*)
Ruffed Grouse (*see Bonasa umbellus*)
Rufous-chested Sparrowhawk (*see Accipiter rufiventris*)
Rufous Night Heron (*see Nycticorax caledonicus*)
Rynchopidae (Skimmers), 442

Sacred Kingfisher (*see Todiramphus sanctus*)
Sage Thrasher (*see Oreoscoptes montanus*)
Sagittariidae (Secretary birds), 59, 441
Sagittarius serpentarius (Secretory-bird), 80, 131
Saker Falcon (*see Falco cherrug*)
salinomycin, 177
Salmonella gallinarum, 521
Salmonella typhimurium, 170
Sandhill Crane (*see Grus canadensis*)
Sarcocystis spp., 25, 163, 204, 214
  clinical signs, 116
  diagnosis, 116
  domestic animal health concerns, 117
  epizootology, 113–116
  etiology of infection, 113
  host range and distribution, 113
  immunity, 116
  pathogenesis and pathology, 116
  public health concerns, 116
  Sarcocystis accipitris, 114
  Sarcocystis alectoributeonis, 114
  Sarcocystis alectorivulpes, 114
  Sarcocystis ammodrami, 114
  Sarcocystis aramidis, 114
  Sarcocystis buteonis, 114
  Sarcocystis cernae, 114
  Sarcocystis cheeli, 114
  Sarcocystis citellibuteonis, 114
  Sarcocystis colii, 114
  Sarcocystis dispersa, 114
  Sarcocystis espinosai, 114
  Sarcocystis falcatula, 113, 114, 115–116, 214
    in Brown-headed Cowbird (*Molothrus ater*), 115
    from feces of Virginia Opossum (*Didelphis virginiana*), 115
    like infections, 214
    in Lory (*Lorinus* sp.) elongate meront of, 115
  Sarcocystis garzettae, 114
  Sarcocystis glareoli, 114
  Sarcocystis horvathi, 114
  Sarcocystis jacarinae, 114
  Sarcocystis kaiserae, 114
  Sarcocystis kirmsei, 114
  Sarcocystis nontenella, 114
  Sarcocystis oliverioi, 114
  Sarcocystis peckai, 114
  Sarcocystis phoeniconaii, 114
  Sarcocystis ramphastosi, 114
  Sarcocystis rauschorum, 115
  Sarcocystis rileyi, 115
  Sarcocystis scotti, 115
  Sarcocystis sebeki, 115
  Sarcocystis setophagae, 115
  Sarcocystis spaldingae, 115
  Sarcocystis sulfuratusi, 115
  Sarcocystis wenzeli, 115
  synonyms, 113
  that parasitize birds, 114–115
  treatment and control, 117
  wildlife population impacts, 117
sarcocystosis, 116
Sarconema, 441–445, 448, 454
  Sarconema eurycera, 520, 521
  Sarconema eurycerca, 450, 451, 458
Sarcophagidae (flesh flies), 550
Sarcoramphus papa (King Vulture), 390
Sarkidiornis melanotos (Comb Duck), 329, 362
Sarus Crane (*see Grus antigone*)
Satin Bowerbird (*see Ptilonorhynchus violaceus*)
Satyr Tragopan (*see Tragopan satyra*)
Scaled Pigeon (*see Patagioenas speciosa*)
Scaled Quail (*see Callipepla squamata*)
Scaly-headed Parrot (*see Pionus maximiliani*)
Scarlet-chested Parrot (*see Neophema splendida*)
Scarlet Macaw (*see Ara macao*)
Scarlet Tanager (*see Piranga olivaceai*)
Schistocephalus, 267
Schistocephalus solidus, 264, 267
Schistogonimus, 231
Schistosoma japonicum, 247
Schistosomatidae, 231, 246

