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Parasites and Parisitic Diseases (Field Manual of Wildlife Diseases)

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Section 5
Parasites and Parasitic Diseases

Hemosporidiosis
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Stained blood smear from a turkey infected with the parasite *Haemoproteus meleagris.*

Photo by Carter Atkinson
Introduction to Parasitic Diseases

“Parasites form a large proportion of the diversity of life on earth.”

(Price)

Parasitism is an intimate relationship between two different species in which one (parasite) uses the other (host) as its environment from which it derives nourishment. Parasites are a highly diverse group of organisms that have evolved different strategies for infecting their hosts. Some, such as lice and ticks, are found on the external parts of the body (ectoparasites), but most are found internally (endoparasites). Some are microscopic, such as the blood protozoans that cause avian malaria; however, many are macroscopic. Life cycles differ greatly between major types of parasites and are generally classified as direct or indirect (Table 1). Direct life cycles do not require an intermediate host (Fig. 1A). For direct life cycles, only a definitive host is required: the species in which the parasite reaches sexual maturity and produces progeny. Indirect life cycles may involve one or more intermediate hosts (Fig. 1B and C). Intermediate hosts are required by the parasite for completion of its life cycle because of the morphological and physiological changes that usually take place in the parasite within those hosts. Wild birds can serve as the definitive hosts for most of the parasites that are discussed in the following chapters. In addition, paratenic or transport hosts are present in some parasite life cycles. The parasites generally do not undergo development in paratenic hosts. Instead, paratenic hosts provide both an ecological and temporal (time) bridge for the parasite to move through the environment and infect the definitive host. Typically, in these situations one or more intermediate hosts are required for development of the parasite but they are not fed upon by the bird. Instead, the bird feeds on the paratenic hosts, which in turn have fed on the intermediate host(s), thereby, “transporting” the parasite to the bird (Fig. 2).

The presence of parasites in birds and other animals is the rule, rather than the exception. Hundreds of parasite species have been identified from free-ranging wild birds; however, the presence of parasites does not necessarily equate with disease. Most of the parasites identified from wild birds cause no clinical disease. Others cause varying levels of disease, including death in the most severe cases. The pathogenicity or the ability to cause disease, of different species of parasites varies with 1) the species of host invaded (infected or infested), 2) the number or burden of parasites in or on the host, and 3) internal factors impacting host response. For example, when birds are in poor nutritional condition, have concurrent infections from other disease agents (including other species of parasites), or are subject to other types of stress, some parasites that do not normally cause disease do cause disease. Lethal infections may result from parasites that generally only cause mild disease.

This section highlights some of the parasitic diseases such as trichomoniasis that are associated with major mortality events in free-ranging wild birds and those that because of the gross lesions they cause (Sarcocystis sp.), their visibility (nasal leeches), or general interest (heartworm) are often the subject of questions asked of wildlife disease specialists.

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Quote from:
<table>
<thead>
<tr>
<th>Type of parasite</th>
<th>Common name</th>
<th>Type of life cycle</th>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nematodes</td>
<td>Roundworms</td>
<td>Indirect and direct</td>
<td>Most significant group relative to number of species infecting birds and to severity of infections. Unsegmented cylindrical worms. Found throughout the body. Generally four larval stages. Sexes are separate. Most are large in size (macroscopic).</td>
</tr>
<tr>
<td>Cestodes</td>
<td>Tapeworms</td>
<td>Indirect</td>
<td>Flattened, usually segmented worms with a distinct head, neck and body. Found primarily in the lumen of the intestines. Lack a mouth or an alimentary canal; feed by absorbing nutrients from the host's intestinal tract. Most are hermaphroditic (self-fertilization; have both male and female reproductive tissues). Attachment is by suckers, hooks. Large size (macroscopic).</td>
</tr>
<tr>
<td>Trematodes</td>
<td>Flukes</td>
<td>Indirect</td>
<td>Flatworms, generally leaf-shaped (some almost cylindrical). Generally found in the lower alimentary tract, respiratory tract, liver, and kidneys. Complex life cycles; usually require two intermediate hosts, one of which is usually a snail. Hermaphroditic except for blood flukes, which have separate sexes. Attachment is usually by suckers.</td>
</tr>
<tr>
<td>Acanthocephalans</td>
<td>Thorny-headed worms</td>
<td>Indirect</td>
<td>Cylindrical, unsegmented worms. Found in the digestive tract. No intestinal tract; nutrients absorbed through the tegument (similar to tapeworms). Sexes are separate. Attachment by means of a retractable proboscis that has sharp recurved hooks or spines.</td>
</tr>
<tr>
<td>Protozoans</td>
<td>Coccidians, malarias, trichomonads, others</td>
<td>Direct and indirect</td>
<td>Microscopic. Different types are found in different parts of the body. Asexual and sexual multiplication.</td>
</tr>
</tbody>
</table>
Figure 1  Examples of (A) direct, (B) simple indirect, and (C) complex indirect parasite life cycles.
Parasite eggs are passed in feces and hatch in water

Larvae (miracidium) swims to a snail and penetrates it, undergoing further larval development within the host

New larval stage emerges from snail (cercaria) and swims to new host where it penetrates and encysts

Other bird species eat second intermediate host and become infected

First intermediate host
Second intermediate host

Infected bird
Figure 2  Hypothetical parasite life cycle illustrating the role of paratenic (transport) hosts.
Chapter 24

Hemosporidiosis

Synonyms
Avian malaria

Cause
Hemosporidia are microscopic, intracellular parasitic protozoans found within the blood cells and tissues of their avian hosts. Three closely related genera, Plasmodium, Haemoproteus, and Leucocytozoon, are commonly found in wild birds. Infections in highly susceptible species and age classes may result in death.

Life Cycle
Hemosporidia are transmitted from infected to uninfected birds by a variety of biting flies that serve as vectors, including mosquitoes, black flies, ceratopogonid flies (biting midges or sandflies) and louse flies (Fig. 24.1) (Table 24.1). When present, infective stages of the parasites (sporozoites) are found in the salivary glands of these biting flies. They gain entry to the tissues and blood of a new host at the site of the insect bite when these vectors either probe or lacerate the skin to take a blood meal. Insect vectors frequently feed

![Diagram of the life cycle of Hemosporidiosis](image)

Figure 24.1 The complex general life cycle of hemosporidian parasites begins with (A), an infected insect biting a susceptible bird. Separate infectious and developmental stages occur in (B), the bird host, and (C), the insect vectors.
on exposed flesh around the eyes (Fig. 24.2), the beak, and on the legs and feet, although black flies, ceratopogonid flies, and louse flies can crawl beneath the bird’s feathers to reach the skin surface. Immediately after they infect a bird, sporozoites invade the tissues and reproduce for one or more generations before they become merozoites. Merozoites penetrate the red blood cells and become mature, infectious gametocytes. The cycle is completed when the gametocytes in the circulating blood cells of the host bird are ingested by another blood-sucking insect, where they undergo both sexual and asexual reproduction to produce large numbers of sporozoites. These invade the salivary glands of the vector and are transmitted to a new host bird during the vector’s next blood meal.

**Species Affected**

The avian hemosporidia are cosmopolitan parasites of birds, and they have been found in 68 percent of the more than 3,800 species of birds that have been examined. Members of some avian families appear to be more susceptible than others. For example, ducks, geese and swans are commonly infected with species of *Haemoproteus*, *Leucocytozoon*, and *Plasmodium*, and more than 75 percent of waterfowl species that were examined were hosts for one or more of these parasites. Wild turkeys in the eastern United States are also commonly infected by these parasites. Pigeons and doves have similar high rates of infection, but members of other families, such as migratory shorebirds, are less frequently parasitized.

Differences in the prevalence, geographic distribution, and host range of hemosporidia are associated with habitat preferences of the bird hosts, the abundance and feeding habits within those habitats of suitable insect vectors, and innate physiological differences that make some avian hosts more susceptible than others. For example, some species of black flies (*Simulium* sp.) prefer to feed on waterfowl within a lim-
lected distance of the shoreline. Ducks and geese that spend more of their time in this zone will be more likely to be exposed to bites that carry infective stages of *Leucocytozoon simondi*. Biting midges or no-see-ums (*Culicoides* sp.) that transmit species of *Haemoproteus* are more active at dusk in the forest canopy. Birds that roost here, for example, increase their chances for being infected with this parasite. Finally, some avian hosts are more susceptible to hemosporidian parasites than others, but the physiological basis for this is still poorly understood.

Species of *Plasmodium* and *Leucocytozoon* are capable of causing severe anemia, weight loss, and death in susceptible birds. Young birds are more susceptible than adults, and the most serious mortality generally occurs within the first few weeks of hatching. This is also the time of year when increasing temperatures favor the growth of the populations of insect vectors that transmit hemosporidia. Major outbreaks of *L. simondi* that caused high mortality in ducks and geese in Michigan and subarctic Canada have been documented. Species of *Haemoproteus* are generally believed to be less pathogenic, with only scattered reports of natural mortality in wild birds.

Penguins and native Hawaiian forest birds are highly susceptible to *Plasmodium relictum*, a common parasite of songbirds that is transmitted by *Culex* mosquitoes. This parasite causes high mortality in both captive and wild populations of these hosts, and it is a major factor in the decline of native forest birds in the Hawaiian Islands.

**Distribution**

Species of *Plasmodium*, *Haemoproteus*, and *Leucocytozoon* have been reported from most parts of the world with the exception of Antarctica, where cold temperatures prevent the occurrence of suitable insect vectors. Studies of the distribution of hemosporidia in North America have shown that areas of active transmission of the parasites coincide with the geographic distribution of their vectors. *Leucocytozoon* is most common in mountainous areas of Alaska and the Pacific Northwest where abundant fast-moving streams create suitable habitat for aquatic black fly larvae. Species of *Haemoproteus* and *Plasmodium* are more evenly distributed across the continent because their ceratopogonid and mosquito vectors are less dependent on the presence of flowing water for larval development. Migratory birds may winter in habitats that lack suitable vectors; therefore, the simple presence of infected birds may not be evidence that the parasites are being transmitted to birds at the wintering grounds.

**Seasonality**

Infections with *Plasmodium*, *Haemoproteus*, and *Leucocytozoon* are seasonal because transmission depends upon the availability of vector populations. In temperate North America, most birds become infected with hemosporidia during the spring when conditions for transmission become optimal. Some of these conditions include the onset of warmer weather; increases in vector populations; the reappearance or relapse of chronic, low-level infections in adult birds; and the hatching and fledging of susceptible, nonimmune juvenile birds. In warmer parts of the United States, these parasites may be transmitted at other times of the year. In Hawaii, *P. relictum* in forest bird populations may be transmitted throughout the year in warm low-elevation forests, but transmission is more seasonal at elevations above 3,000 ft. where cooler winter temperatures limit mosquito populations.

**Field Signs**

Birds with acute infections of *Plasmodium*, *Haemoproteus*, and *Leucocytozoon*, may exhibit similar signs in the field. These include emaciation, loss of appetite, listlessness, difficulty in breathing, and weakness and lameness in one or both legs. Survivors develop persistent, low-level infections in the blood and tissues that stimulate immunity to reinfection. These survivors do not exhibit any signs of disease, but they serve as reservoirs of infection, allowing the parasites to survive droughts and cold winter weather when vector populations have died off.

**Gross Lesions**

Gross lesions associated with acute infections include enlargement of the liver and spleen (Fig. 24.3) and the appearance of thin and watery blood as a result of infected blood cells being destroyed and removed from circulation (Fig. 24.4). In *Plasmodium* and *Haemoproteus* infections, parasites within the red blood cells produce an insoluble black pigment called hemozoin when they digest the host’s oxygen-bearing, iron-laden red blood cell protein or hemoglobin. The hemozoin is deposited extensively in the host’s spleen and liver tissue as the host’s immune system responds to the infection. In very heavy infections, the kidneys may also be affected. These organs typically appear chocolate brown or black at necropsy and they may be two or more times their normal size (Fig. 24.3). Hemozoin pigment is not produced in *Leucocytozoon* infections; therefore, organs will not be as discolored and dark at necropsy, but they will still appear enlarged. Some species of *Haemoproteus* form large, cyst-like bodies in muscle tissue that superficially resemble tissue cysts produced by species of *Sarcocystis* (Fig. 24.5).

**Diagnosis**

Definitive diagnosis of hemosporidian infections is dependent on microscopic examination of a stained blood smear or on an organ impression smear to detect the presence and form of the parasites within the red blood cells (Figs. 24.6, 7, 8). Species of *Leucocytozoon* frequently produce dramatic changes in the host’s cell structure (Fig. 24.6). Parasitized red blood cells are often enlarged and elongated so that they
Figure 24.3  Gross lesions caused by *Plasmodium relictum* in an apapane. Enlargement and discoloration of the (A), liver and (B), spleen are typical in acute infections when large numbers of parasites are found in the circulating red blood cells.
Figure 24.4  Thin and watery blood from an apapane infected with *Plasmodium relictum* before (left) and after (right) centrifugation. In uninfected songbirds, approximately half of the blood volume is occupied by red blood cells. Note that most of the blood cells have been destroyed by the parasite (right).

