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**Bacterial Diseases (Field Manual of Wildlife Diseases)**

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Diseases caused by bacteria are a more common cause of mortality in wild birds than are those caused by viruses. In addition to infection, some bacteria cause disease as a result of potent toxins that they produce. Bacteria of the genus *Clostridium* are responsible for more wild bird deaths than are other disease agents. *Clostridium botulinum*, which causes avian botulism, is primarily a form of food poisoning and it is included within the section on biotoxins (see Chapter 38). Other *Clostridium* sp. that colonize intestinal tissues produce toxins that cause severe hemorrhaging of the intestine, thus leading to tissue death or necrosis and intoxication of the bird due to the exotoxins produced by the bacterial cell. The descriptive pathology is referred to as a necrotizing gastroenteritis or necrotic enteritis and the disease as clostridial enterotoxemia. The classic example in gallinaceous birds such as quail, turkey, pheasant, grouse, and partridge, is ulcerative enteritis or quail disease, which is caused by *Clostridium colinimum*; quail are the species most susceptible to that disease. Necrotic enteritis of wild waterbirds, especially geese, has been reported with increasing frequency during recent years. *Clostridium perfringens* has been associated with these deaths.

The frequency of wild bird mortality events and the variety of infectious bacterial diseases causing that mortality has increased greatly during recent decades. Avian cholera has become the most important infectious disease of waterbirds, but it did not appear in North American waterfowl or other waterbirds until 1944. Most of the geographic expansion and increased frequency of outbreaks of avian cholera has occurred since 1970. Avian tuberculosis is a historic disease of captive birds, but it is relatively rare in North American wild birds. The high prevalence of avian tuberculosis infection that has occurred since 1982 in a free-living foster-parented whooping crane population has challenged the survival of that subpopulation of cranes. Salmonellosis has become a major source of mortality at birdfeeders throughout the Nation, and mycoplasmosis in house finches has become the most rapidly spreading infectious disease ever seen in wild birds. This disease reached the Mississippi River and beyond within 2 years of the 1994 index cases in the Washington, D.C. area.

Avian botulism has also expanded in geographic distribution and has gained increased prominence as a disease of waterbirds. It is undoubtedly the most important disease of waterbirds worldwide. Much of the geographic expansion of avian botulism has occurred during the past quarter-century.

As a group, bacterial diseases pose greater human health risks than viral diseases of wild birds. Of the diseases addressed in this section, chlamydiosis, or ornithosis, poses the greatest risk to humans. Avian tuberculosis can be a significant risk for humans who are immunocompromised. Salmonellosis is a common, but seldom fatal, human infection that can be acquired from infected wild birds. This section provides individual chapters about only the more common and significant bacterial diseases of wild birds. Numerous other diseases afflict wild birds, some of which are identified in the chapter on Miscellaneous Bacterial Diseases included at the end of this section.

Timely and accurate identification of causes of mortality is needed to properly guide disease control operations. The magnitude of losses and the rapidity with which those losses can occur, as reflected in the chapters of this section, should be a strong incentive for those who are interested in the conservation of wild species to seek disease diagnostic evaluations when sick and dead birds are encountered. In order to accurately determine what diseases are present, specimens need to be sent to diagnostic laboratories that are familiar with the wide variety of possible diseases that may afflict wild birds. Those laboratories must also have the capability to isolate and identify the causative agents involved. Several sources of wildlife disease expertise that might be called upon when wildlife mortality occurs are identified within Appendix B.
Chapter 7
Avian Cholera

Synonyms
Fowl cholera, avian pasteurellosis, avian hemorrhagic septicemia

Cause
Avian cholera is a contagious disease resulting from infection by the bacterium Pasteurella multocida. Several subspecies of bacteria have been proposed for P. multocida, and at least 16 different P. multocida serotypes or characteristics of antigens in bacterial cells that differentiate bacterial variants from each other have been recognized. The serotypes are further differentiated by other methods, including DNA fingerprinting. These evaluations are useful for studying the ecology of avian cholera (Fig. 7.1), because different serotypes are generally found in poultry and free-ranging migratory birds. These evaluations also show that different P. multocida serotypes are found in wild birds in the eastern United States than those that are found in the birds in the rest of the Nation (Fig. 7.2).

Acute P. multocida infections are common and they can result in bird deaths 6–12 hours after exposure, although 24–48 hours is more common. Susceptibility to infection and the course of disease — whether or not it is acute or chronic — is dependent upon many factors including sex, age, genetic variation, immune status from previous exposure, concurrent infection, nutritional status, and other aspects of the host; strain virulence and other aspects of the bacterium; and dose and route of exposure. Infection in poultry generally results when P. multocida enters the tissues of birds through the mucous membranes of the pharynx or upper air passages. The bacterium can also enter through the membranes of the eye or through cuts and abrasions in the skin. It is assumed that transmission is similar in wild birds.

Environmental contamination from diseased birds is a primary source for infection. High concentrations of P. multocida can be found for several weeks in waters where waterfowl and other birds die from this disease. Wetlands and other areas can be contaminated by the body discharges of diseased birds. As much as 15 milliliters of nasal discharge containing massive numbers of P. multocida have been collected from a single snow goose. Even greater amounts of bacteria enter the environment when scavengers open the

Figure 7.1 Distribution of Pasteurella multocida serotypes from 561 wild bird isolates from the United States.
Figure 7.2 Distribution of *Pasteurella multocida* serotypes from 561 wild birds isolated by waterfowl flyway.

<table>
<thead>
<tr>
<th>FLYWAY</th>
<th>SEROTYPE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atlantic</td>
<td>All others</td>
</tr>
<tr>
<td>Mississippi</td>
<td>All others</td>
</tr>
<tr>
<td>Central</td>
<td>All others</td>
</tr>
<tr>
<td>Pacific</td>
<td>All others</td>
</tr>
</tbody>
</table>

Flyways are administrative units for the management of waterfowl that are geographic representations of the primary migratory patterns of waterfowl.

**Route of transmission and field situation**

**Comments**

**Bird-to-bird contact**
- Secretions from infected birds shedding *P. multocida*.
- Requires close contact, such as when individuals struggle over aquatic plants that they are feeding upon.

**Ingestion**
- Probably most common route for transmission.
- Consumption of diseased carcasses by scavengers and predators.
- Ingestion of *P. multocida* in food and water from contaminated environments.

**Aerosol**
- May be important in heavily contaminated environments, such as during major die-offs.
- Activities that result in splashing of surface waters result in bacteria-laden sprays when water becomes contaminated.

**Insects**
- Biting insects that feed on birds after having fed upon contaminated carcasses or contaminated environments (ticks, mites, flies).
- Insects fed upon by birds (maggots, flies) following ingestion of *P. multocida* by the insect when feeding.

**Animal bites**
- Not thought to be an important route for infection of wild birds.
- Nonfatal bites from small mammals, such as raccoon, can result in *P. multocida* infections that become systemic and possibly initiate disease outbreaks.
- Thought to occur in some domestic turkey flocks, not yet demonstrated in wild birds.

**Fomites**
- Contaminated cages, equipment, and clothing used in field operations can serve as mechanical transport mechanisms for introducing *P. multocida*.
- Environmental persistence of *P. multocida* is sufficient for this to be a consideration when personnel and equipment are used to combat an avian cholera outbreak and then are to be redirected for other activities.