Schistosome dermatitis, 255
schistosomes
  anatomical locations of infection, 249
  clinical signs, 249
  diagnosis, 254
  domesticated animal health concerns, 255
  epizootiology, 248–249
  etiology, 247–248
  history, 246
  host range and distribution, 246–247
  host responses to eggs, 250
  immunity, 254
  known intermediate and final hosts of avian, 247
  location of the parasite in the host, geographic location, vertebrate host order, and selected references, 251–253
  male and female, 248
  managment implications, 256
  pathogenesis and pathology, 249–254
  public health concerns, 255
  synonyms, 246
  treatment and control, 255–256
  wildlife population impacts, 255
schistosomiasis, 246, 254
Schistotaenia spp., 268
  Schistotaenia scolopendra, 264, 268
  Schistotaenia srivastavi, 264, 268
  Schistotaenia tenuicirrus, 262, 264, 268
Schulmanela, 463
scolices, of cyclophyllidean species, 268
Scolopacidae (Sandpipers), 59, 361, 442, 505
Scolopax minor (American Woodcock), 464, 550
Scopidae (Hamerkop), 441
Secretary-bird (*see Sagittarius serpentarius*)
Sedge Warbler (*see Acrocephalus schoenobaenus*)
See-see Partridge (*see Ammoperdix griseogularis*)
sentinel birds, 86
Sericulus chrysocephalus (Regent Bowerbird), 218
Serinus canaria (Island Canary), 6, 108, 114, 204, 212, 216, 218
serologic tests, 204
Serratospiculiasis, 436
Serratospiculoides spp.
  clinical signs, 436
  diagnosis, 437
  epizootiology, 435–436
  etiology, 435
  history, 434
  host range and distribution, 434–435
  pathology, 436
  public and domestic animal health concerns, 437
Serratospiculoides alii, 435
Serratospiculoides amaculata, 434
  treatment and control, 437
  wildlife population impacts, 437
Serratospiculum spp.
  clinical signs, 436
  diagnosis, 437
  epizootiology, 435–436
  etiology, 435
  history, 434
  host range and distribution, 434–435
  pathology, 436
  public and domestic animal health concerns, 437
Serratospiculum amaculata, 434
Serratospiculum amaculatum, 435
  treatment and control, 437
  wildlife population impacts, 437
Setophaga ruticilla (American Redstart), 115
sexual cycle, of *Haemoproteus* species, 17
Seychelles Blue-Pigeon (*see Alectroenas pulcherrima*)
Sharp-shinned Hawk (*see Accipiter striatus*)
Sharp-tailed Grouse (*see Tympanuchus phasianellus*)
Sheather's sugar flotation, 116, 176
Shikra (*see Accipiter badius*), 130
Short-eared Owl (*see Asio flammeus*)
Short-tailed Shearwater (*see Puffinus tenuirostris*)
Short-toed Eagle (*see Circaetus gallicus*)
Sialia mexicana (Western Bluebird), 282
Sialia sialis (Eastern Bluebird), 333, 483
Siamese Fireback (*see Lophura diardi*)
Siberian Rubythroat (*see Luscinia calliopei*)
Silver-beaked Tanager (*see Ramphocelus carbo*)
Silver Gull (*see Larus novaehollandiae*)
Silver Pheasant (*see Lophura nycthemera*)
Silver Teal (*see Anas versicolor*)
Simocephalus vetulus, 335
Simuliidae, 451
Simulium spp.
  Simulium adersi, 85
  Simulium anatinum, 85
  Simulium angustitarse, 85
  Simulium annulus, 538, 542
  Simulium aureum, 85
  Simulium congareenarum, 85
  Simulium croxtoni, 85
  Simulium equinum, 541
  Simulium fallisi, 85
  Simulium impukane, 85
  Simulium innocens, 85
  Simulium jenningsi, 85
  Simulium latipes, 85
  Simulium lineatum, 541
  Simulium meridionale, 85, 543
  Simulium metatarsale, 85
  Simulium minus, 85
  Simulium nyasalandicum, 85
  Simulium parnassum, 85
  Simulium pictipes, 85
  Simulium quebecense, 85
  Simulium rendalense, 85
  Simulium rugglesi, 85, 539
  Simulium slossanae, 85
  Simulium slossonae, 87
  Simulium venustum, 85
  Simulium vittatum, 85, 541
    Simulium vittatum, larva of, 539
    Simulium vittatum, pupa of, 540
  Simulium vorax, 85
Sitta europaea (Eurasian Nuthatch), 207, 483
Sittidae (Nuthatches), 61, 435, 444
Skrjabinocapillaria, 463
Slaty-breasted Wood-Rail (*see Aramides saracura*)
Small-billed Tinamou (*see Crypturellus parvirostris*)
Smew (*see Mergellus albellus*)
Smokey jungle frog (*Leptodactylus pentadactylus*), 303
Smooth-billed Ani (*see Crotophaga ani*)
Snail Kite (*see Rostrhamus sociabilis*)
Snapping turtle (*Chelydra serpentina*), 303
sneezing, 197
Snow Goose (*see Chen caerulescens*)