Figure 24.5  Pectoral muscles of a turkey infected with *Haemoproteus meleagridis*. Note the white streaks and bloody spots in the muscle tissue of this bird (arrows). The tissue stages of this hemosporidian form large, cystlike bodies that may superficially resemble those caused by species of *Sarcocystis*. 
Figure 24.8  Stained blood smear from an apapane infected with *Plasmodium relictum*. Some red blood cells contain multinucleated, asexually-reproducing stages of the parasite called schizonts (S). These are diagnostic for *Plasmodium* infections and contain one or more centrally-located pigment granules (arrows).

Figure 24.6  Stained blood smear from a turkey infected with *Leucocytozoon smithi*. This parasite causes enlargement and distortion of the infected blood cell. The red blood cell nucleus (N) is divided in two halves that lay on either side of the parasite (P). The membrane of the infected cell is stretched into two hornlike points (arrows).

Figure 24.7  Stained blood smear from a turkey infected with *Haemoproteus meleagridis*. Gametocytes (G) contain a single pink-staining nucleus and contain black or golden brown pigment granules (arrows).

Photo by Carter Atkinson, BRD-PIERC

Photo by Carter Atkinson, BRD-PIERC

Photo by Carter Atkinson, BRD-PIERC
form a pair of horn-like extensions from either end of the cell. Species of *Plasmodium* and *Haemoproteus* produce fewer changes in their host’s red blood cells, but these parasites may cause slight enlargement of infected host cells and displacement of the red blood cell nucleus to one side (Figs. 24.7, 8). Unlike *Leucocytozoon*, *Plasmodium* and *Haemoproteus* produce golden brown or black deposits of hemozoin pigment in the parasite cell (Figs. 24.7, 8). Further differentiation of *Plasmodium* from *Haemoproteus* may be difficult. Diagnosis of a *Plasmodium* infection is dependent on detecting the presence of asexually reproducing stages of its life cycle (schizonts) in the red blood cells of the infected host (Fig. 24.8).

**Control**

Control of the avian hemosporidia is dependent on reducing transmission from infected birds to healthy birds through reduction or elimination of vector populations. Many of the same techniques that were developed for control of vector-transmitted human diseases can be used effectively, but few agencies have the resources or manpower to apply them over large areas. Most techniques rely on habitat management to reduce vector breeding sites or depend on the application of pesticides that affect larval or adult vectors to reduce vector populations. Large-scale treatment of infected survivor birds could prevent disease outbreaks by reducing sources of infection, but the logistics and practicality of treating sufficient numbers of birds to interrupt transmission are prohibitive. Although some experimental vaccines for these parasites have been developed, none are currently available for general use.

**Human Health Considerations**

The avian hemosporidia are closely related to the malarial parasites of humans, but are not capable of infecting people.

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Kilauea Field Station

**Supplementary Reading**


Trichomoniasis

Synonyms
Canker (doves and pigeons), frounce (raptors), avian trichomoniasis

Cause
Avian trichomoniasis is caused by a single celled protozoan, *Trichomonas gallinae*. Avirulent *T. gallinae* strains that do not cause disease and highly virulent strains are found in nature and circulate within bird populations. The factors that make a strain virulent are not known, but they are thought to be controlled genetically within the parasite. Similarly, the reasons why an avirulent or a virulent form of the parasite is found within a bird population at any period of time also remain unknown. Virulent strains of *T. gallinae* have caused major mortality events or epizootics in doves and pigeons in addition to less visible, chronic losses (Table 25.1). Infection typically involves the upper digestive tract of doves and pigeons but other species have also been infected (Fig. 25.1).

Trichomoniasis in doves and pigeons, but not in other species, is generally confined to young birds. The parasite was introduced to the U.S. with the introduction of pigeons and doves brought by European settlers. It has been reported that 80 to 90 percent of adult pigeons are infected, but they show no clinical signs of disease. It is speculated that most of these birds became immune as a result of exposure to avirulent strains of the parasite or because they survived mild infections. In pigeons and mourning doves, the parasites are transmitted from the adults to the squabs in the pigeon milk produced in the crop of the adult. Squabs usually become infected with the first feeding of pigeon milk, which is gen-

<table>
<thead>
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<th>Year</th>
<th>Magnitude</th>
<th>Geographic area</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>1949–51</td>
<td>Tens of thousands of mourning doves</td>
<td>Southeastern United States</td>
<td>Trichomoniasis broke out in virtually all States in the region; the magnitude of losses focused attention on the devastation that could be caused by this disease and stimulated research on the ecology of this disease.</td>
</tr>
<tr>
<td>1950–51</td>
<td>25,000 to 50,000 mourning doves each year</td>
<td>Alabama</td>
<td>Breeding birds were the focus of infection; mortality was thought to have been grossly underestimated.</td>
</tr>
<tr>
<td>1972</td>
<td>Several hundred</td>
<td>Nebraska</td>
<td>Railroad yards and a grain elevator were focal points of infection; birds fed on spilled grain.</td>
</tr>
<tr>
<td>1985</td>
<td>Approximately 800 mourning doves</td>
<td>New Mexico</td>
<td>Losses at birdfeeders near Las Cruces.</td>
</tr>
<tr>
<td>1988</td>
<td>At least 16,000 band-tailed pigeons</td>
<td>California</td>
<td>First major epizootic of trichomoniasis in this species.</td>
</tr>
<tr>
<td>1991</td>
<td>Approximately 500 mourning doves</td>
<td>North Carolina</td>
<td>—</td>
</tr>
</tbody>
</table>
erally within minutes after hatching. The resulting infection may range from asymptomatic or mild disease to a rapidly fatal course resulting in death within 4–18 days after infection. Other modes for infection are through feed, perhaps contaminated drinking water, and feeding on infected birds (Fig. 25.2).

There is no cyst or resistant stage in the parasite’s life cycle; therefore, infection must be passed directly from one bird to another, in contaminated feed or water. Feed and water are contaminated when trichomonads move from the mouth of infected birds, not from their feces. Lesions in the mouth or the esophagus or both of an infected bird (see below) often prevent the passage of ingested grain seeds and cause the bird to regurgitate contaminated food items. Water becomes contaminated by contact with the contaminated bill and mouth. Pigeons that feed among domestic poultry are often blamed for contaminating feed and water and passing the disease to the poultry. Similar transmission has been associated with dove mortality at grain elevators and at birdfeeders. Doves and pigeons cross-feed and bill during courtship, and this behavior facilitates direct transmission as does the consumption of infected birds by raptors. It has been reported that some moist grains can maintain viable *T. gallinae* for at least 5 days and that parasite survival in water can range from 20 minutes to several hours. These conditions are adequate for disease transmission at birdfeeders and waterers because of the gregarious habits of doves and pigeons.

**Species Affected**

Trichomoniasis is considered by many avian disease specialists to be the most important disease of mourning doves in North America. Band-tailed pigeons have also suffered large-scale losses from trichomoniasis. This disease has been reported as a cause of mortality in birds of prey for hundreds of years prior to the causative organism being identified. Songbirds are less commonly reported to be infected, but *T. gallinae* is reported to be the most important trichomonad of caged birds; it is often responsible for epizootics among captive collections. Domestic turkeys and chickens also become infected.

**Distribution**

It is likely that *T. gallinae* is found wherever domestic pigeons and mourning doves are found. Disease in free-ranging wild birds is grossly underreported. Outbreaks at birdfeeding stations and similar locations reported to the National Wildlife Health Center have occurred from coast-to-coast within the United States (Fig. 25.3).

**Seasonality**

Epizootics due to *T. gallinae* can happen yearround, but most outbreaks have been reported during late spring, summer, and fall.

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**Figure 25.1** Relative frequency of trichomoniasis in free-ranging birds.

**Field Signs**

Because oral lesions often affect the ability of the bird to feed, infected birds lose weight, appear listless, and stand grouped together. These birds often appear ruffled. Caseous or cheesy, yellowish lesions may be seen around the beak or eyes of mourning doves and the face may appear “puffy” and distended (Fig. 25.4). Severely infected pigeons may fall over when they are forced to move.

**Gross Lesions**

The severity and appearance of lesions varies with the virulence of the strain of the parasite, the stage of infection, and the age of the bird. The most visible lesions from mildly pathogenic strains may simply appear as excess salivation and inflammation of the mucosa or lining of the mouth and throat. Early oral lesions appear as small, well defined, cream to yellowish spots on the mucosal surface (Fig. 25.5A). As the disease progresses the lesions become larger, thicker, and
Figure 25.2 Transmission of trichomoniasis.
EXPLANATION
Trichomoniasis outbreak sites, 1983–97*

* Outbreaks of trichomoniasis involve doves and pigeons with mortality ranging from tens to hundreds.

Figure 25.3 Locations of outbreaks of trichomoniasis in free-ranging birds, January 1983 through March 1997.

Figure 25.4 Mourning doves at a backyard waterbath. Note the puffy appearance (arrow) of the face of a $T. \text{gallinae}$ infected dove.
are caseous (consistency of cheese) in appearance (Fig. 25.5B). In more advanced lesions, a wet, sticky type of discharge and nodules within the mouth are characteristic of acute disease. Hard, cheesy lesions are most often seen in more chronic infections. Although lesions are generally confined to the inside of the mouth and esophagus, they can extend externally to the beak and eyes and be confused with avian pox (see Chapter 19).

Early lesions of the pharynx to the crop are also cream to yellow in color and caseous. As the disease progresses, these lesions may spread to the esophagus (Fig. 25.5C), and can eventually block its opening (Fig. 25.5D). A bird can suffocate if the blockage is severe enough. A bird will starve when these masses prevent it from swallowing food and water. These large, caseous masses may invade the roof of the mouth and sinuses (Fig. 25.5E) and even penetrate through the base of the skull into the brain. Also, a large amount of fluid may accumulate in the crop of severely infected birds. Lesions may extend down the alimentary tract and the parasite may invade the liver, particularly in domestic pigeons. Other organs such as the lungs, occasionally become involved. The digestive tract below the proventriculus is rarely involved.

**Diagnosis**

A tentative diagnosis can be made for doves and pigeons on the basis of finding caseous, obstructive lesions within the upper areas of the digestive tract. However, other disease agents such as pox virus, *Aspergillus* sp. fungi, *Candida* sp. yeasts, nematodes of the genus *Capillaria*, and vitamin A deficiency can produce similar lesions. Diagnosis is estab-

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**Figure 25.5** Gross lesions of trichomoniasis in mourning doves. *(A)* Small, cream-colored lesion on oral mucosa (arrow). *(B)* Large, caseous lesions in back of mouth (arrow). *(C)* Large lesion in upper esophagus (arrow). *(D)* Occlusion of esophagus by a large, caseous lesion. *(E)* Lesions on the roof of the mouth, in the region of the sinuses (arrow).
lished by finding the trichomonads in the saliva or smears of the caseous lesions of infected birds. Specimens are best taken from sick birds, or from recently dead birds that are kept chilled and reach the diagnostic laboratory within 48 hours after death. Samples of tissues with lesions preserved in 10 percent buffered formalin or frozen whole carcasses can be used if fresh carcasses cannot be provided.

**Control**

The removal of infected birds is recommended for combating trichomoniasis in poultry and captive pigeons and in captive collections of wild birds. The focus in both instances is on birds that harbor virulent strains of the parasite. Elimination of infection from adult birds by drug treatment has also been recommended, but this is not a practical approach for wild birds. Prevention of the build-up of large concentrations of doves at birdfeeders and artificial watering areas is recommended to minimize disease transmission in the wild. Stock tanks, livestock feedlots, grain storage facilities and clusters of urban birdfeeders should be targeted for disease prevention activities. Although the environmental persistence for *T. gallinae* is rather limited, contaminated feed is suspected as a significant source of disease transmission. Therefore, fresh feed should be placed in feeders daily, if it is practical. Platforms and other surfaces where feed may collect, including the area under feeders, should be frequently decontaminated with 10 percent solution of household bleach in water, preferably just prior to placing clean feed in the feeder. Pigeons and doves are high risk food sources for birds of prey; therefore, before they are fed to raptors, pigeons and doves should be inspected first and found to be free of trichomoniasis or other infectious diseases.

**Human Health Considerations**

None. *T. gallinae* has not been reported to infect humans.

*Rebecca A. Cole*

**Supplementary Reading**


Chapter 26
Intestinal Coccidiosis

Synonyms
Coccidiosis, coccidiasis

Cause
Coccidia are a complex and diverse group of protozoan (single-celled organisms) parasites; the coccidia group contains many species, most of which do not cause clinical disease. In birds, most disease-causing or pathogenic forms of coccidia parasites belong to the genus Eimeria. Coccidia usually invade the intestinal tract, but some invade other organs, such as the liver and kidney (see Chapter 27).

Clinical illness caused by infection with these parasites is referred to as coccidiosis, but their presence without disease is called coccidiasis. In most cases, a bird that is infected by coccidia will develop immunity from disease and it will recover unless it is reinfected. The occurrence of disease depends, in part, upon the number of host cells that are destroyed by the juvenile form of the parasite, and this is moderated by many factors. Severely infected birds may die very quickly. Often, tissue damage to the bird’s intestine results in interrupted feeding; disruption of digestive processes or nutrient absorption; dehydration; anemia; and increased susceptibility to other disease agents. In cranes, coccidia that normally inhabit the intestine sometimes become widely distributed throughout the body. The resulting disease, disseminated visceral coccidiosis (DVC) of cranes, is characterized by nodules, or granulomas, on the surface of organs and tissues that contain developmental stages of the parasite.