Figure 7.3 Potential means for transmission of avian cholera to free-ranging wild birds.
carcasses of diseased birds. Avian cholera can be transmitted within this contaminated environment in several ways. Ingestion of bacteria in contaminated food and water, including scavenging of diseased carcasses, is an important source of infection for wild birds. The disease can be transmitted by direct bird-to-bird contact, either between infected and noninfected live birds, or between infected carcasses that serve as “decoys” and noninfected live birds. Aerosol transmission is also thought to take place. In wetlands where avian cholera breaks out, the highest concentrations of *P. multocida* are found near the water surface rather than deep in the water column. Birds landing, taking flight, bathing, and otherwise causing disturbance of the water surface cause bacteria-laden aerosols, which can serve to infect those birds. Other means for transmission of avian cholera have also been reported, each of which may occur for specific situations, but none of which are primary means for disease transmission in wild birds (Fig. 7.3).

The role of disease carriers as a means for initiating avian cholera outbreaks in wild birds has long been postulated because chronically infected birds are considered to be a major source for infection of poultry. It has been reported that the only limit to the duration of the chronic carrier state is the lifespan of the infected bird. Disease carriers have been conclusively established for poultry, and *P. multocida* can commonly be isolated from the mouth area or tonsils of most farm animals, dogs, cats, rats, and other mammals (Fig. 7.4). However, types of *P. multocida* that are found in most mammals do not generally cause disease in birds (see Species Affected, this chapter). The role of disease carriers among migratory species of wild birds has long been suggested by the patterns of avian cholera outbreaks in wild waterfowl, but it has not been clearly established by scientific investigations. Recent findings by investigators at the National Wildlife Health Center (NWHC) have provided evidence that disease carriers exist in snow goose breeding colonies. Shedding of *P. multocida* by disease carriers is likely to be through excretions from the mouth, which is the area where the bacteria are sequestered in carriers and is the means for dissemination of *P. multocida* by poultry. Poultry feces very seldom contain viable *P. multocida*, and there is no evidence that *P. multocida* is transmitted through the egg.

### Species Affected

It is likely that most species of birds and mammals can become infected with *P. multocida*; however, there are multiple strains of this bacterium and those different strains vary considerably in their ability to cause disease in different animals. These differences are most pronounced for cross-infections between birds and mammals. Strains isolated from birds will usually kill rabbits and mice but not other mammals. Strains isolated from cattle and sheep do not readily cause clinical disease in birds. However, some strains from pigs have been shown to be highly virulent (very few organ-

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**Figure 7.4** Partial list of domestic species and wild mammals from which *Pasteurella multocida* has been isolated.
isms cause serious disease) for poultry. Also, cultures from the mouths of raccoons were pathogenic or caused disease in domestic turkeys. Bite-wound infections by raccoons have been postulated as a source of avian cholera outbreaks in poultry. An interspecies chain of avian cholera transmission has been described in free-ranging California wildlife that involved waterbirds, mice, and avian scavengers and predators (Fig. 7.5).

More than 100 species of free-ranging wild birds are known to have been naturally infected with *P. multocida* (Fig. 7.6) in addition to poultry and other avian species being maintained in captivity. Infection in free-ranging vultures has not been reported, although a king vulture is reported to have died from avian cholera at the London Zoo. As a group, waterfowl and several other types of waterbirds are most often the species involved in major avian cholera mortalities of wild birds. Scavenger species, such as crows and gulls, are also commonly diagnosed with avian cholera, but deaths of raptors, such as falcons and eagles, are far less frequent (Fig. 7.7). However, there have been several reports of avian cholera in birds kept by falconers, both from birds consuming infected prey when being flown and from being fed birds that died from avian cholera. Waterfowl and coots experience the greatest magnitude of wild bird losses from this disease (Fig. 7.8). In general, species losses during most major outbreaks are closely related to the kinds of species present and to the numbers of each of those species present during the acute period of the die-off. During smaller events, although several species may be present, mortality may strike only one or several species and the rest of the species that are present may be unaffected. Major outbreaks among wild birds other than waterbirds are uncommon.

Impacts on population levels for various species are unknown because of the difficulty of obtaining adequate assessments in free-ranging migratory birds. However, the magnitude of losses from individual events and the frequency of outbreaks in some subpopulations have raised concerns about the biological costs from avian cholera. Disease that is easily spread through susceptible hosts can be devastating when bird density is high, such as for poultry operations and wild waterfowl aggregations (Fig. 7.9). Mortality from avian cholera in poultry flocks may exceed 50 percent of the population. An outbreak in domestic geese killed 80 percent of a flock of 4,000 birds. Similar explosive outbreaks strike in free-ranging migratory birds. Peak mortality in wild waterfowl has exceeded more than 1,000 birds per day.

![Figure 7.5 Example of an interspecies chain for transmission of avian cholera that occurred in a California wetland.](image)
Figure 7.6  Free-ranging wild birds that have been diagnosed with avian cholera.
Studies by researchers at the NWHC indicate that some flocks of snow geese wintering in California have significantly reduced survival rates because of this disease. Evaluation of band returns from midcontinent white-fronted geese and field assessments of other waterfowl populations also suggest decreased survival rates due to avian cholera during some years. Avian cholera has periodically caused heavy losses of breeding eiders and these outbreaks devastate those colonies.

Avian cholera is clearly an important disease of North American waterfowl and it requires more intensive studies to adequately assess impacts on population dynamics. Avian cholera now rivals avian botulism for the dubious honor of being the most important disease of North American waterfowl. Its threat to endangered avian species is continually increasing because of increasing numbers of avian cholera outbreaks and the expanding geographic distribution of this disease.

**Distribution**

Avian cholera is believed to have first occurred in the United States during the middle to late 1880s, but it was unreported as a disease of free-ranging migratory birds prior to the winter of 1943–44 when many waterfowl died in the Texas Panhandle and near San Francisco, California. Avian cholera outbreaks involving free-ranging wild birds have now been reported coast-to-coast and border-to-border within the United States. Although avian cholera is found in many countries, there have been few reports in the scientific literature of die-offs from avian cholera affecting free-ranging wild birds in countries other than the United States and Canada. This disease undoubtedly causes more infections and deaths than are reported, and it is an emerging disease of North American free-ranging migratory birds.

Sporadic cases of avian cholera have been documented in the United States since the early 1940s, and perhaps before, in species such as crows, starlings, grackles, sparrows, and other birds that are closely associated with poultry operations. Most of these wild species are now seldom found to be infected, perhaps due to changes in poultry husbandry and waste disposal practices. Avian cholera also broke out in California in free-ranging quail during the early 1940s and in cedar waxwings in Ohio during 1968. However, waterfowl are the primary species that are affected by this disease.

The emergence of avian cholera as a significant disease for North American waterfowl began about 1970. The frequency and severity of avian cholera outbreaks vary greatly among years and geographic areas but the pattern of continual spread is of major concern (Fig. 7.10). The first outbreaks in eastern Canada involving wild waterfowl were reported during 1964 in eiders nesting on islands in the St. Lawrence Seaway. The first outbreaks in western Canada took place in snow geese during 1977; this disease has occurred annually in western Canada ever since. Several suspect diagnoses of avian chol-

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**Figure 7.7** Relative occurrence of avian cholera in wild birds.