Snowy Egret (*see Egretta thula*)
Snowy Owl (*see Bubo scandiacus*)
Snowy Plover (*see Charadrius alexandrinus*)
sodium chloride, 509
sodium nitrate, 490
sodium pentachlorophenate, 255
Solitary Tinamou (*see Tinamus solitarius*)
Somateria spp., 465
  Somateria fischeri (Spectacled Eider), 67, 359
  Somateria mollissima (Common Eider), 5, 67, 165, 166, 174, 261, 282, 330, 336, 356, 362, 367, 470
    intestine of, 169
  Somateria spectabilis (King Eider), 327, 330, 358, 470
Song Thrush (*see Turdus philomelos*)
Sooty Grouse (*see Dendragapus fuliginosus*)
Southern Crowned-Pigeon (*see Goura scherpmakeri*)
Southwellina dimorpha, 285
Southwellina hispida, 279
sow bugs (*Porcellio scaber*), 402
Spanish Sparrow (*see Passes hispaniolensis*)
sparganosis, 271
Species Survival Plan, for the Bali Myna, 117
Spectacled Duck (*see Anas specularis*)
Speckled Pigeon (*see Columba quinea*)
Speckled Teal (*see Anas flavirestris*)
Spectacled Eider (*see Somateria fischeri*)
Spelotrema, 230
Sphaeridiotrema, 231
Sphaeridiotrema globulus (Psilostomatidae), 225, 226, 227–228, 236, 238–239, 240
Sphaerirostris, 279
Spheniscidae (Penguins), 59, 441, 505
Sphenisciformes, 14, 59, 163, 174, 175, 217, 235, 291, 441
Spheniscus spp.
  Spheniscus demersus (Jackass Penguin), 217, 291
  Spheniscus humboldti (Humboldt Penguin), 217
  Spheniscus magellanicus (Magellenic Penguin), 217, 416
Sphyrapicus varius (Yellow-bellied Sapsucker), 332
Spinicauda, 394
Spiny softshell turtle (*Apalone spinifer*), 303
Spirometra pleurocercoids, 270, 271
Spirometra sp., 264, 267
Spiroptera spp.
  Spiroptera crassicauda, 337
  Spiroptera nasuta, 327
  Spiroptera pectinifera, 337
  Spiroptera uncinata, 335
Spirurida, 439
Spizaetus cirrhatus (Changeable Hawk-Eagle), 76
spleen enlargement, 116
Splendidofilaria spp., 440, 441–445, 445, 446, 448, 451, 452, 454
  Splendidofilaria algonquinensis, 449, 452
  Splendidofilaria alii, 449
  Splendidofilaria bohmi, 450
  Splendidofilaria brevispiculum, 449
  Splendidofilaria californiensis, 446, 449, 451
  Splendidofilaria caperata, 449, 452, 457
  Splendidofilaria columbensis, 450
  Splendidofilaria falconis, 449
  Splendidofilaria fallisensis, 445, 446, 450, 451, 540
  Splendidofilaria gedoelsti, 450
  Splendidofilaria grettillati, 449
  Splendidofilaria hibleri, 450
  Splendidofilaria longicaudata, 449
  Splendidofilaria kasmirensis, 449
  Splendidofilaria mavis, 450
  Splendidofilaria osmaniae, 449
  Splendidofilaria pachacuteci, 449
  Splendidofilaria pavlovskyi, 449
  Splendidofilaria pectoralis, 450
  Splendidofilaria periarterialis, 449
  Splendidofilaria picacardina, 445, 446, 449, 451
  Splendidofilaria rotundicephala, 450
  Splendidofilaria singhi, 450
  Splendidofilaria skrjabini, 449
  Splendidofilaria smithi, 449, 450
  Splendidofilaria travassosi, 449
  Splendidofilaria tuvensis, 450
  Splendidofilaria verrucosa, 449
  Splendidofilaria wehri, 449
Splendidofilariinae, 448
splenic artery, 449
splenomegaly, 91
sporocyst
  of avian schistosomes, 248
sporocysts, 108, 164
  of *Sarcocystis*, 198
  trematodes, 226
sporogony, 40
  of *Eimeria gruis* and *Eimeria reichenowi*, 187
  Eimeria species, 164–165
  of *Isospora* species, 110
Sporozoasida, 204
sporozoites, 17, 39, 82, 164, 187–188
  development of, 19
  of *Isospora* species, 110
  species of *Cryptosporidium*, 196–197
Spot-billed Duck (*see Anas poecilorhyncha*)
Spotless Starling (*see Sturnus unicolor*)
Spotted Crake (*see Porzana porzana*)
Spotted Dove (*see Streptopelia chinensis*)
Spotted Eagle-Owl (*see Bubo africanus*)
Spotted Eagle (*see Aquila clanga*)
Spotted Imperial-Pigeon (*see* Ducula carole)
Spotted Nothura (*see Nothura maculosa*)
Spotted Owl (*see Strix occidentalis*)
Spotted Sandpiper (*see Actitis macularius*)
Spot-winged Wood Quail (*see Odontophorus capueira*)
spring relapse, 40, 83
Stabler-*gallinae* (SG) strain, 134
Steatornithidae (Oilbirds), 443
Stephanoprora, 230
Stercorariidae (Skuas), 442
Sterna, 465
Sterna hirundo (Common Tern), 205, 332
Sternidae (Terns), 59
Sternostoma trachaecolum, 528, 533
Sternostoma trachaecolum, dorsal and ventral views of adult female, 530
Stictonetta naevosa (Feckled Duck), 357
Stilbometopa impressa, 17, 18
Stock Dove (*see Columba oenas*)
Stomylotrema, 231
Stomylotrematidae, 231
Straw-necked Ibis (*see Threskiornis spinicollis*)
Strepera graculina (Pied Currawong), 24