Collectively, coccidia are important parasites of domestic animals, but, because each coccidia species has a preference for parasitizing a particular bird species and because of the self-limiting nature of most infections, coccidiosis in free-ranging birds has not been of great concern. However, habitat losses that concentrate bird populations and the increasing numbers of captive-reared birds that are released into the wild enhance the potential for problems with coccidiosis.

Life Cycle
Most intestinal coccidia have a complex but direct life cycle in which the infective forms of the parasite invade a single host animal for development to sexual maturity; the life cycle is completed in 1–2 weeks (Fig. 26.1). A mature female parasite in the intestine of an infected host bird produces noninfective, embryonated eggs or oocysts, which are passed into the environment in the feces of the host bird. The oocysts quickly develop into an infective form while they are in the environment. An uninfected bird ingests the infective oocysts while it is eating or drinking, and the infective oocysts invade the bird’s intestine. Within the intestine, the oocysts may or may not undergo several stages of development, depending on the parasite species, before they become sexually mature male and female parasites. The complex life cycle for Eimeria (Fig. 26.2) illustrates the exponential rate of infection and destruction of the intestinal epithelial cells, which are the cells that provide the covering of the intestinal lining. The mature female parasites release noninfective oocysts to the environment, and, thus, the cycle begins anew.

Species Affected
Many animal species, including a wide variety of birds (Table 26.1) may harbor coccidia. Although disease is not common in free-ranging wild birds, several epizootics due to E. aythyae have been reported among lesser scaup in the United States. During those events, predominantly females have died, which suggests that female lesser scaup may be more susceptible to the disease than male lesser scaup. Lesions of DVC were first seen in captive sandhill cranes in the late 1970s. Since then, mortality of captive sandhill and whooping cranes has been attributed to DVC, and the disease has been found in wild sandhill cranes, including the endangered Mississippi sandhill crane.

Characteristics of Intestinal Coccidiosis
All domestic birds carry more than one species of coccidia, and pure infections with a single species are rare.

Different coccidia species are usually found in a specific location within the intestinal tract of the host bird.

After initial exposure to the parasite, the host bird may quickly develop immunity to it but immunity is not absolute. A bird can be reinfected by the same or a different species of the parasite.

Infections do not generally cause a problem of free-ranging birds; instead, coccidiosis is considered a disease of monoculture and of the raising of birds in confinement.
Distribution

Coccidia are found worldwide. The few reported outbreaks of coccidiosis in free-ranging waterfowl have all occurred in the Midwestern United States (Fig. 26.3). Recurrent epizootics have broken out at a single reservoir in eastern Nebraska, and coccidiosis is also believed to be the cause of waterfowl die-offs in Wisconsin, North Dakota, Illinois, and Iowa. DVC has been found in migratory sandhill cranes at several locations, and it is a recurring problem in the only free-ranging population of the nonmigratory Mississippi sandhill crane. These birds reside at the Mississippi Sandhill Crane National Wildlife Refuge in Mississippi.

Seasonality

Birds may be infected with coccidia at any time. Although little is known about the conditions that may lead to the development of clinical disease in wild birds, birds may become diseased more frequently during periods of stress. Most epizootics of intestinal coccidiosis in waterfowl in the Upper Midwest have broken out in early spring, during a stressful staging period of spring migration. Mississippi sandhill cranes also die from DVC most frequently during the spring.

Field Signs

Field signs for free-ranging wild birds have not been reported. Nonspecific clinical signs reported for captive birds include inactivity, anaemia, weight loss, general unthrifty appearance, and a watery diarrhea that may be greenish or bloody. Tremors, convulsions, and lameness are also occasionally seen. Rapid weight loss may lead to emaciation and dehydration followed by death. Young birds that survive severe infections may suffer retardation of growth.

Figure 26.1 Direct life cycle of Eimeria infection in birds.
A. Noninfective parasite oocysts (eggs) containing a single cell referred to as the sporont are passed via feces into the environment.

B. Oocysts become infective after 2 days in the environment at ordinary temperatures through sporulation (sporogony), which is a developmental process that results in the sporont dividing and forming four sporocysts each containing two infective sporozoites.

C. Infective oocysts are ingested by birds in contaminated feed, water, soil, or other ingesta.

D. The oocyst wall breaks within the gizzard of the bird and releases the sporocysts.

E. The sporozoites escape from the sporocysts in the small intestine and enter the epithelial cells, which are cells that line the internal and external surfaces of the body of the intestine.

F. The sporozoites develop within the epithelial cells, and asexual multiple fission results in the formation of first-generation meronts, each of which produces about 900 first-generation merozoites.

G. Merozoites break out of the epithelial cells into the intestinal canal about 2.5–3 days after infection. The merozoites enter new host cells and undergo developmental processes resulting in the formation of second-generation meronts. By dividing many times, each of these meronts produce about 200–350 second-generation merozoites that are 4–8 times larger in size than the first-generation merozoites and that are produced about 5 days after initial oocyst ingestion.

H. The cycle may continue with a third generation of a small number (4–30) of merozoites of intermediate size (between those of the first and second generation). However, many of the second-generation merozoites enter new host cells and begin the sexual phase of the life cycle referred to as gamogony.

I. Most of the second-generation merozoites develop into female gametes or macrogamonts and some become males or microgamonts. The females grow until they reach full size while a large number of tiny microgametes are formed within each of the microgamonts. The macrogamonts are fertilized by the microgametes and new oocysts result.

J. Seven days after ingestion of infected coccidia, the oocysts break out of their host cells and enter the intestinal canal to be passed from the body via feces to continue the cycle.

Figure 26.2 A typical life cycle of Eimeria sp. in birds. (Adapted from Eimeria tenella in chickens.)
Table 26.1  Relative occurrence of coccidia in different groups of birds. [Frequency of occurrence:  • occasional,  ● common, — not reported]

<table>
<thead>
<tr>
<th>Bird types (and examples)</th>
<th>Eimeria sp.</th>
<th>Isospora sp.</th>
<th>Tyzzeria sp.</th>
<th>Cryptosporidium sp.</th>
<th>Wenyonella sp.</th>
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</thead>
<tbody>
<tr>
<td>Poultry (Chicken, turkey)</td>
<td>•</td>
<td>—</td>
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<td>Anseriformes (Ducks, geese)</td>
<td>•</td>
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<td>●</td>
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<td>Charadriiformes (Gulls, shorebirds)</td>
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<td>Columbiformes (Pigeons, doves)</td>
<td>•</td>
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<td>●</td>
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<tr>
<td>Coraciiformes (Kingfishers)</td>
<td>—</td>
<td>●</td>
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<tr>
<td>Falconiformes (Hawks, falcons)</td>
<td>—</td>
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<tr>
<td>Galliformes (Pheasant, quail)</td>
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<td>●</td>
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<td>●</td>
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<td>Gruiformes (Cranes, rails)</td>
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<td>Passeriformes (Songbirds)</td>
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<tr>
<td>Pelicaniformes (Pelicans)</td>
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<td>Piciformes (Woodpeckers)</td>
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<td>—</td>
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<td>Psittaciformes (Parrots)</td>
<td>●</td>
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<td>—</td>
<td>●</td>
<td>—</td>
</tr>
<tr>
<td>Strigiformes (Owls)</td>
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<td>●</td>
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<td>—</td>
</tr>
<tr>
<td>Struthioniformes (Ostriches)</td>
<td>—</td>
<td>●</td>
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<td>—</td>
</tr>
</tbody>
</table>

EXPLANATION

Intestinal coccidiosis outbreaks, by State

Figure 26.3  Location of outbreaks of intestinal coccidiosis in waterfowl.
**Gross Lesions**

The location of lesions varies with the species of coccidia and the severity and intensity of infection. In acutely-affected lesser scaup, bloody inflammation or enteritis is commonly seen in the upper small intestine (Fig. 26.4A). In scaup that survive for longer periods, dry crusts form on the mucosal (internal) surface of the intestinal tract. The severity of this lesion decreases from the small intestine to the large intestine (Fig. 26.4B). Chronic lesions of intestinal coccidiosis take other forms in different species, sometimes appearing as rather distinct light-colored areas within the intestinal wall (Fig. 26.5).

Lesions of DVC in cranes typically consist of small (usually less than 5 millimeters in diameter), raised, light-colored granulomas. These nodules may be found on any surface within the body cavity, but they are commonly seen on the lining of the esophagus near the thoracic inlet area and on the inner surface of the sternum (Fig. 26.6A–C). Light-colored patches may also appear on and within organs such as the heart and liver (Fig. 26.7A, B).

**Diagnosis**

When large numbers of oocysts are found in the feces of live birds concurrent with diarrhea, emaciation, and pallor or pale skin color, coccidiosis should be suspected as the cause of illness. However, a diagnosis of coccidiosis as cause of death requires a necropsy evaluation combined with identification of the causative coccidia. Fecal evaluations are not adequate for a diagnosis of coccidiosis because disease may develop before large numbers of oocysts are present in feces and because oocysts seen in the feces may not be those of pathogenic species. As with other diagnostic evaluations, submit chilled, whole carcasses for necropsy by qualified specialists. When carcasses cannot be provided, remove intestinal tracts and submit them chilled. If submissions will be delayed for several days or longer and carcasses cannot be preserved by freezing, remove the entire intestinal tract and preserve it in an adequate volume of neutral formalin (see Chapter 3).

**Control**

Oocysts can rapidly build up in the environment when birds are overcrowded and use an area for a prolonged period of time. The disease risk increases significantly when these conditions result in oocyst contamination of food and drinking water. In captive situations, good husbandry and sanitation, including continual removal of contaminated feed and litter, can minimize the potential for coccidiosis. Captive birds can be treated with therapeutic agents that control, but that do not eliminate, the level of infection. Therefore, oocyst shedding by those birds after they are removed from therapy should be considered if they are to be released or mixed with other birds. Light infections result in a substantial level of immunity to that species of coccidia and are use-
Figure 26.6  Gross lesions of disseminated visceral coccidiosis of cranes. (A) Granulomas on the lining of the esophagus (arrows); and (B) in the area of the thoracic inlet [the tip of the forceps is between granulomas on the surface of a vessel and nerve (left) and on the thyroid gland (right)]; and (C) on the inside surface of the sternum (arrow).
ful in preventing epizootics from this disease. Therefore, the objective is not to completely eliminate infection with coccidia; instead, the focus should be on preventing heavy infections and the establishment and persistence of high levels of environmental contamination with coccidia. For free-ranging birds, flock dispersal may be warranted when overcrowding continues for prolonged periods of time.

**Human Health Considerations**

None. Coccidia of birds are not infectious for humans.

*Milton Friend and J. Christian Franson*

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**Supplementary Reading**


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*Figure 26.7* Lesions of disseminated visceral coccidiosis also may include light patches as seen here on the (A), surfaces of the heart muscle and (B) on the liver (arrows).
Chapter 27

Renal Coccidiosis

Cause

Renal coccidiosis is caused by protozoal parasites that infect the kidneys and associated tissues. Most of the coccidia that infect the tissues in birds are *Eimeria* sp. As with most other parasitic infections, this infection is not synonymous with clinical or apparent disease. Asymptomatic infections are far more common than those that are severe and cause mortality.

Life Cycle

Typical *Eimeria*-type life cycles have an internal or endogenous phase of development within the host. A bird becomes a host when it feeds or drinks from a source that is contaminated with oocysts (cystic, infectious stage) that have become infectious following multiple fission of the sporont (zygote) to form four sporocysts, each containing two infect-

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**Figure 27.1** Life cycle of *Eimeria truncata*, which is one of the parasites that causes renal coccidiosis.
tious sporozoites (sporogony) within each oocyst. The infective sporozoites within the sporocysts of the oocysts invade the bird’s intestinal lining, where they may undergo several developmental stages depending on the *Eimeria* species. *E. truncata*, the most well known of the renal coccidia, matures and reproduces only in the kidneys and in the cloaca near its junction with the ureter (Fig. 27.1). It is not known how the *E. truncata* sporozoites get from the intestine to the kidneys; the sporozoites probably undergo asexual reproduction or multiple fission before they reach the kidneys. The sexual phase of the *E. truncata* life cycle, or gamogony, takes place in the kidneys, producing noninfectious oocysts which are voided with the host bird’s feces into the environment. Sporulated oocysts are resistant to environmental extremes, and their sporozoites can remain infectious for months.

The life cycles of the coccidia that cause renal coccidiosis are similar to those that cause intestinal coccidiosis (see Chapter 26). However, less is known about the species of *Eimeria* that cause renal coccidiosis than about those that cause intestinal coccidiosis.