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1. Major die-offs occur almost yearly.
2. Mortality in these species is common but generally involves small numbers of birds.
3. Small number of reports, generally involving individual or small numbers of birds.
Figure 7.9  (A) Dense aggregations of waterfowl facilitate the rapid spread of avian cholera because of the highly infectious nature of this disease.  (B) Large-scale mortality has occurred in such situations.
era have been reported for waterfowl mortality events in Mexico during recent years, but these events lack laboratory confirmation. The absence of confirmed reports of this disease in wild waterfowl in Mexico is likely due to lack of surveillance and reporting rather than to the absence of avian cholera.

In the United States, there are four major focal points for avian cholera in waterfowl: the Central Valley of California; the Tule Lake and Klamath Basins of northern California and southern Oregon; the Texas Panhandle; and Nebraska’s Rainwater Basin below the Platte River in the south-central part of the State. The movement of avian cholera from these areas follows the well-defined pathways of waterfowl movement. The spread of this disease along the Missouri and Mississippi River drainages is also consistent with waterfowl movement. No consistent patterns of avian cholera outbreaks exist within the Atlantic Flyway. There are periodic outbreaks in eider ducks nesting off the coast of Maine and occasional major die-offs of sea ducks, including eiders, within the Chesapeake Bay of Maryland and Virginia (Fig. 7.11).

**Seasonality**

Losses can occur at any time of the year. For poultry, outbreaks of avian cholera are more prevalent in late summer, fall, and winter. Those time periods have no special biological associations, except, possibly, with production schedules in response to holiday market demands that influence poultry age-classes within production facilities. Chickens become more susceptible as they reach maturity. Turkeys are much more susceptible than chickens, and turkeys die at all ages, but the disease usually occurs in young mature turkeys. Losses in domestic ducks are usually in birds older than 4 weeks of age.

For wild waterfowl, a predictably seasonal pattern exists in areas where avian cholera has become well established. This pattern is closely associated with seasonal migration patterns and it has resulted in avian cholera becoming a “disease for all seasons,” killing waterfowl during all stages of their life cycle (Fig. 7.12). Some areas experience prolonged periods of avian cholera mortality. Outbreaks in California
normally start during fall and continue into spring. Other areas have seasonal avian cholera outbreaks in the same geographic location. For example, Nebraska, which has had outbreaks most springs since 1975, now frequently also has outbreaks in the fall.

**Field Signs**

Few sick birds are seen during avian cholera outbreaks because of the acute nature of this disease. However, the number of sick birds increases when a die-off is prolonged over several weeks. Sick birds often appear lethargic or drowsy (Fig. 7.13), and they can be approached quite closely before they attempt to escape. When captured, these birds often die quickly, sometimes within a few seconds or minutes after being handled. Other birds have convulsions (Fig. 7.14), swim in circles, or throw their heads back between their wings and die (Fig. 7.15). These signs are similar to those seen in duck plague and in some types of pesticide poisoning. Other signs include erratic flight, such as flying upside down before plunging into the water or onto the ground, and attempting to land a foot or more above the surface of the water; mucous discharge from the mouth; soiling and mattedting of the feathers around the vent, eyes, and bill; pasty, fawn-colored, or yellow droppings; and blood-stained droppings or nasal discharges, which also are signs of duck plague (duck virus enteritis or DVE).

Always suspect avian cholera when large numbers of dead waterfowl are found in a short time, when few sick birds are seen, and when the dead birds appear to be in good flesh. Death can be so rapid that birds may literally fall out of the sky or die while feeding, with no signs of illness. When sick birds are captured and die within a few minutes, avian cholera should also be suspected. None of the signs described above are unique to this disease; these signs should be recorded as part of any history being submitted with specimens and are considered along with lesions seen at necropsy.

**Gross Lesions**

Under most conditions, birds that have died of avian cholera will have substantial amounts of subcutaneous and visceral fat, except for seasonal losses of fat. The most prominent lesions seen at necropsy are in the heart and liver and, sometimes, the gizzard. Hemorrhages of various sizes are frequently found on the surface of the heart muscle or the coronary band or both (Fig. 7.16). Hemorrhages are also sometimes visible on the surface of the gizzard. Areas of tissue death that appear as small white-to-yellow spots are commonly seen within the liver. Where the area of tissue death is greater, the spots are larger and, in some instances, the area of tissue death is quite extensive (Fig. 7.17).

The occurrence of the abnormalities described for the heart, liver, and gizzard are dependent upon how long the bird lived after it became infected. The longer the survival time, the more abundant and dramatic the lesions. In addition, there may be changes in the color, size, and texture of the liver. There is darkening or a copper tone to the liver, and it may appear swollen and rupture upon handling. Because birds infected with avian cholera often die so quickly, the upper portions of the digestive tract may contain recently ingested food. All of these findings are similar to what might also be seen with duck plague; therefore, laboratory diagnosis is needed.

Freshly dead ducks and geese that have succumbed to avian cholera may have a thick, mucous-like, ropy nasal discharge. The lower portions of the digestive tract (below the gizzard) commonly contain thickened yellowish fluid (Fig. 7.17).
Figure 7.12  Relative monthly probability for the occurrence of avian cholera in migratory waterfowl, expressed as a percentage of outbreaks throughout the year. Information from the National Wildlife Health Center database.
Figure 7.13  Lethargic appearance of drake northern pintail with avian cholera.

Figure 7.14  Avian cholera-infected crow in convulsions.

Figure 7.15  Avian cholera-infected drake mallard. (A) Note tossing of head toward back and circular swimming as evidenced by ripples in water. (B) Bird at death with head resting on back.
Figure 7.16  Hemorrhages of varying degrees of severity are often seen on the hearts of avian cholera-infected birds. (A) Pinhead-sized hemorrhages along fatty areas of the heart are readily evident in this bird. (B) Broad areas of hemorrhage also occur.
Lesions in the livers of avian cholera-infected birds generally appear as small, discrete, yellowish spots, which are dead tissue. Note the variation in size and appearance of these lesions. (A) Note also the absence of any apparent heart lesions in one bird, (B) only a few minor hemorrhages on the coronary band of another bird, (C) and more extensive hemorrhages on the heart muscle of the third bird. Also note the abundance of fat covering the gizzards of all these birds. This fat attests to the excellent condition these birds were in before exposure to the bacterium and to the rapidity with which each bird died.
Figure 7.18  The thickened, yellowish fluid present in the intestines of this avian cholera-infected bird contains millions of bacteria. These bacteria contaminate the environment when the carcass decomposes or is scavenged, serving as a source of infection for other wildlife.

Both of these fluids are heavily laden with *P. multocida* and care must be taken to not contaminate the environment, field equipment, or oneself with these fluids.

### Diagnosis

As with all diseases, isolation of the causative agent is required for a definitive diagnosis. A whole carcass provides the diagnostician with the opportunity to evaluate gross lesions seen at necropsy and also provides all appropriate tissues for isolation of *P. multocida*.