Streptocara spp., 337
clinical signs, 338
diagnosis, 338–339
domestic animal health concerns, 339
eggs of, 337–338
epizootiology, 337–338
etiology, 337
history, 337
host range and distribution, 326, 328–333
life cycle of *S. incognita*, 337
pathogenesis and pathology, 338
Streptocara crassicauda, 326, 327, 328, 338
Streptocara crassicauda anseri, 337
Streptocara crassicauda charadrii, 337
Streptocara crassicauda crassicauda, 337
Streptocara crassicauda skrjabini, 337
Streptocara incognita (SI), 328
Streptocara pectinifera, 337
synonyms, 337
treatment and control, 339–340
wildlife population impacts, 339
Streptocariasis, 337
Streptococcus equinus, 521
Streptococcus fecalis, 157
Streptopelia spp., 168, 465
Streptopelia bitorquata (Island Collared-Dove), 73
Streptopelia capicola (Ring-necked Dove), 73
Streptopelia chinensis (Spotted Dove), 71, 126, 208, 398
Streptopelia decaocto (Eurasian Collared Dove), 73, 125, 143, 205, 361, 394, 398
Streptopelia decipiens (African Mourning Dove), 73
Streptopelia lugens (Dusky Turtle-Dove), 73
Streptopelia orientalis (Oriental Turtle-Dove), 72, 398
Streptopelia picturata (Madagascar Turtle-Dove), 126, 134
Streptopelia risoria (Ring Turtle-Dove), 125, 136, 140–142
esophageal junction of a, 140, 141, 142
Streptopelia semitorquata (Red-eyed Dove), 71, 125
Streptopelia senegalensis (Laughing Dove), 72, 114, 126, 206
Streptopelia tranquebarica (Red Collared Dove), 73
Streptopelia turtur (Eurasian Turtle-Dove), 72, 398
stress-mediated changes in the immune system, post malarial infection, 40
Striated Heron (*see Butorides striata*)
Striatofilaria, 441–445, 448, 454
Strigea, 231
Strigea falconis, 237
Strigea spp., 226
Strigeidae, 231, 236, 237
Strigidae (Owls), 59, 443
Strigiformes, 14, 24, 59, 163, 206, 208, 217, 443, 477, 548, 549
Strix spp.
Strix aluco (Tawny Owl), 21, 115, 133, 206, 208, 477, 550
Strix occidentalis (Spotted Owl), 477
Strix uralensis (Ural Owl), 477
Strix varia (Barred Owl), 133, 206, 213, 216–217, 346, 477
strobila, 269
Strongyluris, 394
Strongylus spp.
Strongylus mucronatus, 355
Strongylus pergracilis, 316
Strongylus serratus, 316
Struthio camelus (Ostrich), 57, 154, 196, 208, 240, 395, 516
Struthiofilaria, 441–445, 448, 454
Struthiofilaria megalocephala, 457
Struthionidae (Ostriches), 59, 441
Struthioniformes, 14, 59, 163, 208, 441, 466
Sturgeon (*Acipenser gueldenstaedtii*), 301
Sturnella magna (Eastern Meadowlark), 379
Sturnella neglecta (Western Meadowlark), 284, 379
Sturnia philippensis (Chestnut-cheeked Starling), 484
Sturnidae (Starlings), 61, 108, 435, 444
Sturnus, 465
Sturnus unicolor (Spotless Starling), 484
Sturnus vulgaris (European Starling), 206, 209, 281, 333, 345, 454, 484, 518, 534
subclinical impacts, of *Haemoproteus* infections, 28
subcutaneous tissues, 450
"sucking worm." *see* Trematodes
Sula sula (Red-footed Booby), 217
sulfachloropyrazine, 48
sulfachlorpyrazine (ESB3), 117
sulfachlorpyridazine (Vetisulid), 117
sulfadiazine, 216, 219
sulfadimethoxine, 192
sulfamethazine, 172
sulfamethoxazole, 95
sulfamonomethoxine, 48
sulfaquinoxaline, 172
sulfathiazole, 509
Sulidae (Boobies and Gonnets), 441
Summer Tanager (*see Piranga rubra*)
sunfish (Centrarchidae), 290
Sun Parakeet (*see Aratinga solstitialis*)
Superb Parrot (*see Potytelis swainsenii*)
Superb Starling (*Lamprotornis superbus*)
Surf Scoter (*see Melanitta perspicillata*)
Swainson's Hawk (*see Buteo Swainsoni*)
Swamp Francolin (*see Francolinus gularis*)
Swan Goose (*see Anser cygnoides*)
swans (*see Cygnus* spp.)
swimmer's itch, 246, 255
Sylviidae (Old World Warblers), 61, 435
syncytia, 82
syngamiasis, 343
Syngamidae, 343
Syngamus spp., 345
free-living hosts of, 347
prevalence of *S. trachea* in young rooks, 348
Syngamus alcyone, 345
Syngamus gibbocephalus, 345
Syngamus microspiculum, 345
Syngamus palustris, 345
Syngamus taiga, 345
Syngamus trachea, 343, 344, 347, 348, 349, 350, 351
Syngamus trachealis, 343
Syrmaticus reevesii (Reeves' Pheasant), 392
Syrmaticus soemmerringii (Copper Pheasant), 391

Tachybaptus, 465
Tachybaptus ruficollis (Little Grebe), 291, 360, 466
Tachycineta albilinea (Mangrove Swallow), 543
Tachycineta bicolor (Tree Swallow), 553
Tachymarptis melba (Alpine Swift), 6
Tachyphonus rufus (White-lined Tanager), 111, 380
tachyzoite, 210, 211, 212
Tadorna, 465
Tadorna ferruginea (Ruddy Shelduck), 329, 359, 365
Tadorna radjah (Radjah Shelduck), 359
Tadorna tadorna (Common Shelduck), 357, 363, 389