### Types of birds

<table>
<thead>
<tr>
<th>Types of birds</th>
<th>Species of coccidia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Waterfowl</td>
<td></td>
</tr>
<tr>
<td>Ducks</td>
<td><em>Eimeria boschadis</em>, <em>E. somatarie</em>, <em>E. sp.</em></td>
</tr>
<tr>
<td>Geese</td>
<td><em>E. truncata</em>, <em>E. sp.</em></td>
</tr>
<tr>
<td>Swans</td>
<td><em>E. christianseni</em></td>
</tr>
<tr>
<td>Fish-eating birds</td>
<td></td>
</tr>
<tr>
<td>Gulls</td>
<td><em>E. wobeseri</em>, <em>E. goelandi</em>, <em>E. renicola</em></td>
</tr>
<tr>
<td>Cormorants</td>
<td><em>E. sp.</em></td>
</tr>
<tr>
<td>Loons</td>
<td><em>E. graviae</em></td>
</tr>
<tr>
<td>Marine Birds</td>
<td></td>
</tr>
<tr>
<td>Puffins</td>
<td><em>E. fracterculae</em></td>
</tr>
<tr>
<td>Shearwaters</td>
<td><em>E. sp.</em>, unidentified coccidia</td>
</tr>
<tr>
<td>Land Birds</td>
<td></td>
</tr>
<tr>
<td>Owls</td>
<td>Unidentified coccidia</td>
</tr>
<tr>
<td>Woodcock</td>
<td>Unidentified coccidia (in captive colony)</td>
</tr>
</tbody>
</table>

*Figure 27.2*  Reported occurrences of renal coccidia in wild birds.
Species Affected

Avian coccidiosis was first reported in France. Canadian investigators have reported that virtually all species of wild ducks they examined are susceptible to renal coccidiosis. Different species of renal coccidia are found in different species of birds (Fig. 27.2). Most reports of renal coccidiosis are of asymptomatic birds or birds that show minor physiological or pathological changes due to the parasite. Young birds and those that have been stressed by various conditions are most likely to have clinical cases of renal coccidiosis. Mortality has occurred in free-ranging wild geese, eider ducklings, and double-crested cormorants. Disease in domestic geese is usually acute, lasts only 2–3 days, and can kill large segments of the flock.

Distribution

Renal coccidiosis is found in birds worldwide.

Seasonality

Mortality from renal coccidiosis is most common during periods of the year when birds are densely aggregated on their breeding grounds or wintering areas.

Field Signs

There are no specific field signs that indicate that a bird is infected with renal coccidia. Young birds will often be emaciated and weak, but many other diseases cause similar clinical signs.

Gross Lesions

Infected birds may be emaciated and have a prominent keel. In severe infections, kidneys may become enlarged and pale, containing multiple spots or foci of infection that coalesce into a mottled pattern (Fig. 27.3). Cutting through these white foci may reveal material that has the consistency of chalk due to the build up of uric acid salts (Fig. 27.4).

Figure 27.3  Kidneys from double-crested cormorants. *Top:* normal size and color. *Bottom:* enlarged kidneys with diffuse pale areas from a bird infected with renal coccidia.

Figure 27.4  Cut surfaces from the same two kidneys as in Fig. 27.3. *Bottom* kidney shows chalky material from buildup of uric acid salts.
Diagnosis

Confirmation of renal coccidiosis requires microscopic examination of tissue by the trained staff of a diagnostic laboratory. Whole carcasses are generally needed to determine the cause of death unless kidney damage is so severe that it unquestionably would have caused death. When whole refrigerated carcasses cannot be provided for evaluation because of field circumstances, the kidneys should be removed, preserved in a 10:1 volume of 10 percent buffered neutral formalin and submitted for diagnosis (see Chapter 2).

Control

Control of renal coccidiosis in free-ranging birds is not feasible. Crowded conditions facilitate transmission of the parasite through fecal contamination of the environment. Prevention of degradation of habitat quantity and quality on breeding grounds and wintering areas is needed to minimize disease risks.

Human Health Considerations

There are no reports of human health concerns with this disease.

Rebecca A. Cole

Supplementary Reading


Chapter 28

Sarcocystis

Synonyms
Rice breast disease, sarcosporidiosis, sarcocystosis

Cause
Sarcocystis is a nonfatal, usually asymptomatic infection that is caused by a parasitic protozoan. Various species of this parasite affect mammals, reptiles, and birds. The most commonly reported species of the parasite in North America is Sarcocystis rileyi, the species most commonly found in waterfowl.

Life Cycle
The Sarcocystis sp. parasites have an indirect life cycle (Fig. 28.1) that requires a paratentic or transport host animal (a bird), in which they live for a time before they are transported to a definitive host animal (a carnivore), in which they reach maturity. Birds ingest the eggs or oocysts of the mature parasite in food or water that is contaminated by carnivore feces, which contain the oocysts. The oocysts develop in the intestine of the bird into an intermediate form, the sporozoites, that enter the bird’s bloodstream and infect specific cells of the blood vessels. Multiplication of these cells gives rise to a second intermediate form, merozoites, that are carried by the blood to the voluntary muscles, where elongated cysts or macrocysts are eventually produced (Fig. 28.2). The life cycle is completed when a carnivore ingests the infected muscle tissue of a bird and the parasite reaches maturity and releases oocysts in the intestines of the carnivore. The carnivore is infected only in its intestine. Macrocyts do
not develop in the carnivore, and the *Sarcocystis* sp. parasite rarely causes the carnivore illness or other forms of disease.

**Species Affected**

Dabbling ducks (mallard, northern pintail, northern shoveler, teal, American black duck, gadwall, and American wigeon) commonly have visible or macroscopic forms of *Sarcocystis* sp.; these forms are far less frequently found in other species of ducks and are infrequently found in geese and swans. Recent studies of wading birds in Florida have disclosed a high prevalence of *Sarcocystis* sp.; similar findings have previously been reported from South Africa. Land birds, such as grackles and other passerine birds, as well as mammals and reptiles can have visible forms of sarcocystis, but it is unlikely that *S. rileyi* is the species of parasite involved. With the exception of waterfowl, this parasite has received little study in migratory birds. This must be taken into account when considering the current knowledge of species affected (Fig. 28.3).

**Distribution**

*Sarcocystis* is a common parasitic infection of some waterfowl species, and it is found throughout the geographic range of those species in North America. Less is known about *Sarcocystis* sp. in other species of wild birds, but this parasite has been reported from waterbirds in South Africa, Australia, Canada, and Mexico in addition to the United States.

**Seasonality**

Infected birds can be found yearround, but waterfowl that are infected with *Sarcocystis* sp. are usually observed during the hunting season. Infection is not seen in prefledgling waterfowl, nor is it often seen in juveniles. Two possible reasons for these differences between the age classes may be that the development of visible forms of the parasite requires time or that birds may not be infected until after they have left their breeding grounds. Because visible forms of sarcocystis are more frequently developed in older birds, hunter detection tends to be greatest during years of poor waterfowl production when the bag contains a greater proportion of adult birds. A moderate percentage of juvenile mottled ducks that were collected in Louisiana primarily after the hunting season were recently found to have light sarcocystis infections. Because this species does not migrate, this suggests that the birds were infected within the general geographic area where they were collected and that the later collection date allowed the macrocyst lesions to be visible.

Too little is known about sarcocystis in other groups of wild birds to evaluate its seasonality.

**Field Signs**

*Figure 28.2* Rice-grain sized cysts of *Sarcocystis* sp. evident in parallel streaks in *A*, breast muscle fibers of a mallard and *B*, thigh and leg muscle of an American black duck.

Usually, there is no externally visible sign of this disease nor is it recognized as a direct cause of migratory bird mortality. Severe infections can cause loss of muscle tissue and result in lameness, weakness, and even paralysis in rare cases. The debilitating effects of severe infections could increase bird susceptibility to predation and to other causes of mortality.

**Gross Lesions**

Visible forms of infection are readily apparent when the skin is removed from the bird. In waterfowl and in many other species, infection appears as cream-colored, cylindrical cysts (the macrocysts) that resemble grains of rice running in parallel streaks through the muscle tissue. The cysts are commonly found in the breast muscle (Fig. 28.2A), but they are also found in other skeletal and cardiac muscle (Fig. 28.2B). Calcification of the muscle tissue around these cysts...
Sarcocystis makes them obviously discrete bodies. The degree of calcification is often sufficient to give a gritty feeling to the tissue when it is cut with a knife.

Lesions that were observed in wading birds differed in appearance; the cysts were white and opaque, and they generally extended throughout the entire length of the infected muscle fiber. Cysts were present in the heart muscle and they were confined to striated muscles.

Diagnosis

The visible presence of sarcosporidian cysts in muscle tissue is sufficient to diagnose this disease. Visible cysts may vary in size and shape in different bird species. Good quality color photographs (prints or 35 millimeter slides) of the external surface of infected muscle are generally sufficient for a disease specialist to recognize this disease if tissues or a whole carcass cannot be provided. Whole birds should be submitted if possible. If only tissues can be submitted, then a portion of the infected muscle should be fixed in a 10 percent formalin solution. Frozen muscle tissue is also suitable for diagnosis, and the distinctive appearance of these cysts allows a diagnosis from even partially decomposed carcasses.

Control

There are no known control methods for this disease, nor do any seem to be needed or are any being developed. Control of sarcocystis would require interruption of the life cycle of the parasite. Although the life cycles of the *Sarcocystis* sp. that affect wild birds are not precisely known, they are probably similar to the two-host, indirect life cycle known for some other *Sarcocystis* sp. (Fig. 28.1). The predator-prey relationship between the intermediate bird hosts and the definitive carnivore hosts may be the primary reason that juvenile birds or some bird species are seldom found to be infected. The appropriate carnivores may not be present on the breeding grounds.

Different species of carnivores seem to be involved in the infection of different bird species, which suggests that birds are infected by more than one species of the genus *Sarcocystis* sp. If the carnivore-bird cycle is species-specific, that is, if a specific species of bird can only be infected by oocysts that are produced by a parasite in a specific carnivore species, then selective control of sarcocystis might be feasible. However, current knowledge of the disease does not indicate a need to initiate control because there is little evidence that bird health is often compromised by infection. Nevertheless, the role of carnivores in the life cycle of *Sarcocystis* sp. infections should be considered when feeding

*Figure 28.3* Relative frequency of grossly visible forms of sarcocystis in selected groups of North American migratory birds.
uncooked, infected waterfowl to house pets and to farm animals such as hogs.

**Human Health Considerations**

*Sarcocystis* sp. presents no known health hazard to humans. The primary importance to humans of sarcocystis in waterfowl is the loss of infected birds for food; the unaesthetic appearance of parasitized muscle may prompt hunters to discard the carcass. Limited evaluations of hunter responses to infected carcasses indicate no reduction in carcass consumption in areas where the infection is commonly seen. Also, the recognized high prevalence of infection in northern shovelers in some areas results in this species often being left unretrieved by some hunters and focuses additional hunting pressure on other species.

*Benjamin N. Tuggle and Milton Friend*

*(Modified from and earlier chapter by Benjamin N. Tuggle)*

**Supplementary Reading**


Eustrongylidosis

Chapter 29

Synonyms
Verminous peritonitis

Cause
Eustrongylidosis is caused by the nematodes or roundworms *Eustrongylides tubifex*, *E. ignotus*, and *E. excisus*. *Eustrongylides* sp. can cause large die-offs of nestlings in coastal rookeries, especially of egrets and other wading birds.

Life Cycle
The three species of *Eustrongylides* that cause disease in birds have similar indirect life cycles that require two intermediate hosts (Fig. 29.1). Four developmental stages of the parasite are required from egg to sexually mature worm. The first larval stage develops within the eggs that are shed in the feces of the bird host and are eaten by freshwater oligochaetes or aquatic worms. The oligochaetes serve as the first intermediate host. The eggs hatch within the oligochaetes, where they develop into second- and third-stage larvae. Minnows and other small fish, such as species of *Fundulus* and *Gambusia*, feed upon the infected oligochaetes and serve as the second intermediate host. The third-stage larva becomes encapsulated on the internal surface areas of the fish, develop into infective fourth-stage larvae, and await ingestion by birds. Predatory fish, which consume infected fish, can serve as paratenic or transport hosts when they are fed upon by birds. Amphibians and reptiles have also been reported as second-stage intermediate hosts and serve as paratenic hosts. Larvae that are infective for birds can penetrate the ventriculus (stomach) within 3–5 hours after a bird ingests an intermediate or paratenic host, and the larvae quickly become sexually mature worms that begin shedding eggs 10–17 days postinfection.

Species Affected
*E. tubifex* has been reported from four different bird families, *E. ignotus* from three, and *E. excisus* from three (Fig. 29.2). Young wading birds are the most common species to have large mortalities from eustrongylidosis (Table 29.1). *Eustrongylides* sp. have also been reported in birds of prey.

Distribution
*Eustrongylides* sp. have been reported from birds throughout much of the world. *E. tubifex* and *E. ignotus* are the species reported within the United States (Table 29.2). Eustrongylid infections within the United States have been reported from many areas (Fig. 29.3). Typical rookeries where birds are infected with *Eustrongylides* sp. are found in coastal areas and consist of dense populations of birds nesting on low islands, often surrounded by canals or ditches. Nesting habitat often includes stands of low trees, such as willows, with an understory that may be submergent, semisubmergent, or upland mixed-prairie species. Inland rookeries are usually adjacent to lakes or rivers, and nesting trees, particularly those used by great blue herons, may be much higher than those in coastal rookeries. Several wading bird species may nest in these areas, but typically one or two species account for most of the birds in the rookery (Fig. 29.4).

Seasonality
Birds can harbor infections yearround. Mortality usually is reported in spring and summer and birds less than 4 weeks old are more likely to die than adults. Disease in older birds tends to be of a more chronic nature and infection may be seen at any time of the year.

