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

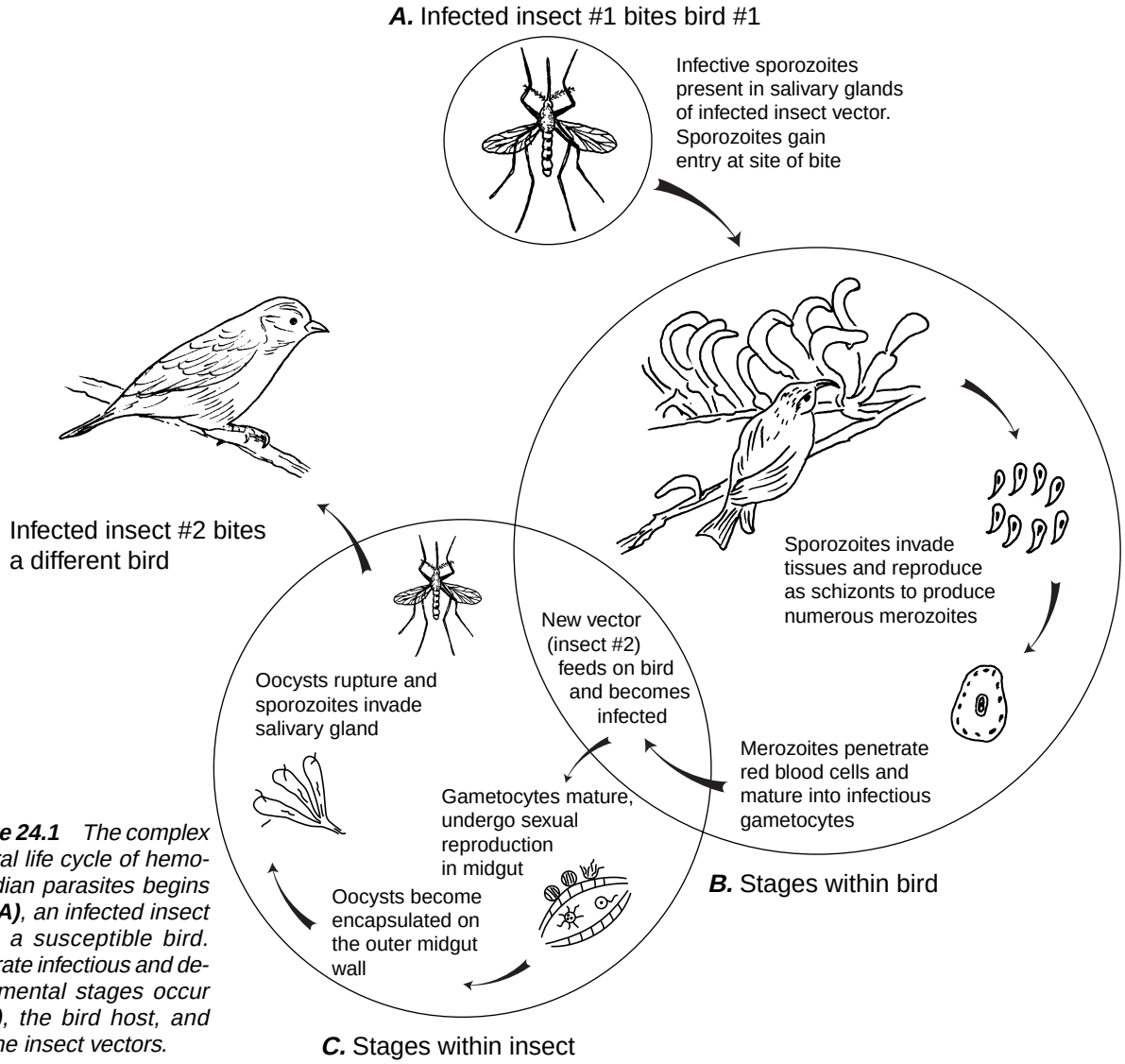

Figure 24.1 *The complex general life cycle of hemosporidian parasites begins with* ***(A)****, an infected insect biting a susceptible bird. Separate infectious and developmental stages occur in* ***(B)****, the bird host, and* ***(C)****, the insect vectors.*

Table 24.1 *Avian hemosporidia parasites and their documented vectors.*

Parasite	Vector type	Common name
Haemoproteus	Ceratopogonidae (*Culicoides* sp.)	Punkies, no-see-ums, sand flies
	Hippoboscidae (*Ornithomyia* sp.)	Hippoboscid or louse flies
Plasmodium	Culicidae (*Culex, Aedes* sp.)	Mosquitoes
Leucocytozoon	Simulidae (*Simulium* sp.)	Black flies