Taeniopygia guttata (Zebra Finch), 113, 378
Taiga Bean-Goose (*Anser fabalis*), 390
Tambourine Dove (*see Turtur typanistria*)
Tangara spp.
Tangara cayana (Burnished-buff Tanager), 111
Tangara chilensis (Paradise Tanager), 111
Tangara mexicana (Turquoise Tanager), 111
Tangara schrankii (Green-and-gold Tanager), 111
tapeworms. *see* Cestodes
Tataupa Tinamou (*see Crypturellus tataupa*)
Tawny Eagle (*see Aquila rapax*)
Tawny Owl (*see Strix aluco*)
T-cell-mediated immune response, 6
temephos, 94
Tenoranema, 463
Terranova sp., 423
Terek Sandpiper (*see Xenus cinereus*)
Tetrabothriidea, 262, 266
Tetrabothriidean scolices, 262
Tetrabothrius skoogi, 264, 269
Tetrabothrius spp., 263, 264, 267
tetracyclines, 28
Trameres spp., 337, 376
Tetrameres americana, 379, 381
Tetrameres cardinalis, 378
Tetrameres crami, 381
Tetrameres fissispina, 378, 381
Tetrameres globosa, 378
Tetrameres mohtedai, 379
in fowl, 379
Tetrameres pattersoni, 378, 381
Tetrameres striata, 379
Tetrameres strigiphila, 376
Tetrameres zakharowi, 381
tetrameridosis
clinical signs, 379
diagnosis, 380–381
domestic animal health concerns, 381
epizootiology, 377–379
etiology, 377
history, 376
host range and distribution, 376–377
immunity, 381
management implications, 381
pathology, 379–380
public health concerns, 381
treatment and control, 381
wildlife population impacts, 381
tetramisole, 406
Tetrao, 465
Tetrao urogallus (Eurasian Caporcaille), 214, 396, 404, 475
Tetrao parvirostris (Black-billed Capercaillie), 360
Tetrao tetrix (Black Grouse), 390, 396, 475
Tetrao urogallus (Eurasian Capercaillie), 215, 360, 391, 396, 475
Tetraogallus caspius (Caspian Snowcock), 390
Tetraogallus himalayensis (Himalayan Snowcock), 392, 396
Tetraonidae (Grouse, Ptarmigans, and Prairie Chickens), 59
Tetrathyridia (*Mesocestoides*), 269
Tetratrichomonas gallinarum, 140
Tetrax tetrax (Little Bustard), 393
Thalasseus, 465
Thalasseus maximus (Royal Tern), 238
Thapariella, 231
Thapariellidae, 231
Thayer's Gull (*see Larus thayeri*)
Thermocyclops hyalinus, 384, 385
Thermocyclops taihokuensis, 385
Theromyzon spp.
Theromyzon affinis, 505
Theromyzon bifarium, 505, 507
Theromyzon cooperi, 501, 505, 506, 508
Theromyzon garjaewi, 502, 505, 508
Theromyzon (Glossiphoniidae), 503
Theromyzon maculosum, 502, 505, 507
Theromyzon matthaii, 502, 505, 508
Theromyzon mollissimum, 505
Theromyzon pallens, 505
Theromyzon propinquum, 502, 505, 508
Theromyzon rude, 505, 507
Theromyzon sexoculatum, 505
Theromyzon tessellatoides, 505
Theromyzon tessulatum, 501–502, 504, 505, 507, 509
Theromyzon trizonare, 502, 504–507, 505
thiabendazole, 339, 352, 424
thick-walled megalomeronts, 19, 21
Thinocoridae (Seedsnipes), 442
thin-walled branching meronts, 22
thin-walled meronts, of *Haemoproteus*, 39
Thominx, 463
Thomix tridens, 473, 478, 479, 481, 482, 483, 484, 485
Thraupidae (Tanagers), 60, 435
Thraupis episcopus (Blue-gray Tanager), 111, 209, 333, 451
Thraupis palmarum (Palm Tanager), 206, 209
threadworm, 463
Three-toed amphiuma (*Amphiuma tridactylum*), 303
Threskiornis melanocephalus (Black-headed Ibis), 296
Threskiornis spinicollis (Straw-necked Ibis), 215
Threskiornithidae (Ibises and Spoonbills), 59, 441
Thryothorus ludovicianus (Carolina Wren), 332
Thryothorus modestus (Plain Wren), 208
Timaliidae (Babblers), 61
Tinamidae (Tinamou), 441
Tinamiformes (Tinamous), 14, 56, 59, 163, 441, 465, 466, 517
Tinamus solitarius (Solitary Tinamou), 389
Tinamus tao (Gray Tinamou), 389, 466
tissue injury cost, to the host, 4
Toco Toucan (*see Ramphastos toco*)
Todidae (Todies), 443
Todiramphus sanctus (Sacred Kingfisher), 24
toltrazuril (Baycox), 117, 200
Torgos tracheliotus (Lappet-faced Vulture), 74
Torresian Imperial-Pigeon (*see Ducula spilorrhoa*)
toucans (*Ramphastos* spp.), 516
Toxascaris leonina, 425
Toxocara spp., 425
Toxoplasma gondii, 204
clinical signs, 212
developmental stages of, 210–211
diagnosis, 214–215
distribution and host range, 204
DNA of, 214
epizootiology, 211–212
etiology, 204–211
history, 204
isolated strains, 210
isolation of *Toxoplasma gondii* from tissues of naturally infected wild birds, 205–207
isolation of, from tissues of naturally infected wild birds, 205–207
life cycle of, 211
oocysts, brain of birds fed, 214