Field Signs
Disease results in a variety of clinical or apparent signs that are not specific to eustrongylidosis. However, consideration of the species affected, the age class of birds involved, and the full spectrum of signs may suggest that eustrongylidosis is the cause of mortality. Very early in the infection as the worm is penetrating the ventriculus, some birds will shake their heads, have difficulty swallowing, have dyspnea or labored breathing and, occasionally, regurgitate their food. Anorexia or loss of appetite has been noted in experimentally infected nestlings. It has been speculated that anorexia in combination with sibling competition for food may contribute to the emaciation seen in naturally infected birds. Infected nestlings also may wander from the nest predisposed to predation or trauma or both. Affected nestlings observed during one mortality event became progressively weakened and showed abdominal swelling. Palpation of worms on the ventriculus has been useful for detecting infection in live nestlings.

Gross Lesions
Birds that have been recently infected often have large, tortuous, raised tunnels that are visible on the serosal surface of the proventriculus, ventriculus, or intestines (Fig. 29.5A). The nematodes reside within these tunnels, which are often encased with yellow, fibrous material, and maintain openings to the lumen of the organ so that parasite eggs may be passed out with feces into the environment. A fibrino-peritonitis or fibrin-coated inflammation of the surfaces of the peritoneal cavity (the area containing the organs below
Figure 29.1  Life cycle of *Eustrongylides* sp.

Birds feed on infected transport hosts. Infective larvae reach sexual maturity within bird host.

First stage larvae develop in eggs eaten by oligochaetes (freshwater aquatic worms).

Minnows and other small fish feed upon oligochaetes.

First intermediate host

Eggs hatch within oligochaetes; second-and third-stage larvae are produced within oligochaetes.

Second intermediate host

Bird feeds on infected fish then larvae develop to sexual maturity.

First stage larvae develop in eggs eaten by oligochaetes (freshwater aquatic worms).

Transport (paratenic hosts)

Infected minnows are fed upon by species other than birds.

Bird sheds parasite eggs into the environment in feces.

Definitive bird host

Third stage larvae become encapsulated within body of fish.

Bird feeds on infected transport hosts. Infective larvae reach sexual maturity within bird host.
the heart and lungs) and the intestinal surfaces may be present when larvae or adult worms have perforated the surface of the intestines (Fig. 29.5B). Movement of bacteria from the lumen of the digestive tract to the body cavity results in bacterial peritonitis and secondary infections that can cause the death of an infected bird. Thick-walled granulomas, which are firm nodules consisting of fibrous tissue that forms in response to inflammation with necrotic (dead) centers, caseous (cheesy) airsacculitis or inflammation of the air sacs and intestinal blockages have also been reported. The presence of the parasite is also striking when carcasses are examined. Adult worms can be quite large (up to 151 millimeters in length and 4.3 millimeters in width) and are reddish.

Lesions in chronic or resolving infections are less remarkable and appear as raised, yellow or tan-colored tunnels filled with decomposed worms or worms encased with yellow fibrous material. Some lesions will not have recognizable worm structures intact. Lesions seen in bald eagles that were examined at the National Wildlife Health Center were in the esophagus and were much less severe than those in other fish-eating birds.

**Figure 29.2** Groups of water birds reported to be infected with *Eustrongylides* sp.

Diagnosis

Large tortuous tunnels on the surface of the proventriculus, ventriculus, or intestine of fish-eating birds are most likely due to *Eustrongylides* sp. However, the presence of eustrongylid worms is not diagnostic of the cause of death, especially in older nestlings and adult birds. Therefore, entire carcasses should be provided for disease diagnosis. If interest is limited to confirming the presence of *Eustrongylides* sp., then infected organs and the gastrointestinal tract should be removed and shipped chilled on cool packs to an appropriate laboratory. If shipment is not possible within 24–48 hours, the organs can be frozen or preserved in 10 percent neutral formalin and shipped. Speciation of worms requires a diagnostician who has appropriate training.

**Control**

Control of eustrongylidosis depends on the difficult task of disrupting the parasite life cycle, which is further complicated by the length of time that the eggs can remain viable and that intermediate hosts can remain infective. Under experimental conditions, *Eustrongylides* sp. eggs have remained viable up to 2.5 years and freshwater fish and oligochaetes have been reported to remain infected for more than 1 year. Also, the rather quick maturation of the parasite (once it is inside the bird definitive host), along with the long time period that intermediate and paratenic hosts can remain infected, are a perfect parasite strategy for infecting transient or migratory birds. Thus, the birds in a rookery can quickly infect intermediate/paratenic hosts, which can maintain the parasite until next season’s nesting.

It is known that eutrophication and warm water temperatures (20–30 °C) create optimal conditions for the parasite. It has been reported that infection among fish is highest where external sources of nutrients or thermal pollution alter natural environments. Therefore, water quality is an important factor that in some situations is subject to actions that may decrease transmission of the parasite. Water-quality improvement as a means of disease prevention should be taken into consideration relative to land-use practices and wastewater discharges that may negatively impact egret and heron rookeries and feeding areas for wading birds.

Food sources used for birds being reared in captivity or being rehabilitated for return to the wild should be free of infection with *Eustrongylides* sp. The types of fish and the sources of those fish should be considered before they are used to feed birds.
Table 29.1  Examples of reported wild bird mortality attributed to eustrongylidiosis.

<table>
<thead>
<tr>
<th>Geographic location</th>
<th>Primary species affected</th>
<th>Time of Year</th>
<th>Parasite species</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Virginia Beach, Va.</td>
<td>Red-breasted merganser</td>
<td>Dec.</td>
<td>E. sp.</td>
<td>50 dead, 95 moribund; mature birds were affected.</td>
</tr>
<tr>
<td>Madison County, Ind.</td>
<td>Great blue heron</td>
<td>May</td>
<td>E. ignotus</td>
<td>25 dead and moribund; most birds had fledged the previous year.</td>
</tr>
<tr>
<td>Pea Patch Island, Del.</td>
<td>Snowy egret</td>
<td>May-July</td>
<td>E. ignotus</td>
<td>Approximately 300 hatchlings in one outbreak; most deaths occurred within the first 4 weeks after hatching; other outbreaks have been reported for this location.</td>
</tr>
<tr>
<td>Goat Island, Texas</td>
<td>Snowy egret</td>
<td>Not reported</td>
<td>E. sp.</td>
<td>Nestlings and young of undetermined numbers; high infection prevalence in colony.</td>
</tr>
<tr>
<td>Several colonies in central and southern Florida.</td>
<td>Snowy egret, Great egret</td>
<td>Not reported</td>
<td>E. ignotus</td>
<td>More than 250 nestlings during one event; this geographic area has recurring losses from this parasite.</td>
</tr>
</tbody>
</table>

Table 29.2  Reported geographic occurrence in wild birds of Eustrongylides sp.

<table>
<thead>
<tr>
<th>Geographic area</th>
<th>E. tubifex</th>
<th>E. ignotus</th>
<th>E. excisus</th>
</tr>
</thead>
<tbody>
<tr>
<td>United States</td>
<td>●</td>
<td>●</td>
<td>—</td>
</tr>
<tr>
<td>Canada</td>
<td>●</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Brazil</td>
<td>●</td>
<td>●</td>
<td>—</td>
</tr>
<tr>
<td>Europe</td>
<td>●</td>
<td>—</td>
<td>●</td>
</tr>
<tr>
<td>Russia</td>
<td>●</td>
<td>—</td>
<td>●</td>
</tr>
<tr>
<td>Middle East</td>
<td>—</td>
<td>—</td>
<td>●</td>
</tr>
<tr>
<td>Taiwan</td>
<td>—</td>
<td>—</td>
<td>●</td>
</tr>
<tr>
<td>India</td>
<td>—</td>
<td>—</td>
<td>●</td>
</tr>
<tr>
<td>Australia</td>
<td>—</td>
<td>—</td>
<td>●</td>
</tr>
<tr>
<td>New Zealand</td>
<td>—</td>
<td>●</td>
<td>—</td>
</tr>
</tbody>
</table>
Fig. 29.3 States where *Eustrongylides* sp. infections in wild birds have been reported.

Fig. 29.4 Although many species may nest in wading bird rookeries, one or two species are often predominant.
Human Health Considerations

Humans who have consumed raw or undercooked fish that carry the larval stages of the parasite have experienced gastritis or inflammation of the stomach and intestinal perforation requiring surgical removal of worms.

Rebecca A. Cole

Supplementary Reading


Figure 29.5  (A) Raised tunnels caused by Eustrongylides sp. on intestines of a snowy egret. (B) The debris on the intestinal surfaces of this snowy egret is characteristic of the peritonitis often caused by Eustrongylides sp. infection.
Chapter 30

Tracheal Worms

Synonyms
Gape worm, syngamiasis, gapes

Cause

Infection by tracheal worms often results in respiratory distress due to their location in the trachea or bronchi and their obstruction of the air passage. Infections by these parasitic nematodes or roundworms in waterbirds, primarily ducks, geese, and swans, are usually due to Cyathostoma bronchialis and infection of land birds are usually due to Syngamus trachea. However, both genera infect a variety of species, including both land and waterbirds. Infections with S. trachea have been more extensively studied than infections with Cyathostoma sp. because of its previous importance as a disease-causing parasite of poultry in many parts of the world. Changes in husbandry practices to modern intensive methods for poultry production have essentially eliminated S. trachea as an agent of disease in chickens, but it is an occasional cause of disease in turkeys raised on range.

Life Cycle

Tracheal worms have an indirect life cycle (Fig. 30.1) that requires a paratenic or transport host which transmits the infectious larvae to the definitive host bird, where they reach adulthood and reproduce. Adult S. trachea reside within the trachea. The female releases fertilized eggs, which are swallowed by the bird and voided with the feces into the soil. Eggs may also be directly expelled onto the ground from the trachea. After embryonation (1–2 weeks), infective larvae develop within the egg. Birds can become infected by eating invertebrate paratenic hosts such as earthworms, snails, slugs, or fly larvae that have consumed the eggs. Infective larvae are released from the egg and become encysted within the bodies of these invertebrates and can remain infective for up to three and one-half years. Upon ingestion by birds, the larvae are believed to penetrate the intestinal wall. Some larvae enter the abdominal cavity but most enter the bloodstream, where they are carried to the lungs. After further development in the lungs, the young worms migrate up the bronchi to the trachea. Larvae can reach the lungs within 6 hours after ingestion and eggs are produced by worms in the trachea about 2 weeks after ingestion of those larvae. C. bronchialis is very similar in that earthworms transmit the infective stage to the bird. Infection of birds with C. bronchialis by direct consumption of fully embryonated eggs has been documented experimentally; however, worm burdens were extremely low.

Species Affected

Disease caused by tracheal worms is not commonly reported for free-ranging birds within the United States and Canada, but it is common within the United Kingdom and in some other countries. High infection rates within wild birds in England attest to the potential for this parasite to be a serious pathogen. More than 50 percent of nestling and fledgling starlings, more than 85 percent of jackdaws, and 100 percent of young rooks were found to be infected in one study. Infection rates in adult birds were considerably lower, but they still exceeded 30 percent for starlings and rooks. Within the United States, S. trachea infections have been reported from wild turkeys, other gamebirds, a variety of passernines, (songbirds), and occasionally from other bird species. Large-scale mortalities have occurred among pheasants and other gamebirds being propagated for sporting purposes. Findings from captive bird collections have led to the conclusion that almost any species of cage or aviary bird is susceptible to infection.

S. trachea has been reported infrequently in waterfowl, but members of the genus Cyathostoma sp. are “characteristic” or common parasites of waterfowl. Mortality has been reported for several species of young geese, leading some investigators to suggest that C. bronchialis are potentially important pathogens for geese. Juvenile free-ranging sandhill cranes have also been reported to have died from Cyathostoma sp. infection.

Distribution

S. trachea and Cyathostoma sp. are found worldwide.

Seasonality

Infected birds can be found yearround. Young birds are most commonly affected and, therefore, disease is associated with breeding cycles in the spring to summer months for free-ranging birds.

Field Signs

Most birds that are infected show no signs of disease. In general, the severity of disease is dependent upon the degree of infection and the size of the bird. Small birds are more severely affected than larger birds because their narrower tracheal openings result in greater obstruction by the worms. Respiratory distress is the primary clinical sign of disease. Birds with severe infections open their mouth widely and at the same time stretch out their necks, assuming a “gaping” posture. The adult worms that are attached to the lining of
1. Adult worms produce eggs by sexual reproduction within trachea

2. Female worm releases fertilized eggs, which are swallowed by bird and voided with feces

3. Infective larvae develop within egg and hatch

4. Eggs are eaten by earthworms, snails, slugs, or fly larvae

5. Infective larvae hatch and become encysted within bodies of invertebrates

6. Bird eats infected invertebrate and larvae penetrate intestinal wall and enter blood stream

7. Larvae are carried to lungs via blood stream where larvae undergo further development

8. Young worms migrate up the bronchi to the trachea

*Figure 30.1* Tracheal worm life cycle.
the trachea cause irritation and excess mucus production. This often results in agitated bouts of coughing, head shaking, and sneezing as the birds attempt to dislodge the parasites. Severely infected birds may have most or all of the tracheal opening obstructed by worms, may stop feeding, and may rapidly lose body condition.

**Gross Lesions**

Severely affected birds experience severe weight loss and have poorer development of body mass than uninfected birds, and they often die from starvation (Fig. 30.2). Anemia may also be present due to the blood-feeding habits of the parasites.