Photo by Jack Jeffrey, U.S. Fish and Wildlife Service

Figure 24.2 *A* ***Culex*** *mosquito feeding on the unfeathered area surrounding the eye of an apapane, a native Hawaiian honeycreeper.*

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-

ited 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

Photos by Carter Atkinson, BRD-PIERC

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.*

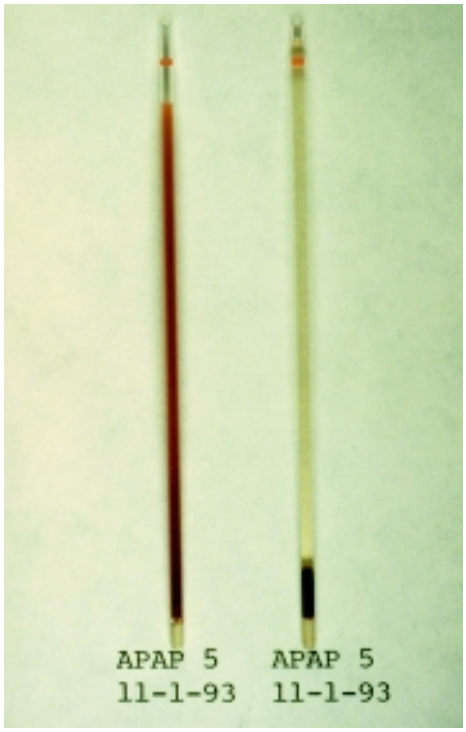

Photo by Carter Atkinson, BRD-PIERC

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).*

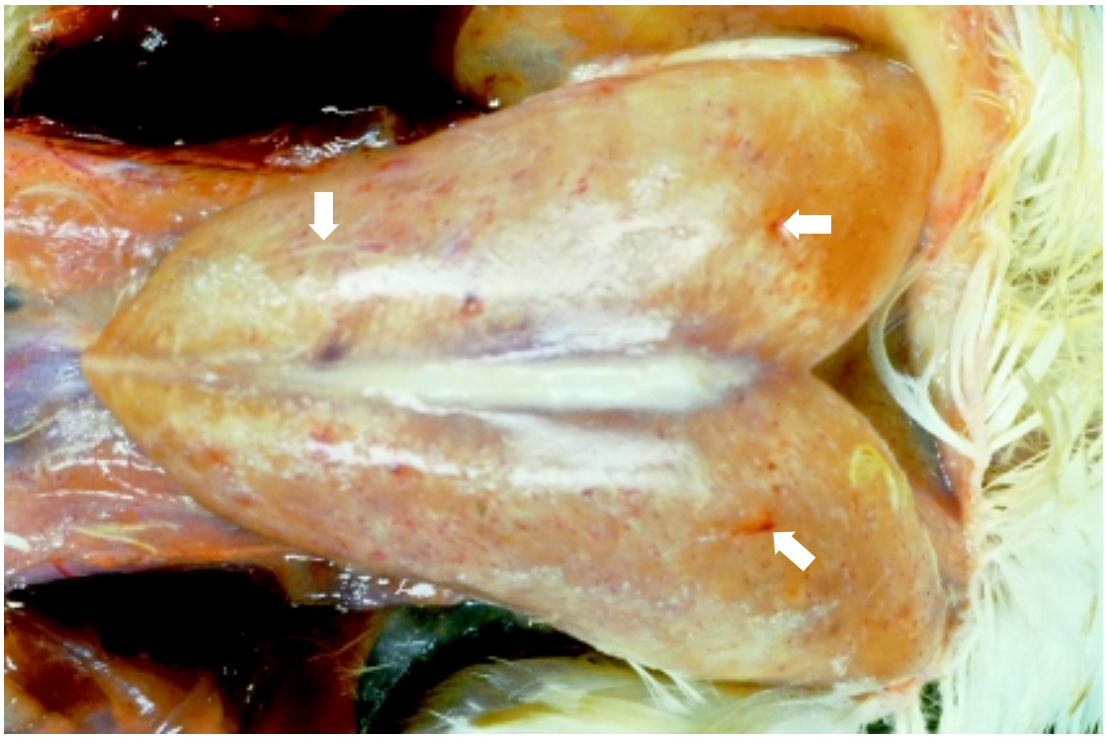

Photo by Carter Atkinson, BRD-PIERC

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.*

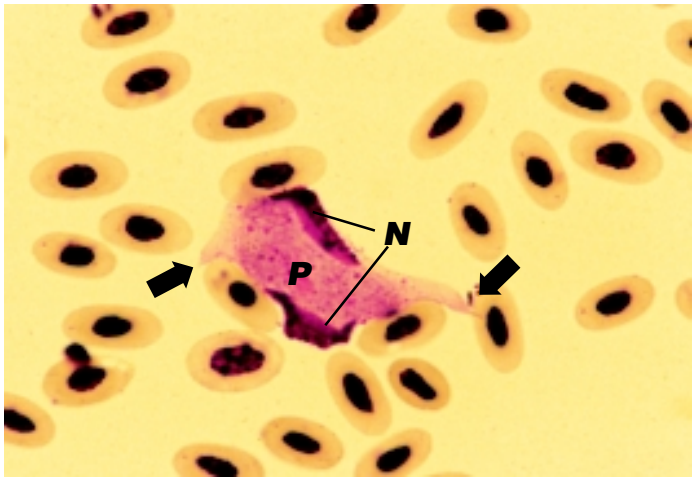

Photo by Carter Atkinson, BRD-PIERC

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).*

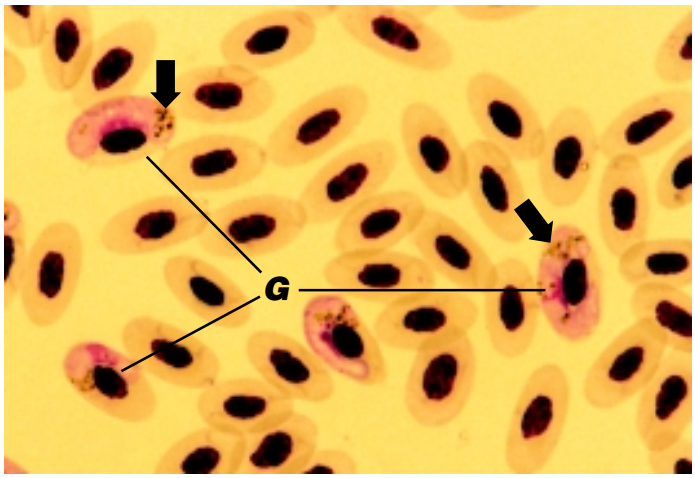

Photo by Carter Atkinson, BRD-PIERC

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).*

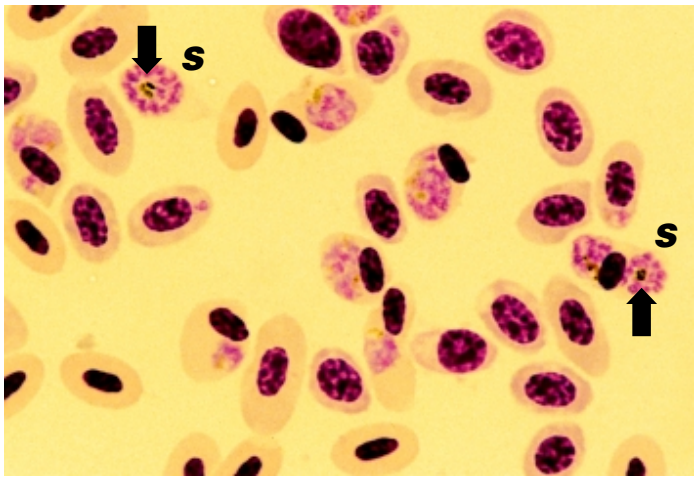

Photo by Carter Atkinson, BRD-PIERC

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).*

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.

Carter T. Atkinson
Pacific Island Ecosystems Research Center
Kilauea Field Station

Supplementary Reading

Atkinson, C.T., 1991, Vectors, epizootiology, and pathogenicity of avian species of *Haemoproteus*: Bulletin of the Society for Vector Ecology, vol.16, p. 109–126.

Atkinson, C.T., and van Riper, C., III, 1991, Pathogenicity and epizootiology of avian haematozoa: *Plasmodium*, *Leucocytozoon*, and *Haemoproteus*, *in* Loye, J.E., and Zuk, M., eds., Bird-parasite interactions, Ecology, evolution, and behavior: New York, Oxford University Press, p. 19–48.

Bennett, G.F., Whiteway, M., and Woodworth-Lynas, C.B., 1982, Host-parasite catalogue of the avian haematozoa: Memorial University of Newfoundland Occasional Papers in Biology Number 5, p. 243.

Greiner, E.C., Bennett, G.F., White, E.M., and Coombs, R.F., 1975, Distribution of the avian hematozoa of North America, v. 53, p. 1,762–1,787.

Greiner, E.C., 1991, Leucocytozoonosis in waterfowl and wild galliform birds: Bulletin of the Society for Vector Ecology, v. 16, p. 84–93.