Toxoplasma gondii (*Cont.*)
oocysts of, 211–212
pathogenesis and pathology, 212–213
public health and domestic animal concerns, 215
serologic prevalence of antibodies, 208–209
tachyzoites, 216
tissue cysts of, 214
tissue necrosis and, 212
treatment and control, 216–219
wildlife population impacts, 215–216
Toxoplasma gondii-specific antibodies, 211
Toxoplasma-like parasites in sparrows, 204
toxoplasmosis, 204, 215
toxoplasmosis, at necropsy from birds that died in captivity (C) or in the wild (W)., 217–218
trachea/esophagus, 449
tracheal worms
clinical signs, 349
diagnosis, 351
distribution and host ranges, 344
domestic animal health concerns, 351–352
eggs of, 347, 350
etiology, 343–344
history, 343
immunity, 351
infection, 343
life cycle and epizootiology, 344–349
pathogenesis and pathology, 349–351
public health concerns, 351
synonyms, 343
treatment and control, 352
wildlife population impacts, 351
Trachyphonus erythrocephalus (Red-and-yellow Barbet), 345
trade-offs, against parasitism, 5, 7, 28
Tragopan satyra (Satyr Tragopan), 393
Travassosascaris, 425
Tree Pipit (*see Anthus trivialis*)
Tree Swallow (*see Tachycineta bicolor*)
trematodes
anatomical location in avian hosts, 229–232
air sacs, 233
bile ducts and gall bladder, 233
eyes, 234
gastrointestinal tracts, 233–234
kidneys, 233
oviduct, 234
clinical signs, 228
density-dependent effects, 227
diagnosis, 234–239
digenetic, 225, 226
life cycle of, 228
domestic animal health concerns, 239
epizootiology, 226–228
etiology, 225–226
history, 225
host range and distribution, 226
immunity, 239
larval stages of, 227
management implications, 240
migrations and, 226
parasite families and species that can cause disease in wild birds, 235–238
pathogenicity of a, 228
pathology, 228–234
public health concerns, 239
synonyms, 225
treatment and control, 240
wildlife population impacts, 239–240
Treron spp.
Treron australis (Madagascar Green-Pigeon), 71
Treron calvus (African Green-Pigeon), 397
Treron phoenicopterus (Yellow-footed Pigeon), 398
Treron sieboldii (White-bellied Pigeon), 71
Treron sphenurus (Wedge-tailed Pigeon), 71
Treron vernans (Pink-necked Pigeon), 71
Trichinella sprialis, 491
Trichobilharzia spp., 247, 249, 250, 251, 255
Trichobilharzia alaskensis, 252
Trichobilharzia anatina, 252
Trichobilharzia adamsi, 251
Trichobilharzia arcuata, 249
Trichobilharzia aureliani, 249
Trichobilharzia australis, 249
Trichobilharzia berghei, 252
Trichobilharzia brantae, 252
Trichobilharzia brevis, 252
Trichobilharzia burnetti, 252
Trichobilharzia cameroni, 252
Trichobilharzia cerylei, 252
Trichobilharzia corvi, 252
Trichobilharzia duboisi, 249
Trichobilharzia elvae, 252
Trichobilharzia filiformis, 252
Trichobilharzia franki, 254
Trichobilharzia horiconensis, 252
Trichobilharzia indica, 252
Trichobilharzia jequitibaensis, 252
Trichobilharzia jianensis, 252
Trichobilharzia kegonsensis, 252
Trichobilharzia kossarewi, 252
Trichobilharzia kowalewskii, 252, 253
Trichobilharzia littlebi, 252, 253
Trichobilharzia maegraithi, 252
Trichobilharzia nasicola, 249
Trichobilharzia ocellata, 248–250, 253, 254–255
Trichobilharzia oregonesis, 252, 253
Trichobilharzia paoi, 253, 255
Trichobilharzia parocellata, 252
Trichobilharzia physellae, 253, 255
Trichobilharzia polonica, 252, 253
Trichobilharzia regenti, 247, 249–250
Trichobilharzia rodhaini, 249
Trichobilharzia salmanticensis, 252
Trichobilharzia schoutedeni, 252
Trichobilharzia spinulata, 249
Trichobilharzia stagnicola, 255
Trichobilharzia szidati, 250, 253
Trichobilharzia waubesensis, 252, 253
Trichodectidae, 516
Trichoglossus haematodus moluccanus (Rainbow Lorikeet), 218
Trichomonadida Order, 154
Trichomonas foetus, 139
in cattle, 140, 142
Trichomonas gallinae, 120–121, 142
in birds, 140
in columbiforms, 141
cultured trophozoites of, 134
in free-ranging and captive columbiforms, 122–129
from free-ranging and captive falconiforms and strigiforms, 130–133
life cycle of, 136
pathogenicity of strains of, 134