When it is not possible to send whole carcasses, send tissues that can be collected in as sterile a manner as possible in the field. The most suitable tissues for culturing are heart blood, liver, and bone marrow. Remove the entire heart and place it in a Whirl-Pak® bag for shipment as identified in Chapter 2, Specimen Collection and Preservation; do not attempt to remove the blood from the heart. The liver should also be removed and placed in a separate bag. A major portion of this organ (at least half) should be submitted if it cannot be removed intact. These samples must be refrigerated as soon as possible after collection and kept cool during shipment. When shipment is to be delayed for more than 1 day or when transit time is expected to exceed 24 hours, freeze these specimens.

*P. multocida* persists several weeks to several months in bone marrow. The wings of badly scavenged or decomposed carcasses should be submitted whenever avian cholera is suspected as the cause of death, and when more suitable tissue samples are not available.

### Control

Numerous factors must be considered in combating avian cholera (Fig. 7.19). Avian cholera is highly infectious and it spreads rapidly through waterfowl and other bird populations. This process is enhanced by the gregarious nature of most waterfowl species and by dense concentrations of migratory waterbirds resulting from habitat limitations. The prolonged environmental persistence of this bacterium further promotes new outbreaks (Table 7.1). Pond water remained infective for 3 weeks after dead birds were removed from one area in California; bacterial survival in soil for up to 4 months was reported in another study; and the organism can persist in decaying bird carcasses for at least 3 months.

Early detection of avian cholera outbreaks is a first line of defense for controlling this disease. Frequent surveillance of areas where migratory birds are concentrated and the timely submission of carcasses to disease diagnostic laboratories allows disease control activities to be initiated before
Figure 7.19 Some of the many interrelated factors associated with avian cholera outbreaks in free-ranging wild birds.
### Table 7.1 Examples of reported environmental persistence for *Pasteurella multocida*.

<table>
<thead>
<tr>
<th>Substrate</th>
<th>Survival time</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>General</td>
<td>Highly variable</td>
<td>Amount of moisture, temperature, and pH affect survival of <em>P. multocida</em>.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Survival in soils enhanced when moisture content is 50 percent or greater.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Survival in water is enhanced by high organic content and turbidity.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Survival in wetland waters enhanced by presence of magnesium and chloride ions.</td>
</tr>
<tr>
<td>Garden soil</td>
<td>3 months</td>
<td></td>
</tr>
<tr>
<td>Unspecified soil</td>
<td>113 days at 3 °C;</td>
<td>Soil chemistry information needed to properly evaluate data.</td>
</tr>
<tr>
<td></td>
<td>15–100 days at 20 °C;</td>
<td></td>
</tr>
<tr>
<td></td>
<td>21 days at 26 °C</td>
<td></td>
</tr>
<tr>
<td>Poultry yard</td>
<td>2 weeks</td>
<td>Infectious for birds after last death and removal of all birds.</td>
</tr>
<tr>
<td>Water</td>
<td>3 weeks</td>
<td>Following removal of 100 dead snow geese; no other waterfowl use.</td>
</tr>
<tr>
<td></td>
<td>99 days</td>
<td>Water contaminated with turkey litter.</td>
</tr>
<tr>
<td></td>
<td>30 days</td>
<td>In marsh near carcass that had been opened.</td>
</tr>
<tr>
<td>Infected tissues</td>
<td>120 days but not 240 days</td>
<td>American coot hearts buried in marsh after birds died from avian cholera.</td>
</tr>
<tr>
<td>Fomites (Inanimate objects)</td>
<td>8 days but not 30 days</td>
<td>Dried turkey blood on glass at room temperature.</td>
</tr>
</tbody>
</table>

The opportunity to prevent substantial losses is greatest during the early stages of outbreaks, and costs are minimal in comparison with handling a large-scale die-off. Control actions need to be focused on minimizing the exposure of migratory and scavenger bird species to *P. multocida* and minimizing environmental contamination by this organism.

The NWHC recommends carcass collection and incineration as standard procedures. Carcass collection contributes to avian cholera control in several ways. Several milliliters of fluids containing large concentrations of *P. multocida* are often discharged from the mouths of birds dying from this disease, resulting in heavy contamination of the surrounding area. Carcass decomposition results in additional contamination. These carcasses attract (decoy) other birds, thereby increasing the probability for infection. Scavenging of carcasses also transmits the disease through the direct consumption of diseased tissue (oral exposure).

Care must be exercised during carcass collection to minimize the amount of fluid discharged from the mouths of birds into the environment. Birds should be picked up head first, preferably by the bill, and immediately placed in plastic bags. Care must also be taken to avoid contaminating new areas while carcasses are transported to the disposal site. Double-bagging is recommended to prevent fluids leaking from punctures to the inner bag. Bags of carcasses should always be securely closed before they are removed from the area.

Prompt carcass removal also prevents scavenging by avian species that can mechanically transport infected material to other sites or by feeding or drinking at other locations fol-
lowing consumption of infected tissue. This situation is aggravated by apparently longer disease-incubation times in gulls, crows, and some other avian scavengers. Instead of dying within hours or 1–2 days after exposure to virulent strains of *P. multocida*, avian scavengers more typically die after several days to 1–2 weeks, and they may die far from the site of exposure. When these birds die, they may serve as new potential focal points for contamination.

In some instances, population reduction of gulls and crows has been used to limit the role of these species in spreading and transmitting avian cholera. This technique has limited application and it is not recommended as normal operating procedure. To be most effective, population reduction must be undertaken before there is a major influx of scavengers in response to carcass availability. Also, the techniques used must not result in dispersal of infected birds out of the area.

### Population reduction of infected American coots, gulls, terns, and eiders has also been used to directly combat avian cholera. Destruction of migratory birds infected with this disease can be justified only under special circumstances and conditions:

1. The outbreak must be discreet and localized rather than generalized and widespread.
2. Techniques must be available that will allow complete eradication without causing widespread dispersal of potentially infected birds.
3. The methods used must be specific for target species and pose no significant risk for nontarget species.
4. Eradication must be justified on the basis of risk to other populations if the outbreak is allowed to continue.
5. The outbreak represents a new geographic extension of avian cholera into an important migratory bird population.

Habitat management is another useful tool for combating avian cholera outbreaks. In some instances, it may be necessary to prevent further bird use of a specific wetland or impoundment because it is a focal point for infection of waterfowl migrating into the area. Drainage of the problem area in conjunction with creation or enhancement of other habitat within the area through water diversion from other sources or pumping operations denies waterfowl the use of the problem area and redistributes them into more desirable habitat. The addition of a large volume of water to a problem area can also help to dilute concentrations of *P. multocida* to less dangerous levels. These actions require careful evaluation of bird movement patterns and of the avian cholera disease cycle. Movement of birds infected with avian cholera from one geographic location to another site is seldom desirable.

Under extreme conditions, disinfection procedures to kill *P. multocida* may be warranted in wetlands where large numbers of birds have died during a short time period. The environmental impact of such measures must be evaluated and appropriate approvals must be obtained before these actions are undertaken. A more useful approach may be to enhance the quality of the wetland in a way that reduces the survival of *P. multocida*; the best means of accomplishing this is still being investigated.