**Diagnosis**

Identification of the worms (Fig. 30.3) and evaluation of any associated disease signs are required for a diagnosis. Clinical signs are not diagnostic because similar signs can be seen with some mite infections, aspergillosis, and wet pox.

**Control**

There is no feasible method for controlling tracheal worms in free-ranging birds. Disease prevention should be practiced by minimizing the potential for captive-propagation and release programs to infect invertebrates that are then fed upon by free-ranging birds. Land-use practices that provide direct contact between poultry rearing and wild birds and the disposal of bird feces and litter should also be considered because environmental contamination with infective larvae is a critical aspect of the disease cycle.

**Human Health Considerations**

There are no reports of these nematodes infecting humans.

*Rebecca A. Cole*

**Supplementary Reading**


Heartworm of Swans and Geese

Synonyms
*Filarial heartworm, Sarconema, Sarconema eurycerca*

Cause
Heartworm in swans and geese is caused by a filarial nematode or a roundworm of the superfamily Filarioidea which is transmitted to the bird by a biting louse. The nematode and the louse both are parasites. *Sarconema eurycerca* is the only one of several species of microfilaria or the first stage juvenile of the parasite found in the circulating blood of waterfowl that is known to be pathogenic or cause clinical disease.

Life cycle
*Sarconema eurycerca* has an indirect life cycle (Fig. 31.1) that requires the parasite larvae to develop in an intermediate host before they can become infective for and be transmitted to a definitive host, where they mature and reproduce. Female adult heartworms release microfilariae into the bloodstream of the definitive host bird. The microfilariae infect a biting louse, *Trinoton anserinum*, that subsequently feeds upon the bird. The larvae go through three stages of development within the louse, and the third stage is infectious to birds. A new host bird becomes infected when the louse bites it to feed on its blood and the third-stage larvae move into

![Image](image.png)

*Figure 31.1  Indirect life cycle of Sarconema eurycerca.*
the bird’s bloodstream. The larvae migrate through the bloodstream to the myocardium, which is the middle and thickest layer of the heart wall composed of cardiac muscle. They are nourished by and develop to sexual maturity within the myocardium. The cycle continues as this next generation of mature heartworms release microfilariae into the bloodstream.

Infection with the parasite is not synonymous with disease; that is, the parasite may infect and develop within the bird but not debilitate it.

**Species Affected**

*Sarconema eurycerca* was first identified from a tundra swan (whistling swan) in the late 1930s. It has since been reported from trumpeter, Bewick’s, and mute swans and, from Canada, snow, white-fronted, and bean geese. Varying percentages of swans (4–20 percent) have been found to be infected on the basis of blood smears that were taken from apparently healthy birds during field surveys. Canadian investigators have reported a prevalence of approximately 10 percent of snow geese that were examined at necropsy and which had died from other causes. 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.

**Distribution**

Heartworm is found throughout the range of its swan and goose hosts.

**Seasonality**

It is suspected that while swans and geese are on the breeding grounds, louse infestation and colonization on birds is prevalent. Therefore, the possibility of infection by heartworm is highest while birds are on the breeding grounds.

**Field Signs**

Field signs are not always present in infected birds, and infection cannot be determined by the presence of clinical signs alone. Chronic types of debilitating diseases, such as lead poisoning, may exacerbate louse infestation because birds become lethargic and do not preen. No specific field sign is diagnostic for infection.

**Gross Lesions**

The severity of infection dictates the lesions that are seen at necropsy. Birds may be emaciated or in comparably good flesh. The heart may be enlarged and have pale foci or spots within the myocardium. The thin, long thread-like worms may be visible under the surface layer or epicardium of the heart or the worms may be embedded within the deeper muscle tissue of the myocardium (Fig. 31.2).

**Diagnosis**

A diagnosis of heartworm as the cause of death must be supported by pathologic lesions seen during examination of the heart tissues with a microscope and consideration of other causes. Therefore, whole carcasses should be submitted for diagnostic assessments. If the transit time is short enough to avoid significant decomposition of the carcass and if the carcass can be kept chilled during transit, then chilled whole carcasses should be submitted to qualified disease diagnostic laboratories. If those conditions cannot be met, then carcasses should be submitted frozen.

**Control**

Control of heartworm is not practical for free-ranging birds. Decreasing the opportunity for heavy infestation of the louse intermediate host will result in reduced opportunity for heartworm infection.

**Human Health Considerations**

*Sarconema eurycerca* has not been reported to infect humans.

*Rebecca A. Cole*

**Supplementary Reading**


Chapter 32

Gizzard Worms

Synonyms
Stomach worm, ventricular nematodiasis, amidostomiasis

Cause
Gizzard worms are comprised of several species of parasitic nematodes or roundworms of birds. Severe infections can result in birds becoming unthrifty and debilitated to the extent that they are more susceptible to predation and to infection by other disease agents. The two gizzard worms that are emphasized here are trichostrongylid nematodes that belong to the genera Amidostomum sp. and Epomidiostomum sp. These long (10–35 millimeter), sometimes coiled, thread-like roundworms are found just beneath the surface lining and the grinding pads of the gizzard, and they are most frequently found in waterfowl. Other species of gizzard worms are found in upland gamebirds such as grouse, in psitticine birds such as parakeets, and in passerine or perching birds such as robins in various parts of the world.

Life Cycle
Amidostomum sp. and Epomidiostomum sp. have a direct life cycle in which the infective parasite larvae invade a single host animal for development to reproductive maturity (Fig. 32.1). Embryonated eggs are passed in the feces of an infected host bird. First-stage larvae hatch from the eggs into the surrounding environment in about 24–72 hours, depending on the ambient temperature. These larvae molt twice after they hatch, and the time between molts also depends on the temperature. Larvae are quite resilient, surviving low temperatures and even freezing; they do not, however, survive drying.

After a bird ingests the larvae, most commonly when a bird feeds or drinks, they enter the gizzard and burrow into its surface lining where they molt again before they become adult worms. Adult worms become sexually mature in about 10–15 days after the final molt, and females shed eggs within 15–20 days. The development from egg to adulthood may take as few as 20 days or as many as 35 days depending on environmental conditions. Once a bird is infected, it can harbor gizzard worms for several years.

In contrast to the direct parasite life cycle, other gizzard worms such as Cheilospirura spinosa have indirect life cycles (Fig. 32.2) in which they undergo one or more stages of development in an arthropod (insect) intermediate host. C. spinosa is a common gizzard worm of North American ruffed grouse that also infects partridges, pheasants, quail, and wild turkey. Embryonated C. spinosa eggs that are discharged in the feces of grouse and other infected upland gamebirds are...
ingested by grasshoppers, the intermediate host, and the eggs hatch within the body of the grasshopper. Experimental studies indicate that the larvae then migrate into the body cavity of the grasshopper, where they become loosely encysted or where they invade the muscles. They then become third-stage larvae that are infective for birds; this infective stage is reached about three or three and one-half weeks after the grasshopper ingests the parasite eggs. Fourth-stage larvae (immature adult worms) have been found underneath the gizzard lining of bobwhite quail 14 days after ingestion of infected grasshoppers. Sexual maturity of the parasite is reported to be reached in bobwhites 32 days following ingestion of infected grasshoppers and in 45 days for ruffed grouse.

**Species Affected**

*Amidostomum* sp. and *Epomidiostomum* sp. can be found in a variety of migratory birds, and gizzard worms have been reported in ducks, geese, swans, American coot, grebes, and pigeons (Fig. 32.3). Birds can die from gizzard worm infection, and death of very young birds is more common than death of adult birds. These worms are among the most common parasites of waterfowl, and they generally are more common in geese than in ducks or swans. However, a very high prevalence of infection of canvasback ducks with *Amidostomum* sp. (80 percent) was reported in one study. Infection is most severe in snow geese and Canada geese.

**Seasonality**

Migratory birds first become exposed to gizzard worms on breeding grounds, and they can continue to be exposed throughout their lives. Therefore, no seasonality is associated with this parasitism. The loss of young birds may be particularly high during the fall and winter months because of the combined effects of large worm burdens, the stresses of migration, and competition for food.
Field Signs

There are no field signs that indicate gizzard worm infection. Heavy worm burdens can result in poor growth of young birds, and birds of all ages are subject to emaciation and general weakness. Severe infections can interfere with food digestion by the bird as a result of extensive damage to the gizzard lining and muscle.

Gross Lesions

Obvious changes from the normal appearance of the gizzard result from the development, migration, and feeding of gizzard worms in that organ. The gizzard lining can slough off, become inflamed, hemorrhagic, and become ulcerated as a result of erosion of the grinding pads (Fig. 32.4). Large numbers (greater than 35) of worms can denude the surface lining of the gizzard, causing the edges of the grinding pads to degenerate and separate the pads from the underlying tissue (Fig. 32.5). In geese, portions of the gizzard muscle can die due to the presence of variable numbers of *Epomidiostomum* sp., which migrate through the tissue. Oblong tissue cavities 1–4 centimeters long can also be present (Fig. 32.6), and they can contain granular material that results from tissue reaction to worm migration through the muscle.

Diagnosis

Gizzard worm infection can be determined in live birds by finding and identifying gizzard worm eggs in the feces. The eggs of *Amidostomum* sp. and *Epomidiostomum* sp. are similar in size and appearance, and they require speciation by trained personnel.

Large numbers of worms and lesions in the gizzard lining or gizzard muscle of carcasses are highly suggestive of death caused by gizzard worms. Submit whole carcasses to disease diagnostic laboratories for more thorough evaluation. If it is not possible to submit a whole carcass and you suspect gizzard worms as the cause of mortality, then remove the gizzard (see Chap. 2) and ship it chilled or frozen. If the gizzard has been opened, remove with forceps as many whole worms as possible and place them in a 10 percent formalin solution or a 70 percent ethanol solution; do not freeze these worms. Submit the opened gizzard with the worms or preserve slices of the gizzard muscle in 10 percent formalin and forward them for microscopic examination (see Chap. 2).

*Figure 32.3* Relative frequency of gizzard worms in selected groups of North American migratory birds.

<table>
<thead>
<tr>
<th>Group</th>
<th>Occasional</th>
<th>Rare or not reported</th>
<th>Frequent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Geese</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Swans</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coots</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Puddle ducks</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diving ducks</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sea ducks</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wading birds</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Shorebirds</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gulls and terns</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pelicans</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Raptors</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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Figure 32.4  Canada goose gizzard showing ulcerations in the gizzard lining caused by gizzard worm (*Amidostomum* sp.) infection.

Figure 32.5  Closeup of Canada goose gizzard showing A, denuded surface lining, and B, degeneration of the edges of the grinding pads. Note also C, the separation of the pads from the gizzard lining and D, the presence of worms.
Control

Methods of controlling gizzard worms in free-ranging birds have not been developed. Attempts to do so would involve disruption of the parasite’s life cycle. *Amidostomum* sp. and *Epomidiostomum* sp. have a direct life cycle (Fig. 32.1), and this suggests that transmission potential is greatest in crowded and continuously used habitat because of accumulative fecal contamination, provided that ambient temperatures are warm enough (68–77 °F) for larval development. Newly hatched birds are least resistant to infection, and birds of all ages are susceptible to reinfection.

Gizzard worms such as *C. spinosa* that have indirect life cycles could, theoretically, be controlled by reducing the availability of intermediate hosts to a number that is less than that which would allow transmission to be frequent enough to maintain the parasites. However, such actions, which would require habitat control or the use of insecticides, are generally not warranted because the parasite does not cause a significant number of bird deaths. Also, intermediate hosts, such as grasshoppers, have high food value for birds.

Human Health Considerations

Gizzard worms are not a threat to humans. Nevertheless, people who eat waterfowl gizzards should cook them thoroughly and should discard those that appear unhealthy because other infections may also be present.

Supplementary Reading


Figure 32.6 Areas of tissue destruction and reaction to migrating *Epomidiostomum* sp. in the gizzard muscle of a snow goose.
Acanthocephaliasis

Synonyms
Thorny-headed worms, acanths

Cause
The phylum Acanthocephala contains parasitic worms referred to as thorny-headed worms because both the larval and adult parasites have a retractable proboscis or a tubular structure at the head, which has sharp, recurved hooks or spines. Much like the cestodes or tapeworms, they lack digestive tracts and absorb nutrients from the bird’s intestinal canal. This may weaken the bird and may make it more susceptible to other diseases and to predation.

Adult acanthocephalans are found in a variety of bird species and in other vertebrates. More than 50 species of acanthocephalans have been reported in waterfowl, but reevaluations of acanthocephalan taxonomy are resulting in revised speciation. Nevertheless, numerous species within the phylum are found in birds.

Definitive bird hosts

Intermediate/transport (paratenic) hosts

Figure 33.1 Indirect life cycle of acanthocephalan worms.
Life Cycle

All acanthocephalan species thus far examined have an indirect life cycle (Fig. 33.1) that requires at least one intermediate host. Intermediate hosts tend to be preferred food items of the definitive host; thus, the parasite also uses the intermediate host as a means of transport to the definitive host. Crustacea of the orders Amphipoda, Isopoda, and Decapoda have been identified as common intermediate hosts of acanthocephalans that infect waterfowl. Some acanthocephalans that affect passerines or perching birds are reported to use terrestrial insects as intermediate hosts. Fish, snakes, and frogs have been identified as paratenic hosts in the life cycle of some acanthocephalans that infect birds.