prevalences of, 136–137
transmission via contaminated water, 136
Trichomonas gallinae, distribution of, 121
Trichomonas gallinae, formation of pseudocysts of a strain of, 135
Trichomonas gallinae, gross lesion (arrow) of, 138
Trichomonas phasiani, 134
Trichomonas vaginalis, 139
in humans, 134, 142
trichomoniasis, 490
trichomonosis, 137, 143
clinical signs, 137
diagnosis, 139–141
distribution, 120
domestic animal health concerns, 142–143
epizootiology, 136–137
etiology, 121–136
history, 120
host range, 121
immunity, 141–142
management implications, 145
pathogenesis and pathology, 137–139
public health concerns, 142
synonym, 120
treatment and control, 144–145
wildlife population impacts, 143–144
Trichosomum contorta, 470
Trichosomum contortum, 471
trichostrongyliasis, 316
trichostrongylosis, 316
Trichostrongylus spp.
avian species of, 317
clinical signs, 320
diagnosis, 321
domestic animal health concerns, 321–322
epizootiology, 317–320
etiology, 317
history, 316–317
host infection by species of, 317
immunity, 321
impacts of disease, 321
larvae, 317, 320
life cycle of, 317–318
management implications, 322
pathology, 320–321
synonyms, 316
treatment and control, 322
wildlife population impacts, 322
Trichostrongylus cramae, 316–317, 321
Trichostrongylus cramae caudal bursa, 318
Trichostrongylus pergracilis, 316
Trichostrongylus tenuis, 316, 319
caudal bursa, 319
in Red Grouse, 320
Trichostrongylus tenuis caudal bursa, 318
trichurids, 464
Trichuris vulpis, 491
Tricolored Heron (*see Egretta tricolor*)
Tridentocapillaria, 463
Tridentocapillaria eurycerca, 478
Tridentocapillaria hirundinis, 478, 481
Tridentocapillaria parusi, 482, 483
Trigrisoma lineatum (Rufescent Tiger-Heron), 295
Trimenoponidae, 516
trimethoprim, 95
trimethoprim-sulfadiazine, 117
trimethoprim-sulfamethoxazole, 117, 192
Tringa, 465
Tringa glareola (Wood Sand piper), 361
Tringa melanoleuca (Greater Yellowlegs), 238
Tringa nebularia (Common Greenshank), 331
Tringa semipalmata inornata (Willet), 476
Tringa totanus (Redshank), 6
Tringa totanus (Common Redshank), 332
Trinoton anserinum, 521
Trinoton anserium, 520
Tritrichomonas foetus
in cattle, 134
Trochilidae (Hummingbirds), 59, 443
Troglodytes aedon (House Wren), 553
Troglodytidae (Wrens), 61, 444
Troglotrematidae, 231, 238
Trogonidae (Trogons and Quetzals), 59, 443
Trogoniformes, 14, 59, 163, 443
trombidiosis, 531
Tropical fowl mite (*Ornithonyssue bursa*), 534
Tropocyclops prasinus, 385
Trumpeter Swan (*see Cygnus buccinator*)
Trypanosoma, 27
tsutsugamushi disease (scrub typhus), 534
Tubifex tubifex, 301
tubules, of *Eustrongylides* spp., 305
Tufted Duck (*see Aythya fuligula*)
Tui Parakeet (*see Brotogeris sanctithomae*)
Tundra Swan (*see Cygnus columbianus*)
Turdidae (Thrushes), 61, 435
Turdus, 465
Turdus grayi (clay-colored Robin), 208
Turdus grayi (Clay-colored Robin), 333
Turdus iliacus (Redwing), 345, 484
Turdus merula (Eurasian Blackbird), 206, 345, 451, 484
Turdus migratorius (American Robin), 208, 238, 280, 333, 345, 484, 532–533, 550
Turdus philomelos (Song Thrush), 207, 345, 485
Turdus pilaris (Fieldfare), 485
Turdus ruficollis (Dark-throated Thrush), 485
Turdus viscivorus (Mistle Thrush), 207, 485, 401
turkey, domestic (*see Meleagris gallopavo*)
Turkey Vulture (*see Cathortes aura*)
Turnicidae (Buttanquail), 442
Turquoise Parrot (*see Neophema pulchella*)
Turquoise Tanager (*see Tangara mexicana*)
Turtur spp.
Turtur abyssinicus (Black-billed Wood-Dove), 73
Turtur afer (Blue-spotted Wood-Dove), 73
Turtur chalcospilos (Emerald-spotted Wood-Dove), 73
Turtur tympanistria (Tambourine Dove), 73
Tylotropidus patagiatus, 378
Tympanuchus spp.
Tympanuchus cupido attwateri (Attwater's Prairie Chicken), 321, 322, 543
Tympanuchus cupido (Greater Prairie-Chicken), 155, 391, 396
Tympanuchus pallidicinctus (Lesser Prairie-Chicken), 392
Tympanuchus phasianellus (Sharp-tailed Grouse), 331, 393, 397, 406
Typhlocoelum, 229
Typhlocoelum cucumerium, 235
Tyrannidae (Tyrant Flycatchers), 435, 444
Tyto alba (Barn Owl), 114, 132, 208, 269, 477
Tyto novaehollandiae (Australian Masked-Owl), 477
Tytonidae (Barn Owls), 59, 443
Tyzzeria, 163

UK moors, 317
Undulated Tinamou (*see Crypturellus undulates*)
Upland Goose (*see Chloephaga picta*)
Upupidae (Hoopoes), 60, 443
Ural Owl (*see Strix uralensis*)
Uria, 465
Uria aalge (Common Murre), 291, 332
Urotocus, 230
Uvulifer, 229
Uvulifer ambloplitis, 226

vaccine
  for coccidiosis, 172
  protection, against leucocytozoonosis, 94
  protection, against parasitism, 7
  protection, against *Eimeria*, 172
  protection, against malaria, 48
Vanellus, 465
Vanellus vanellus (Northern Lapwing), 331, 361, 365, 476
Vangidae (Vangas), 61, 444
Variegated Tinamou (*see Crypturellus variegates*)
Varied Thrush (*see Ixoreus naevius*)
vector–parasite associations, 20
vector proofing, of aviaries, 544
vectors
  of avian filarioids, 451
  distribution, 14–15
  of *Haemoproteus*, 18
  of *Leucocytozoon*, 85
  of Plasmodium, 41
Venezillo evergladensis, 327
ventral suckers, 226
ventricular nematodiasis, 355
verminous encephalitis, 424
Verminous peritonitis, 289
Vermivora peregrina (Tennessee Warbler), 333
Vermivora ruficapilla (Nashville Warbler), 116
Vestiaria coccinea (Iiwi), 45, 111
Victoria Crowned-Pigeon (*see* Goura Victoria), 217
Viduidae (Indigobirds), 60
Vinaceous Parrot (*see Amazona vinacea*)
Violaceous Euphonia (*see Euphonia violacea*)
Vireo griseus (White-eyed Vireo), 485
Vireonidae (Vireos), 61, 435, 444
Virginia opossums (*Didelphis virginianus*), 115
visceral larva migrans (VLM), 424, 425
vitamin B12 supplements, 117
Viviparus georgianus, 240
Volatinia jacarina (Blue-black Grassquit), 114
Vulturine Guineafowl (*see Acryllium vulturinum*)