Hazing with aircraft has been successfully used to move whooping cranes away from a major outbreak of avian cholera. This type of disease prevention action can also be accomplished by other methods for other species. Eagles can be attracted to other feeding sites using road-killed animals as a food source, and waterfowl can be held at sites during certain times of the year by providing them with refuge and food. During an avian cholera outbreak in South Dakota, a large refuge area was temporarily created to hold infected snow geese in an area by closing it to hunting. At the same time, a much larger population of snow geese about 10 miles away was moved out of the area to prevent transmission of the disease into that population. The area closed to hunting was reopened after the desired bird movement had occurred.

Vaccination and postexposure treatment of waterfowl have both been successfully used to combat avian cholera in Canada goose propagation flocks. The NWHC has developed and tested a bacterin or a killed vaccine that totally protected Canada geese from avian cholera for the entire 12 months of a laboratory study. This product has been used for several years with good results in a giant Canada goose propagation flock that has a great deal of contact with free-flying wild waterfowl and field outbreaks of avian cholera. Before use of the bacterin, this flock of Canada geese suffered an outbreak of avian cholera and was successfully treated with intramuscular infections of 50 milligrams of oxytetracycline followed by a 30-day regimen of 500 grams of tetracycline per ton of feed. A NWHC avian cholera bacterin has also been used to successfully vaccinate snow geese on Wrangle Island, Russia, and Banks Island, Canada. Vaccine use in these instances was in association with studies to evaluate avian cholera impacts on survival rates rather than to control disease in those subpopulations.

As yet, there is no practical method for immunizing large numbers of free-living migratory birds against avian cholera. However, captive propagation flocks can be protected by this method. Endangered species can be trapped and immunized if the degree of risk warrants this action. Live vaccines should not be used for migratory birds without adequate safety testing.

### Human Health Considerations

Avian cholera is not considered a high risk disease for humans because of differences in species susceptibility to
different strains of *P. multocida*. However, *P. multocida* infections in humans are not uncommon. Most of these infections result from an animal bite or scratch, primarily from dogs and cats. Regardless, the wisdom of wearing gloves and thoroughly washing skin surfaces is obvious when handling birds that have died from avian cholera.

Infections unrelated to wounds are also common, and in the majority of human cases, these involve respiratory tract exposure. This is most apt to happen in confined areas of air movement where a large amount of infected material is present. Processing of carcasses associated with avian cholera die-offs should be done outdoors or in other areas with adequate ventilation. When disposing of carcasses by open burning, personnel should avoid direct exposure to smoke from the fire.

*Milton Friend*

**Supplementary Reading**


Synonyms

*Mycobacteriosis, tuberculosis, TB*

**Cause**

Avian tuberculosis is usually caused by the bacterium *Mycobacterium avium*. At least 20 different types of *M. avium* have been identified, only three of which are known to cause disease in birds. Other types of *Mycobacterium* rarely cause tuberculosis in most avian species; however, parrots, macaws, and other large perching birds are susceptible to human and bovine types of tuberculosis bacilli. Avian tuberculosis generally is transmitted by direct contact with infected birds, ingestion of contaminated feed and water, or contact with a contaminated environment. Inhalation of the bacterium can cause respiratory tract infections. Wild bird studies in the Netherlands disclosed tuberculosis-infected puncture-type injuries in birds of prey that fight at the nest site (kestrels) or on the ground (buteo-type buzzards), but tuberculosis-infected injuries were not found in accipiters (falcons), which fight in the air and seldom inflict such wounds.

**Species Affected**

All avian species are susceptible to infection by *M. avium*. Humans, most livestock species, and other mammals can also become infected. Recent molecular studies with a limited number of isolates from birds, humans, and other mammals clearly indicated that *M. avium* can be transmitted between birds and pigs, but the studies did not disclose a similar cross transmission between birds and humans for the isolates tested. It is generally accepted that pigs, rabbits, and mink are highly susceptible to *M. avium*; deer can also become infected. Dogs appear to be quite resistant to the avian type of tuberculosis (Fig. 8.1).

In captivity, turkeys, pheasants, quail, cranes, and certain birds of prey are more commonly infected than waterfowl. However, when avian tuberculosis becomes established, it can be a common and lethal disease in captive waterfowl flocks. Chronic infections exist in some captive nene goose flocks, making these flocks unsuitable donors to supplement the wild population of this endangered species. Pheasants are unusually susceptible to avian tuberculosis.

In free-ranging wild birds, avian tuberculosis is found most often in species that live in close association with domestic stock (sparrows and starlings) and in scavengers (crows and gulls). The prevalence of tuberculosis in free-ranging North American birds has not been determined, although generally less than 1 percent of birds examined at postmortem are affected. Sampling biases due to the limited numbers of speci-

![Figure 8.1](image-url)

**Figure 8.1** Relative susceptibility of various animal groups to *M. avium.*
mens examined preclude extending findings to reflect actual prevalence (Fig. 8.2). A decade-long study of nearly 12,000 wild birds necropsied in the Netherlands disclosed that 0.7 percent of the birds had tuberculosis. The sample included waterbirds, birds of prey, songbirds, and pheasants. Studies in the United States disclosed that 0.3 percent of 3,000 waterfowl necropsied were infected with tuberculosis, and a study in British Columbia found tuberculosis in 0.6 percent of more than 600 wild birds. Tuberculosis in whooping cranes stands in marked contrast to other wild birds; approximately 39 percent of the western population’s free-ranging whooping cranes necropsied at the National Wildlife Health Center have been infected with avian tuberculosis.

**Distribution**

Avian tuberculosis is a ubiquitous and cosmopolitan disease of free-ranging, captive, and domestic birds. The disease is most commonly found in the North Temperate Zone, and, within the United States, the highest infection rates in poultry are in the North Central States. Distribution of this disease in free-ranging wild birds is inferred from birds submitted for necropsy; however, the sampling underrepresents both the geographic distribution and the frequency of infection for individual species. Avian tuberculosis likely exists in small numbers of free-ranging wild birds wherever there are major bird concentrations.

**Seasonality**

Seasonal trends of tuberculosis in wild birds have not been documented. The chronic nature of this disease guarantees its presence yearround for both wild and captive birds.

Factors that may influence seasonal exposure to tuberculosis in migratory birds are changes in habitat used, food base during the year, and interspecies contacts. Contaminated sewage and wastewater environments containing tubercle bacilli are more likely to be used by waterfowl during fall and winter than during warmer months. Wastewater sites are often closed to hunting, thereby serving as refuge areas, and warm water discharges to these sites maintain open water in subfreezing temperatures, thus inviting ready use by waterfowl. Predatory and scavenger species such as raptors and crows often ingest many different food items during different periods of the year; scavengers, therefore, may be exposed to tuberculosis through contaminated food yearround. Contact between wild birds and poultry and livestock is often restricted to specific periods of the year owing to husbandry practices. Wild birds may be exposed to *M. avium* in manure that is spread on fields during early spring.

Environmental conditions can greatly affect the susceptibility of birds to tuberculosis and the prevalence of tuberculosis in captive birds. Captive birds that are on an inadequate

![Figure 8.2 Relative occurrence of avian tuberculosis in birds.](image-url)
diet and that are maintained in crowded, wet, cold, poorly ventilated, and unhygienic aviaries have increased susceptibility to tuberculosis.