The adult female parasite within the definitive bird host produces eggs that are passed with the bird’s feces into the environment. When the egg is ingested by the intermediate host (insect, crustacean, or centipedes and millipedes), the infective juvenile stage or cystacanth develops within the intermediate host. In many life cycles of acanthocephalans, if this intermediate host is eaten by a vertebrate host which is unsuitable as a definitive host, the cystacanth will penetrate the vertebrate's gut, encyst and cease development. This vertebrate is now a paratenic host. If the paratenic or intermediate host is eaten by a suitable definitive host the cystacanth will attach to the definitive host’s intestinal mucosa via the spined proboscis, mature, mate and produce eggs. A change in body coloration has been noted in some crustaceans infected with certain species of acanthocephalan. It is thought that this change in color increases predation by definitive hosts. This might be an evolutionary adaptation which increases the chances of life cycle completion by the acanthocephalan.

Species Affected

Acanthocephalans infect all classes of vertebrates and are common in birds. Ducks, geese, and swans are considered to be the most commonly infected birds along with birds of prey, and some species of passerines. All age classes can become infected. Severe disease outbreaks have been repeatedly reported from common eiders. Eider mortality from acanthocephalans has been documented throughout the arctic areas of their range and has been attributed to food habits rather than to any increased susceptibility of their species. Historical U.S. Fish and Wildlife Service disease diagnostic records reported heavy infections of acanthocephalans and mortality in trumpeter swans from Montana.

Distribution

Worldwide.

Seasonality

Birds can be infected with acanthocephalans yearround. Epizootics usually correspond with food shortages, exhaustion (resulting from migration or breeding), or stressful circumstances. Mortality in immature and adult male eiders is commonly seen in late winter and early spring. Adult females experience mortality during or after brooding. Eider ducklings often suffer from both acanthocephalans and renal coccidia. Swan cygnets are also susceptible to lethal infections.

Field Signs

Lethargy and emaciation are nonspecific but common clinical signs associated with severe infections.

Gross Lesions

Gross lesions include white nodules on the serosal or external surface of the intestine (Fig. 33.2). Dissection of the nodules will reveal the proboscis of an acanthocephalan. Examination of the intestinal mucosa or internal lining will reveal white-to-orange colored parasites that are firmly attached to the mucosa (Fig. 33.3). Some parasites can penetrate the gut wall and project into the abdominal cavity. Adhesions between the loops of intestine are not uncommon in the severe cases where the intestine has been penetrated.

Figure 33.2 Intestinal loops of a bird infected with acanthocephalans. Parts of the worms protrude through the intestinal wall.

Figure 33.3 Acanthocephalans attached to the inner surface of the intestine of a bird.
Diagnosis

Postmortem examinations are required to reach a diagnosis of cause of death. When possible, submit chilled carcasses for evaluation. Fecal evaluations can be used for determining the presence of infection, but they do not provide a definitive diagnosis of disease. Evaluation by a parasitologist is required to differentiate the acanthocephalan species. If chilled carcasses cannot be submitted, the following alternatives in order of preference are: whole refrigerated intestine, frozen intestine, and formalin-fixed intestine. The formalin-fixed intestine may prohibit species identification but it will allow identification to the genus level as well as assessments of worm burden and tissue response to the parasites.

Control

Control of acanthocephaliasis in free-ranging birds is not practical. Captive flocks can be managed so that aggregations of birds and crustaceans are minimized. One approach that has been suggested for captive flocks is to limit infection in young birds by segregating them on water areas that are not used by other birds. These segregated water areas will presumably have much lower numbers of infected intermediate hosts for the birds to be exposed to. Acanthocephalans that infect mammals have been successfully treated with ivermectin or fenbendazole. Thiabendazole has been recommended for use in birds, but treatment is acknowledged to be difficult and success low.

Human Health Considerations

None

Rebecca A. Cole

Supplementary Reading

Chapter 34

Nasal Leeches

Synonyms
Duck leeches

Cause
Bloodsucking leeches of the genus *Theromyzon* sp. are the only leeches in North America known to feed directly in the nasal passages, trachea, and beneath the nictitating membrane of the eyes of migratory birds. Three species of nasal leeches have been reported from North America, *T. rude*, *T. tessulatum*, and *T. biannulatum*. Other genera of leeches feed on the exposed surfaces of waterfowl.

Species Affected
Nasal leeches affect many aquatic bird species (Fig. 34.1). Affected waterfowl include northern pintail, teal, American wigeon, northern shoveler, ring-necked duck, canvasback, redhead, lesser scaup, bufflehead, gadwall, ruddy duck, white-winged scoter, surf scoter, trumpeter swan, and tundra swan. Geese may also be parasitized but they are parasitized less frequently than ducks and swans.

Distribution
Nasal leech infestations of waterfowl and other migratory birds have not been reported south of the 30th parallel and are most commonly observed in northern areas because these parasites are better adapted to cold-water lakes (Fig. 34.2).

Seasonality
Peak parasitism usually occurs during the spring and summer months when leeches are actively seeking potential hosts and reproducing. During the winter months, the ambient temperatures in frozen ponds and marshes considerably slow their metabolic rate and, thus, their activity. In wetlands kept free of ice during the winter, bird activity may stimulate opportunistic feeding by leeches.

Field Signs
Birds that have leeches protruding from the nares or attached externally to the mucous membranes of the eyes are easily recognized from a distance with the aid of binoculars (Fig. 34.3). Leeches may be so blood-engorged that they resemble small sacks of blood (Fig. 34.4). Infested birds may be seen vigorously shaking their heads, scratching at their bills with their feet, or sneezing in an effort to dislodge the leeches and to force air through blocked nasal passages. These efforts are usually unsuccessful. Nasal and respiratory tract

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**Figure 34.1** Relative frequency of nasal leech infestations in selected groups of migratory birds.
**Figure 34.2** Reported distribution of nasal leeches in North America.

**Figure 34.3** External nasal passage infestations of nasal leeches on (A), the eyes and (B), on the nares of a female redhead duck.
Leech infestations can cause labored breathing and gaping similar to that seen among birds suffering from aspergillosis.

**Gross Lesions**

Severe leech infestations of the eye can result in temporary blindness. Eye damage may be seen as an accumulation of a stringy, cheeselike material beneath the nictitating membrane, as clouding of the cornea, and, in some instances, as collapse of the globe of the eye. Nasal passages (Fig. 34.5), throat, and trachea can become blocked by engorged leeches. The feeding action of *Theromyzon* sp. can cause inflammation and extensive damage to the lining of the nasal cavity.

**Diagnosis**

Nasal leeches are 10–45 millimeters long when they are blood-engorged, are amber or olive colored, and have four pairs of eyes. Those found in the free-living state are green, with variable patterns of spots on the top surface. Diagnosis of parasitism is usually made by seeing blood-engorged leeches protruding from the nares or attached to the eyes (Fig. 34.3), especially in birds that cannot be handled. Birds with internal leech infestations cannot be diagnosed by observation. In cases where the suspected cause of death is nasal leech parasitism, submit the entire carcass for examination. Leeches may depart a dead bird, making diagnosis difficult, or they may move to other areas of the body where they may be overlooked. Therefore, leeches found on carcasses should be collected and submitted with the carcass. They can be shipped alive in pond water and can be maintained in that condition for several months if they are kept refrigerated.

If leeches are to be killed before shipment, they must be preserved in a relaxed state so that species identification can be made. Straighten specimens between two glass slides (Fig. 34.6A) and flood them with a 10 percent formalin solution for 3–5 minutes while applying pressure to the top micro-

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*Figure 34.4* Blood-engorged nasal leeches removed from the nares of a trumpeter swan.

*Figure 34.5* (A) Nasal passages of trumpeter swan infested with blood-engorged *Theromyzon* sp. leeches. (B) Dissected nasal passages of a mallard showing leech infestation in sinus passages.
scope slide (Fig. 34.6B); then place them in a 10 percent formalin solution for about 12 hours to complete the fixation. Afterwards, transfer leeches to a 5 percent formalin solution for preservation (Fig. 34.6C).

Leeches protruding externally from the nares or attached to eyes can be removed with forceps. Leeches in the nasal passages can be removed by submerging the bird’s bill for 5–10 seconds in a 10–20 percent salt solution, and this can be repeated several times if necessary.

**Human Health Considerations**

*Theromyzon* sp. feed exclusively on avian hosts and are not considered a threat to humans.

*Benjamin N. Tuggle*

**Supplementary Reading**


**Control**

Leech infestations in waterfowl are common and can be fatal, especially in cygnets and ducklings. Tissue damage resulting from heavy infestations can facilitate secondary bacterial infections. However, no preventive measures have been developed for combating infestations in wild birds. When they are not feeding on birds, nasal leeches occur as free-living organisms in aquatic environments. Control measures to reduce leech populations might be possible if nasal leeches could be selectively killed. However, leech control must be weighed against the value of leeches as an aquatic bird food item.
Chapter 35

Miscellaneous Parasitic Diseases

Free-ranging wild birds are afflicted with numerous other parasites that occasionally cause illness and death. Some of these parasites, such as two of the trematodes or flukes highlighted below, can cause major die-offs. This section about parasitic diseases concludes with descriptions of some additional parasites that field biologists may encounter in wild birds. This listing is by no means complete and it is intended only to increase awareness of the diversity of types of parasites that might be encountered during examinations of wild birds. One should not assume that the parasites found during the examination of bird carcasses caused their death. Because parasites of birds vary greatly in size from a protozoa of a few microns in length to tapeworms of several inches in length and because they can be found in virtually all tissues, body cavities and other locations within the bird, the observation of the parasites will depend on their visibility and the thoroughness of the examination. Therefore, it is generally beneficial to submit bird carcasses to qualified disease diagnostic laboratories to obtain evaluations of the significance of endoparasites or of ectoparasites. The methods that are used to preserve the carcass, tissues, or other specimens can enhance or compromise the ability of specialists to identify the parasite to species, and even to genera, in some instances. Therefore, whenever possible, it is best to contact the diagnostic laboratory that will receive the specimens and obtain instructions for collecting, preserving, and shipping field samples (See Chapters 2 and 3).

Endoparasites

Trematodes

Most trematodes or flukes have complex life cycles that require two intermediate hosts (Fig. 35.1) in which the parasites develop before they become infective for the definitive, final bird host. In general, a mollusc is the first intermediate host, and is often a species that lives in the aquatic environment. Therefore, the aquatic environment brings potential hosts (waterbirds) and these parasites into close proximity with the definitive hosts (waterbirds) and these parasites into close proximity. Disease caused by this trematode is suspected as a recurring event on the St. Lawrence River in southern Quebec. Infections by S. globulus have also been found elsewhere in Canada. The trematode is also present in the Old World and in Australia. Field signs include lethargy and bloodstained vents, although these signs are also found with duck plague (See Chapter 16). Gross lesions can appear as an inflammation or enteritis that is characterized by obvious hemorrhages in the lower small intestine or as inflammation of the intestinal wall with areas of ulcer-like erosions and the presence of a mixture of blood and fibrin (Fig. 35.2).

Experimental infections of mute swans with S. globulus indicated that as few as 100 metacercariae could be lethal. Juvenile mallard ducks that had no previous exposure to S. globulus died when infected with 550 metacercariae. Some immunity has been shown to exist in experimental infections of mallards. Adult mallards that were given 100 metacercariae and that later were challenged with 2,500 metacercariae survived, but those that received only 2,500 metacercariae died. Depending on how heavily snail populations are infected, some birds can receive a lethal dose during less than 24 hours of feeding. Susceptible waterfowl generally die 3–8 days postinfection after ingesting a lethal dose of S. globulus. Younger birds are generally more susceptible than older birds.

No parasite control measures have been developed. Any attempts at control would need to take into account the fact that the intermediate hosts are different within different geographic areas of the United States. There have been no reports of this parasite infecting humans.

Cyathocotyle bushiensis is another trematode that infects waterfowl and coot in the United States and Canada. This trematode can be found in the lower intestine, most commonly in the cecae or blind pouches extending out from the beginning of the large intestine (Fig. 35.3). The worms are slightly larger than S. globulus, measuring 1.7–1.8 millimeters in length. The life cycle for this parasite is similar to that of S. globulus. Birds become infected by consuming snails harboring the metacercariae. Disease caused by this trematode has been reported in black duck, blue-winged teal, green-winged teal, and coot. This trematode was first described in England and it is most likely limited by the geographic dis-
distribution of the snail, *Bithynia tentaculata*, which serves as the first intermediate host. This snail is found within the Great Lakes Basin in the United States.

There are no field signs associated with infection by *C. bushiensis* that have any diagnostic value. Common gross lesions in birds include hemorrhagic areas with plaque formation and some cheese-like or caseous core formation in the lumen of the cecae. Hemorrhage and plaque formation within the cecae are often present during early stages of infections (5–7 days after infection), whereas semisolid cellular debris that plugs the cecae are found in later infections (day 9 and later). Tissue damage within the birds is directly related to the attachment of the fluke to the mucosa and the effect of secretions by the fluke on cellular process within the intestine. Studies have indicated that there is a negative relationship between the number of flukes present and weight gain by the bird and a positive relationship between the number of flukes present and the number of white blood cells and body temperature. These findings reflect the nutritional impacts (reduced weight gain or weight loss) and body response to infection (increased white blood cells and body temperature). It is not known if weight loss in infected birds is due to cecal and lower intestine dysfunction or if the birds do not feed as much as noninfected birds. It has been suggested that morbidity and mortality are directly related to vascular leakage in the cecae from tissue damage that leads to dehydration of the bird. Experiments have shown that a fluke burden as low as 32 resulted in deaths of Pekin ducklings on day 8 postinfection, which indicates that ducks may succumb rather quickly to infection.