Wahlberg's Eagle (*see Aquila wahlbergi*)
Wardianum, 229
waterfowl, 316, 339, 355
  infected with cestodes, 270
  infected with gastrointestinal trematodes, 228
  infected with *Philopthalmus gralli*, 234
  infected with species of *Corynosoma* and *Polymorphus*, 279
  infected with various hymenolepidid species, 267–268
  infection with *Echinuria uncinata*, 336
Wattled Starling (*see Creatophora cinerea*)
weak ammonia, 509
weak gastric juice, 509
Wedge-tailed Pigeon (*see Treron sphenurus*)
Western Bluebird (*see Sialia mexicana*)
Western Crowned-Pigeon (*see Goura cristala*)
Western Grebe (*see Aechmophorus occidentalis*)
Western Marsh-Harrier (*see Circus aeruginosus*)
Western Meadowlark (*see Sturnella neglecta*)
West Indian Whistling-Duck (*see Dendrocygna arborea*)
Whimbrel (*see Numenius phaeopus*)
Whip snake (*Masticophis flagellum*), 302
Whiskered Auklet (*see Aethia pygmaea*)
Whiskered Tern (*see Chilidonias hybrida*)
White-backed Vulture (*see Gyps africanus*)
White-bellied Pigeon (*see Treson sieboidii*)
White-Breasted Waterhen (*see Amaurornis phoenicurus*)
White-browed Babbler (*see Pomatostomus superciliosus*)
White-copped Redstart (*see Chaimarrornis leucocephalus*)
White-crowned Pigeon (*see Patagioenas leucocephala*)
White-eared Dove (*see Phapitreron leucotis*)
White-eared Parakeet (*see Pyrrhura leucotis*)
White-eyed Parakeet (*see Aratinga leucophthalma*)
White-eyed Vireo (*see Vireo griseus*)
White-faced Whistling-Duck (*see Dendrocygna viduala*)
White-headed Duck (*see Oxyura leucocephala*)
White Ibis (*see Eudocimus albus*)
White-lined Tanager (*see Tachyphonus rufus*)
White-naped Crane (*see Grus vipio*), 182, 184
white pulp arteriolar endothelium, hyperplasia, of, 21
White Stork (*see Ciconia ciconia*)
White-tailed Eagle (*see Haliaeetus albicilla*)
White-tailed Ptarmigan (*see Lagopus leucura*)
White-throated Sparrow (*see Zonotrichia albicollis*)
White-tipped Dove (*see Leptotila verreauxi*)
White Wagtail (*see Motacilla alba*)
White-winged Dove (*see Zenaida asiatica*)
White-winged Duck (*see Cairina scutulata*)
White-winged Scoter (*see Melanitta fusca*)
Whooper Swan (*see Cygnus cygnus*)
Whooping Crane (*see Grus americana*)
Wild Turkey (*see Meleagris gallopavo*)
Willet (*see Tringa semipalmata inornata*)
Willow Ptarmigan (*see Lagopus lagopus*)
Wilsonia citrina (Hooded Warbler), 485
Wohlfahrtia spp
  Wohlfahrtia magnifica, 550, 552
  Wohlfahrtia opaca, 550
  Wohlfahrtia spp., 553
  Wohlfahrtia vigil, 550
Wonga Pigeon (*see Leucosarcia melanoleuca*)
Wood Duck (*see Aix sponsa*)
Wood Sandpiper (*see Tringa glareola*)
Wood Stork (*see Mycteria americana*)
Wood Thrush (*see Hylocichla mustelina*)
Wormersia midwayensis, 527

Xenicidae, 444
Xenopus laevis (African clawed frog), 303
Xenus cinereus (Terek Sandpiper), 476

Yellow-bellied Sapsucker (*see Sphyrapicus varius*)
Yellow-billed Chough (*see Pyrrhocorax graculus*)
Yellow-billed Duck (*see Anas undulata*)
Yellow-billed Pintail (*see Anas georgica*)
Yellow Bittern (*see Ixobrychus sinensis*)
Yellow Bunting (*see Embesiza sulphurata*)
Yellow-collared Lovebird (*see Agapornis personatus*)
Yellow-crested Cockatoo (*see Cacatua sulphurea*)
Yellow-crowned Night-Heron (*see Nyctanassa violacea*)
Yellow-crowned Parrot (*see Amazona ochrocephala*)
Yellow-footed Pigeon (*see Treron phoenicopterus*)

Yellow-fronted Parakeet (*see Cyanoramphus auriceps*)
Yellow-fronted woodpecker (*see Melanerpes flavifrons*)
Yellowhammer (*see Emberiza citronella*)
yellowish caseous lesions, 137
Yellow-legged Tinamou (*see Crypturellus noctivagus*)
Yellow perch (*Perca flavescens*), 298
Yellow-throated Warbler (*see Dendroica dominica*)

Zebra Dove (*see Geopelia striata*)
Zebra Finch (*see Taeniopygia guttata*)
Zeinaida spp. (Doves), 425, 465
Zenaida asiatica (White-winged Dove), 71, 128, 398, 404
Zenaida auriculata (Eared Dove), 394
Zenaida galapagoensis (Galapagos Dove), 128
Zenaida macroura (Mourning Dove), 17, 22, 23, 71, 126, 136, 144, 332, 398
zinc sulfate solution, 490
zoalene, 172, 177
Zonotrichia albicollis (White-throated Sparrow), 20, 22, 23, 234
Zosteropidae (White-eyes), 60, 444
Zosterops japonicus (Japanese White-eye), 111
Zygocotyle, 231
Zygocotyle lunata, 236