**Field Signs**

No clinical signs specifically identify avian tuberculosis in birds. Advanced disease and clinical signs are seen most often in adult birds because of the chronic, insidious nature of the disease. Infected birds are often emaciated, weak, and lethargic, and they exhibit wasting of the muscles. These signs are similar to those of lead poisoning and other debilitating conditions. Other signs depend on which body system is affected and signs may include diarrhea, lameness, and unthrifty appearance. Darkening and dulling of plumage have been reported in the United Kingdom for wood pigeons infected with tuberculosis, but not for other species.

**Gross Lesions**

Typical cases of avian tuberculosis in wild birds involve emaciated carcasses with solid-to-soft or crumbly, yellow-to-white or grey nodules that are less than 1 millimeter to several centimeters in size and that are deeply embedded in infected organs and tissues. The liver (Fig. 8.3A) most often contains such nodules, but the spleen (Fig. 8.3B), lung, and intestines (Fig. 8.3C) may also contain similar nodules. Aggregations of these nodules may appear as firm, fleshy, grape-like clusters. Abscesses and nodular growths (Fig. 8.4) have been reported on the skin of birds in the same locations where pox lesions are commonly seen — around the eyes, at the wing joints, on the legs, side of the face, and base of the beak. Other birds have died of avian tuberculosis without any obvious clinical signs or external lesions.

*Figure 8.3* The raised, firm nodules in these organs are typical lesions of avian tuberculosis; (A) liver; (B) spleen; and (C) intestine.
Nodular tuberculosis lesions in internal organs are often grossly similar to those of aspergillosis, and laboratory diagnosis is required to differentiate the two diseases as well as others that produce similar lesions. Less typical lesions resemble those of other diseases. Sometimes the primary lesions seen at necropsy are enlarged livers and spleens that are so fragile that they easily rupture upon being handled. Most of these cases have livers and spleens with a tan-to-green translucence due to amyloid deposits. Less commonly, in situations where nodules are not formed nor is amyloid deposited, the liver and spleen can be large, pale, and firm.

The location of primary lesions is an indication of route of exposure. Intestinal lesions suggest ingestion of *M. avium* in contaminated feed or water. Lesions in the lungs and other areas of the respiratory tract suggest inhalation as the route of exposure.

**Diagnosis**

Typically, tuberculosis is discovered in captive birds during routine investigation of mortality, and in wild birds during carcass examinations associated with die-offs due to other causes. The gross lesions described above (Fig. 8.3) are suggestive of tuberculosis, but a definitive diagnosis is based on bacteriological isolation and identification of the organism. Because *M. avium* is slow-growing and other bacteria can easily overgrow it, a noncontaminated sample is needed for examination. Whole carcasses are preferred, but when a whole carcass cannot be submitted, remove the leg at the hip joint, wrap it in clean aluminum foil, place it in a plastic bag, and freeze it for shipment to a qualified disease diagnostic laboratory. The marrow within the femur has the lowest potential for being contaminated and it provides a good sample for the bacteriologist. When carcass or tissue submissions to a laboratory are not possible within a short time, tissue preserved in 10 percent buffered formalin solution is useful for diagnostic purposes (see Chapter 2, Specimen Collection and Preservation).

The bacterium can also be isolated from infected tissues that show gross lesions. Microscopic studies can provide a diagnosis of tuberculosis, although such studies cannot determine the species of *Mycobacterium*. Because this disease is transmissible to humans, extra care must be taken when handling infected carcasses.

**Control**

Tuberculosis is difficult to detect in free-ranging birds despite its broad geographic distribution. Tuberculosis rarely causes a major die-off, and there are no practical nonlethal testing procedures for mobile wild birds. Therefore, there is no focal point and, hence, no method developed for disease control in wild bird populations. By contrast, tuberculosis can cause die-offs in captive flocks, and mortality has been reported in sea ducks and other birds, including chukar partridge and pheasants. Some captive flocks of wild birds have experienced losses of nearly 30 percent or more from tuberculosis.

Close monitoring of the health of bird populations — free-ranging or captive — is an essential first step toward detecting tuberculosis so that control efforts can be developed and initiated when feasible. Monitoring can best be accomplished by the timely submission of carcasses to disease diagnostic laboratories. Tuberculosis testing of birds maintained in captivity and laboratory analyses of fecal samples from captive and wild flocks also can be used to identify the presence of
this disease. These tests do not detect all infected birds, but the tests are useful for identifying infected flocks.

Fecal contamination of the environment is the major means of tuberculosis dissemination; ingestion of the bacterium in contaminated feed and water is the most common means of disease transmission. Because this bacterium can survive outside of the vertebrate host for long time periods in an organic substrate (Fig. 8.5), a few infected animals can contaminate an area that has prolonged bird-use patterns. The long-term environmental survival of *M. avium* that is shed by disease carriers when combined with repeated site use and, possibly, a high degree of susceptibility to avian tuberculosis may be the factors contributing to the high prevalence of this disease observed in whooping cranes. A site can also be contaminated by wastewater discharges containing *M. avium* and by the application of contaminated manure for fertilizer. Tuberculosis outbreaks in birds have been associated with sewage effluents and discharges from slaughter houses, meat processing plants, and dairies. In one instance, an outbreak occurred in a captive waterfowl flock when contaminated water was sprayed into the enclosure. These events illustrate the importance of disease prevention for addressing tuberculosis in free-ranging and captive wild birds.

The use of wastewater for maintaining captive waterfowl and other wild birds is questionable without adequate testing or treatment or both to assure that the wastewater does not contain tubercle bacilli. Also, the use of wetlands for wastewater discharges and the use of wastewater to create wetlands for migratory bird habitat should be carefully considered because of the possible presence of *M. avium* in the wastewater. Other actions that should be considered include preventing land use that could place tuberculosis-infected swine in close proximity to major wild bird concentrations and not using unexamined chicken and pigeon carcasses as food for raptors being reared in captivity for release into the wild.

Infected flocks of captive birds should be destroyed because treatment is ineffective and because not all infected birds will be detected by current testing procedures. Because of the long-term environmental persistence of the tubercle bacilli, additional bird use of the site should be avoided for approximately 2 years. Vegetation removal and turning of the soil several times during this period will facilitate sunlight-induced environmental decay of the bacilli. Eradication of free-ranging migratory flocks is rarely feasible. However, when a major outbreak of tuberculosis occurs in wild birds, the circumstances should be assessed, and limited population reduction should be considered if the remaining population-at-risk is well defined, limited in immediate distribution, and involves species that can withstand this action. Habitat manipulation, such as drainage, and scaring devices, such as propane exploders, can sometimes be used to deny birds use of areas where tuberculosis outbreaks occur.

The insidious nature of avian tuberculosis combined with the long environmental persistence of the causative bacterium strongly indicate a need to prevent the establishment of this disease in wild bird populations. When the disease becomes established in free-ranging populations, interspecies transmission and the mobility of free-ranging birds could serve to spread it widely. The continued persistence of avian tuberculosis as a major cause of avian mortality in zoological collections attests to the difficulty of disease control.
Human Health Considerations

There are many authenticated cases of *M. avium* infection in people, although humans are considered highly resistant to this organism. Avian tuberculosis is generally considered noncontagious from an infected person to an uninfected person. Infection is more likely to occur in persons with pre-existent diseases, especially those involving the lungs, and in persons whose immune systems are impaired by an illness, such as AIDS or steroid therapy.