Control of this parasite, as for all trematode infections, would require preventing birds from feeding on infected snails or other invertebrate intermediate hosts. No reports of human infection have been reported for this trematode.

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**Fig. 35.1** General trematode (fluke) life cycle.
Fig. 35.2  Small intestine of lesser scaup that died from infection with *Sphaeridiotrema globulus*. Note the white flecks, which are *S. globulus*, and obvious hemorrhage.

**Fig. 35.3** *Cyathocotyle bushiensis* from the cecae of an American coot.
*Leyogonimus polyoon* (Fig. 35.4) is a newly reported trematode in North America that caused the deaths of over 1,500 and 11,000 coots in Northeastern Wisconsin during the falls of 1996 and 1997, respectively (Fig. 35.5). This trematode is of similar small size (0.7–1.0 millimeters in length) as *S. globulus* (Fig. 35.2). It is known to cause death in coot and common moorhen in Europe, but the Wisconsin outbreaks are the first documentation of this parasite causing mortality in North America. The susceptibility of other North American birds, beside coot, remains unknown. At the Wisconsin location, various waterfowl species were dying from infections of *S. globulus* while the coot were dying from *L. polyoon* infections. None of the waterfowl were found to be infected with *L. polyoon*.

The life cycle for *L. polyoon* is not known, although the suspected first intermediate host is the snail *Bithnia tentaculata*. Investigations at the National Wildlife Health Center (NWHC) have disclosed that snails that were collected in Wisconsin were infected with a cercariae or a larval form of the parasite that fits the literature description of *L. polyoon*. Additional studies are required to confirm that these larval forms are *L. polyoon*. Also, the intermediate host for the infective larval form or metacarcariae has not yet been found. *L. polyoon* infects primarily the upper and middle areas of the small intestine. No significant field signs are associated with infection by *L. polyoon*. Gross lesions seen at necropsy include severe enteritis characterized by thickening of the intestinal wall and a fibrous-to-caseous core of necrotic debris that blocks the lumen of the intestine (Fig. 35.6).

The location where these outbreaks occurred is a lake that is drained by a stream, that is underlain with sand, and that has substantial growths of water weeds. The shoreline has been extensively developed for home sites and other human use, and the lake is used for recreation. *L. polyoon* has not been reported to infect humans.

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**Fig. 35.4** *Leyogonimus polyoon* from the small intestine of an American coot.

**Fig. 35.5** American coot and various species of waterfowl from die-off in Shawano Lake, Wisc. Coot mortality was due to *Leyogonimus polyoon*, but waterfowl mortality was due to *Sphaeridiotrema globulus*. 
Fig. 35.6  (A) Gastrointestinal tract from an American coot that was infected with *Leyogonimus polyoon*. Note the enlarged or swollen areas (arrows). (B) Intestinal tract of an American coot. The intestinal tract has been incised to expose cheesy cores of dead tissue debris caused by *Leyogonimus polyoon* (arrows).
Cestodes

Tapeworms are common in wild birds, but they seldom cause death. Heavy burdens of these parasites may reduce the vigor of the bird and serve as a predisposing factor for other disease agents, or the parasites may occlude the intestine (Fig. 35.7). One genus, *Gastrotaenia* sp., lives in the gizzard and penetrates the keratohyalin lining or the horny covering of the gizzard pads, causing inflammation and necrosis. *Cloacotaenia* sp. inhabit the ureter or the tubular area that transports wastes from the kidneys to the cloaca in some waterfowl.

Nematodes

Trichostrongylidosis, the disease that is caused by *Trichostrongylus tenius*, is not currently a significant problem within the United States. However it is included because trichostrongylidosis, in natural populations of grouse in their native habitat in Scotland and elsewhere in the United Kingdom, demonstrates the impact that a parasite can have on the population dynamics of the bird host that it infects. *T. tenius* is a common nematode or roundworm that is found within the ceca of some types of wild birds, primarily grouse, geese, and poultry. This parasite has a direct life cycle that is closely associated with host food preferences for terrestrial vegetation (Fig. 35.8). Trichostrongylidosis outbreaks can occur whenever birds are hatching because of the synchronous phase of the ecology of the parasite and the feeding habits of bird hosts.

Hatchability of the parasite eggs and survival of the free-living larval stages are moisture and temperature dependent. The parasite eggs do not develop under dry conditions and the free-living larvae are generally killed by freezing temper-
temperatures. When the feces that contain the parasite eggs are kept moist and when the ambient air temperature is suitable, first-stage larvae develop within the egg and hatch in about 2 days; free-living second-stage larvae develop within another 1 1/2-to-2 days; and development to infective third-stage larvae requires an additional 8–16 days. Studies in Scotland indicate that infective larvae crawl to the tips of moist heather and accumulate there in drops of water provided by the misty weather. Grouse that feed on the tips of the heather ingest the larvae along with their food. Infective larvae molt twice more within the ceca of the bird before they become sexually mature adults.

Infections have been reported in chicken, turkey, guinea fowl, pheasant, quail, pigeons, ducks, and geese; but infections are most notable for red grouse because this parasite has clearly been shown to regulate natural populations of this species. Because adult worms can survive in their bird host for more than 2 years, all adult birds evaluated in some populations have been found to be infected, thereby, providing a reservoir for infection of young. In addition, larvae can arrest development within the ceca, overwinter, and then resume development in the spring. The temperature and moisture requirements for hatchability of the parasite eggs and survival of larvae results in synchronized availability of parasites during the period of production of young grouse. This results in the primary occurrence of disease and mortality in grouse during the spring. The hatchlings are exposed to infective larvae soon after the diet for chicks changes from insects to vegetation.

The impacts of infection by *T. tenius* are greater than just chick mortality. These parasites also decrease available energy for egg laying by adult birds. The resting metabolic rate is increased and there is a decrease in food intake by the bird. The resulting impact is reduced fecundity within the population (fewer chicks) along with high chick mortality. This combination of impacts controls population levels.

*Echinuria uncinata* is a common nematode that infects the proventriculus of various waterfowl species. The life cycle is indirect, and the parasite uses zooplankton, especially *Daphnia* sp., as intermediate hosts (Fig. 35.9). Worms mature in a duck approximately 51 days after it eats infected zooplankton. Adult worms, which are approximately 5 millimeters long, burrow headfirst into the mucosa and submucosa of the proventriculus, causing tissue swelling and inflammation. Tumor-like nodules form and can be large enough to obstruct the lumen of the proventriculus. This parasite can be especially dangerous to waterfowl where zooplankton blooms coincide with the hatching of young. Often birds that are late to hatch and do not have fully developed immune systems can consume enough infected zooplankton in a very short period of time to become severely infected. In areas where water is shallow, where zooplankton populations are numerous, and where birds are crowded into the area, this roundworm can be transmitted to many birds during a short time.

This occurred within a population of the endangered Laysan duck on Laysan Island, Hawaii during the fall of 1993. A drought had struck the island and the brine flies that the
ducks feed heavily on were believed to be scarce. This depressed food base may have resulted in a reduced level of nutrition and may have compromised the ability of the birds to withstand infections by *E. uncinata*. Birds from this die-off were severely emaciated, had thickened proventriculi, and nodules along the proventriculi and intestines. The glands within the proventriculus were severely distorted, which suggests that the function of the proventriculus was compromised. Blood samples taken from sick birds suggested that they were emaciated and severely infected with parasitic worms. It was thought that the combination of the drought, aggregation of birds around freshwater seeps, and scarce food sources, combined with the severe parasitism, caused the ducks to die. Other examples exist where the combination of overcrowded waterfowl, a zooplankton population explosion, large numbers of infected zooplankton, and high retention of worms within waterfowl resulted in sufficient pathology by the worms to cause clinical disease and death. Maintaining fast water flow to prevent zooplankton explosions has been successfully employed for disease prevention in captive flocks such as those at waterfowl parks. *E. uncinata* is widely distributed geographically and has not been reported to infect humans.
Protozoa

Histomoniasis is capable of causing catastrophic losses in the wild turkey, a species whose restoration has become a major wildlife management success story. *Histomonas meleagridis*, the protozoan that causes histomoniasis, utilizes the cecal worm, *Heterakis gallinarum* (a nematode), as a vector for entry into the bird hosts (Fig. 35.10). The disease is commonly called blackhead because infections sometimes cause a bluish or blackish appearance of the skin of the head in some birds due to an excessive concentration of reduced hemoglobin in the blood or cyanosis.

Earthworms and other soil invertebrates can become part of the parasite’s life cycle when they feed on fecal-contaminated soil that contains cecal worm eggs infected with histomonads. The cecal worm larvae and histomonads are stored in the body of the earthworm and are transmitted to birds when worms are fed upon. However, earthworms are not required for the life cycle; cecal worm larvae that contain histomonads may be ingested by birds when they feed in a contaminated environment.

Most, if not all, gallinaceous birds are susceptible hosts. Turkey, grouse, chicken, and partridge develop severe disease and suffer high mortality rates that can exceed 75 percent of those infected. Disease is less severe in Hungarian partridge and bobwhite quail. In contrast, pheasant and some other species often do not exhibit signs of disease, but they instead become carriers that maintain the disease cycle. Canada geese that were examined at the NWHC have also been found to have a histomoniasis-like disease. In North America, wild turkey and bobwhite quail are the species most commonly infected in the wild. The disease is found worldwide.

There are no clinical signs specific to histomoniasis. Wild turkeys affected with this disease often are listless, have an unthrifty appearance of ruffled feathers, and stand with drooped wings. The birds may appear depressed, and their feces are often sulfur-yellow in color. This fecal coloration generally occurs early in the disease and, combined with other field signs, it is highly suggestive of histomoniasis. The primary gross lesions seen upon necropsy of infected birds are numerous large, pale grey, discrete circular crater-like areas of necrosis or tissue death within the liver (Fig. 35.11) and thickened caecal walls that often also become ulcerated and hemorrhagic. The lumen of the ceca may also be obstructed by aggregations of yellowish necrotic debris referred to as cecal cores (Fig. 35.12).

Disease prevention should be the major focus for addressing histomoniasis. The introduction of gallinaceous bird species that are disease carriers, such as pheasants, into habitat occupied by highly susceptible species, such as wild turkey, is unwise and it can have catastrophic results. Similarly, because chickens are often carriers of *H. gallinarum* (cecal worms), and often shed histomonads, spreading uncomposted chicken manure onto fields can distribute cecal worm eggs to wild and susceptible species.

Histomoniasis has caused the deaths of wild turkeys that were provided feed in barnyards frequented by chickens. Therefore, when attempting to reestablish wild turkey flocks in areas where they no longer exist and during periods of inclement weather that create food shortages for wild birds, placement of feed stations should be done with consideration of potential carriers of *H. meleagridis* or *H. gallinarum*.

Ectoparasites

In addition to being vectors that transmit disease to birds, ectoparasites can be direct causes of illness and death. Just a few adult ticks feeding on a small bird can cause anemia, reduced growth, weight loss, and contribute in other ways to a depressed state of health. The fowl tick, a soft-bodied tick of the family Argasidae, is the most important poultry ectoparasite in many countries and it is often a factor limiting raising chickens and turkeys. Chickens have also been reported to suffer tick paralysis, which is a motor paralysis or paralysis of the voluntary muscles, from bites of *Argas* sp. ticks. Tick paralysis in songbirds has been associated with...
the bite of the hard-bodied bird tick, *Ixodes brunneus*. Fatal paralysis from bites by this tick has been reported in numerous species of small birds. The engorged ticks in fatal cases are generally found on the bird’s head and they may be attached to its eyelids. Death results from a powerful neurotoxin that is secreted by the tick while it feeds on the bird. Other species of *Ixodes* ticks have been associated with tick paralysis and mortality in marine birds, including albatross and petrels. An ascending motor paralysis that starts at the feet, progresses for 7–10 days, and ends in death has been reported.

Heavy infestations of lice, mites, fleas, flies, and other biting insects have also been responsible for causing illness and even death of wild birds, especially among nestlings. Conditions caused by these insects range from feather loss and skin damage from acariosis or mange, to myiasis or infestation with fly maggots, and anemia. Mites of the genus *Knemidocoptes* are the primary cause of mange in birds, and the mites belong to the same family (Sarcoptidae) of mites that cause mange in mammals and humans. The *Knemidocoptes* sp. mites are specific to birds and they are not a human health hazard.

More knowledge is needed about the role of ectoparasites as causes of bird death. Proper identification of the species associated with bird mortality is an important component of such assessments; therefore, the presence of insects within bird nests in which freshly dead nestlings are found should be recorded, representative parasite specimens should be collected along with any visible parasites on the carcass, and the parasites submitted should be with the carcass. Ticks and any heavy infestations of fleas, lice, and other insects on live birds being handled for banding or other purposes should also be noted, and, when practical, samples should be collected and submitted for identification to a parasitologist or disease diagnostic laboratory. An abundance of such parasites may be indicators of other health problems for the birds.

**Supplementary Reading**