*Milton Friend*
(Modified from an earlier chapter by Thomas J. Roffe)

Supplementary Reading

Chapter 9

Salmonellosis

Synonyms
Salmonellosis; paratyphoid; bacillary white diarrhea (a synonym for pullorum disease); pullorum disease\(^1\), fowl typhoid\(^2\)

Cause
Avian salmonellosis is caused by a group of bacteria of the genus salmonella. Approximately 2,300 different strains of salmonellae have been identified, and these are placed into groupings called “serovars” on the basis of their antigens or substances that induce immune response by the host, such as the production of specific antibody to the antigen. Current taxonomic nomenclature considers the 2,300 different serovars to be variants of two species, Salmonella enterica and S. bongori. S. enterica is further subdivided into six subspecies on the basis of biochemical characteristics. This results in complex nomenclature for each serovar, such as, S. enterica subsp. enterica serovar typhimurium. Readers should be aware of this convention for naming salmonellae because they will find this nomenclature in the current scientific literature. In this chapter, different serovars of salmonellae will be referred to by their previous, less complex nomenclature, such as S. typhimurium.

Pullorum disease, (S. pullorum) and fowl typhoid (S. gallinarum) are two classic and distinctive diseases of poultry that have received considerable attention because of their economic impacts. Wild birds have been infected with pullorum disease and fowl typhoid, but wild birds are more commonly infected by the variants of salmonellae that are collectively referred to as paratyphoid forms, of which S. typhimurium is a prominent representative. The paratyphoid forms constitute the great majority of salmonellae, and they are becoming increasingly important as causes of illness and death in wild birds (Table 9.1).

Salmonella infections can be transmitted in many ways (Table 9.2), and the importance of different modes for transmission varies with the strain of salmonellae, behavioral and feeding patterns of the bird species, and husbandry practices when human intervention becomes part of the hatching and rearing processes. For example, ovarian transmission of S. typhimurium occasionally occurs in turkeys, but it is uncommon in chickens. Egg transmission and environmental contamination of rearing facilities are of more importance for infecting poultry than are contaminated feeds. For wild birds and humans, contaminated foods are the primary source for infection; food and water become contaminated by fecal discharges from various sources. Rats, mice, and other species, including reptiles and turtles, in addition to birds, are sources of fecal discharges of paratyphoid forms of salmonellae. Inhalation of the bacterium during close confinement in high humidity environments such as hatching and brooder operations, direct contact with infected birds and animals, and insects are other demonstrated transmission routes for salmonellosis.

Intestinal microflora are an important factor influencing infection and disease by salmonellae in poultry. Very small numbers of salmonellae can cause infection of poultry during the first few weeks of life. Thereafter, the infectious dose becomes progressively higher, apparently because poultry acquire intestinal microflora that protect them against infection even in the presence of a highly salmonella-contaminated environment. This may explain the high prevalence of salmonellosis occasionally found in chicks of some colonial nesting species, such as gulls and terns, and in heron and egret rookeries, but the lower-than-expected infection rates in adult birds from those same colonies. Experimental studies with full-grown herring gulls disclosed a rapid elimination of salmonella bacteria from the intestines of these birds, which suggests that adult herring gulls may be passively, rather than actively, infected and may simply serve as a mechanical transport mechanism for the movement of salmonellae ingested from contaminated environments.

Individual infected birds can excrete salmonella bacteria for prolonged periods of time ranging from weeks to months. Prolonged use of sites by birds and high density of individuals at those sites can result in cycles of salmonellosis within those populations. Persistently contaminated environments result from a small percentage of birds which remain as life-long carriers that intermittently excrete salmonellae into the environment. The environmental persistence of these bacteria is another factor influencing the probability for infections of birds using that site (Table 9.3). The common practices of using sewage sludge and livestock feces and slurry as fertilizers provide another means for infecting wild birds. Tests of sewage sludge often disclose contamination with salmonellae. Survival periods for salmonellae in cattle slurry samples have been reported to range from 11 to 12 weeks and for months in fields where the slurry has been applied as fertilizer. There are numerous reports of the isolation of salmonellae from rivers and streams as a result of pollution by sewage effluent and slurry runoff from fields.

\(^1\) Distinct forms of salmonellosis caused by specific variants of salmonellae.
### Table 9.1  Characteristics of important salmonellae-causing disease in birds.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Salmonella pullorum</th>
<th>Salmonella gallinarum</th>
<th>Salmonella typhimurium</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Common name</strong></td>
<td>Pullorum disease</td>
<td>Fowl typhoid</td>
<td>Salmonellosis</td>
</tr>
<tr>
<td><strong>Natural hosts</strong></td>
<td>Chickens (primary), turkeys</td>
<td>Chickens, turkeys</td>
<td>Wide range of vertebrates; not restricted to birds.</td>
</tr>
<tr>
<td><strong>Age susceptibility</strong></td>
<td>Mortality usually confined to the first 2–3 weeks of age.</td>
<td>Generally infects growing and adult birds; disease also infects young due to egg transmission.</td>
<td>All ages affected; more common in young and often in association with concurrent disease agents.</td>
</tr>
<tr>
<td><strong>Transmission</strong></td>
<td>Infected hatching eggs followed by spread from infected chicks to un-infected chicks that hatch.</td>
<td>Infected carrier birds most important; egg transmission of secondary importance.</td>
<td>Contaminated environment resulting in ingestion through food and water; egg transmission can also occur.</td>
</tr>
<tr>
<td><strong>Relative occurrence in wild birds</strong></td>
<td>Rare in free-ranging species; not maintained within wild populations.</td>
<td>Uncommon in free-ranging species; not maintained within wild populations.</td>
<td>Prevalence varies with species; most common in those species associated with landfills, sewage lagoons, and other waste-disposal sites and those with close associations with livestock and poultry operations.</td>
</tr>
<tr>
<td><strong>Other naturally infected avian species</strong></td>
<td>Ducks, coots, pheasants, partridges, guinea fowl, sparrows, European bullfinch, magpies, canaries, hawk-headed parrot.</td>
<td>Ducks, swans, curlews, pheasants, quail, partridge grouse, guinea fowl, peafowl, wood pigeon, ring dove, rock dove, owls, rooks, jackdaws, sparrows, blackbirds, goldfinches, ostrich, parrots.</td>
<td>Wide range of species; commonly found in gulls and terns and passerine birds using birdfeeding stations. Also reported in herons, egrets, ducks, geese, cormorants, cranes, owls, eagles, falcons, hawks, and other species.</td>
</tr>
<tr>
<td><strong>Current geographic occurrence</strong></td>
<td>Rare in most advanced poultry-producing areas.</td>
<td>Essentially eliminated from commercial poultry within the United States. Low incidence in Canada, USA, and several European countries; significant disease in Mexico, Central and South America, Africa, and Middle East.</td>
<td>Worldwide due to wide range of species infected.</td>
</tr>
<tr>
<td><strong>Relative human health significance</strong></td>
<td>Occasional infections following massive exposure (contaminated food); prompt recovery without treatment.</td>
<td>Rare and of little public health significance.</td>
<td>One of the most common causes of food-borne disease in humans.</td>
</tr>
</tbody>
</table>
Table 9.2  Pathways for transmission of *Salmonella* sp. in birds.

<table>
<thead>
<tr>
<th>Type of transmission</th>
<th>Means</th>
<th>Consequences/processes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vertical</td>
<td>Through contaminanted eggs from infected female; embryo may be infected or surface of egg becomes infected as it passes down oviduct.</td>
<td>Infection of hatchlings at age of greatest susceptibility. Infected hatchlings become source of infection for other hatchlings.</td>
</tr>
<tr>
<td>Horizontal</td>
<td>Bird-to-bird contact</td>
<td>Infected birds shed organism in feces. Birds in close contact inhale salmonellae that become airborne or ingest salmonellae when pecking at contaminated surfaces of infected birds.</td>
</tr>
<tr>
<td>Contaminated environments</td>
<td>Multiple sources of fecal contamination from a wide variety of warm- and cold-blooded species results in ingestion of salmonellae when pecking at contaminated feathers, litter, and other materials. Infected birds and other animals that are fed upon by birds with predatory and scavenging food habits become exposed to salmonellosis. Birds that feed in landfills, dung piles, wastewater discharge areas, and sewage lagoons are at highest risk to acquire infections.</td>
<td></td>
</tr>
<tr>
<td>Contaminated feeds</td>
<td>Salmonella-contaminated feed has been the source of salmonella outbreaks in poultry. Little is known about levels of salmonella contamination in commercial feed used at birdfeeding stations.</td>
<td></td>
</tr>
<tr>
<td>Inapparent infections</td>
<td>Stress of translocation or conditions causing birds to be brought into rehabilitation can result in shedding of salmonellae by carrier birds or result in clinical disease in birds with subclinical infections. Disease can be transmitted to other birds in close proximity; contamination of the environment can result in further transmission, and release of actively shedding birds can serve to spread the disease and contaminate other environments.</td>
<td></td>
</tr>
<tr>
<td>Substrate</td>
<td>Temperature</td>
<td></td>
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<tr>
<td>-------------------------------</td>
<td>-------------</td>
<td>--------</td>
</tr>
<tr>
<td></td>
<td>11 °C</td>
<td>25 °C</td>
</tr>
<tr>
<td>Poultry feed</td>
<td>18 months</td>
<td>16 months</td>
</tr>
<tr>
<td>Poultry litter</td>
<td>18 months</td>
<td>18 months</td>
</tr>
<tr>
<td>Soil from vacated turkey pens</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Urban garden soil</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Hatchery fluff</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Avian feces</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Reptilian feces</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Manure</td>
<td>—</td>
<td>—</td>
</tr>
</tbody>
</table>
Species Affected

All species of birds should be considered susceptible to infection by salmonellae. The outcome of salmonella infections is reported to be highly dependent upon the age of the birds, concurrent stress, serovar and strain virulence, and susceptibility of the host species.

Salmonellosis has been studied as a disease of poultry since at least 1899. Wild bird surveys have often been concurrent with studies of this disease in poultry and as sources for human infections. These and other investigations have resulted in numerous strains of *Salmonella* sp. being isolated from free-ranging (Fig. 9.1) and captive wild birds. However, findings from these studies have also disclosed a much lower infection rate than anticipated and have caused numerous investigators to conclude that in general, salmonellosis is not an important disease of free-ranging wild birds.

The historic patterns of salmonellosis in wild birds are of isolated mortality events involving individual or very small numbers of birds and incidental findings associated with concurrent infections involving other disease agents. Before the 1980s, major mortality events from this disease were rare in free-ranging wild birds.

Prior to the 1980s most isolations of *Salmonella* sp. from free-ranging wild birds were made from apparently healthy birds, were incidental findings from birds with other disease conditions, or were from lethal cases of salmonellosis involving small number of birds. This is no longer the situation. Large-scale mortalities of birds using feeding stations have become common in the United States (Fig. 9.2), and such mortalities are also reported from Canada and Europe, including Scandinavia. Typically, these events are caused by *S. typhimurium* and usually involve passerine birds (Fig. 9.3). European starling, blackbirds, common grackle, and mourning dove are also among the species that have been found dead from *S. typhimurium* at birdfeeding stations.

Salmonellosis has also been the cause of die-offs of aquatic birds including several species of ducks, mute swan, various species of gulls and terns, American coot, double-crested cormorant, eared grebe, and several species of egrets and herons. However, large-scale mortality events in free-ranging populations, except for songbirds and colonial nesting birds, have rarely been reported.

Many species of captive-reared birds commonly become infected with salmonellae and die from salmonellosis. Aquatic species have died from salmonellosis in zoological gardens and other captive collections. Gamebirds, such as grouse and pheasants, being reared in captivity for sporting purposes and cranes being reared for species conservation efforts are often victims of salmonellosis. Mortality is generally confined to chicks.

**Figure 9.1** Relative rates of isolation of *Salmonella* sp. in free-ranging wild birds.
Distribution

Extensive and prolonged control programs have essentially eliminated pullorum disease as a disease confronting commercial poultry production in most of the world and fowl typhoid from most Western countries. In contrast, salmonellosis due to paratyphoid infections occurs worldwide (Table 9.1) and is increasingly prevalent among wild birds in a wide variety of habitats. Salmonellosis in songbirds is clearly an emerging disease of urban and suburban environments and it has also been introduced into remote bird populations, such as Antarctic penguins and skua. The geographic distribution of salmonellosis in free-ranging wild birds is closely associated with sources of environmental contamination that enters the food web of birds and is passed to other species when infected individuals are fed upon by predators and scavengers.

Seasonality

Salmonellosis can present itself at any time of year. Outbreaks at birdfeeding stations are closely associated with the periods of greatest use of those stations (Fig. 9.4); fall and spring die-offs of songbirds from salmonellosis are common in England. Other outbreaks occur among the young of colonial nesting species, such as gulls and terns, shortly after the young are hatched during the summer (Fig. 9.5).

Field Signs

There are no distinctive signs associated with salmonellosis in wild birds. Different species and ages of birds may have different signs even if they are infected with the same serovar; young birds typically exhibit more pronounced signs of disease. Infection may result in acute disease with sudden onset of death, or it may result in a more prolonged course...
of infection that may become septicemic or be characterized by the presence and persistence of bacteria in the blood, or result in localized infection within the body. The disease in poultry has been described to result in gradual onset of depression over a few days and by unthrifty appearance. These birds huddle, are unsteady, shiver, and breathe more rapidly than normal; their eyes begin to close shortly before death; and they exhibit nervous signs including incoordination, staggering, tremors, and convulsions. Blindness has also been reported in some birds.

The rapid death of songbirds at feeding stations has often caused observers to believe the birds had been poisoned. Neurological signs, such as those described above for poultry, have also been reported in infected songbirds. In contrast, young domestic ducklings are reported to die slowly, exhibiting tremors and gasping for air. Their wings often droop and they sometimes stagger and fall over just before death. Like infected chickens, these birds often have pasted vents and eyelids that are swollen and stuck together by a fluid discharge. Commonly reported signs among all species include ruffled feathers, droopiness, diarrhea, and severe lethargy. Chronically infected birds often appear severely emaciated.

Figure 9.4 Seasonal occurrence of salmonellosis outbreaks at birdfeeding stations within the United States.

Figure 9.5 (A) Salmonellosis can cause large-scale losses of colonial nesting birds. (B) Young birds are especially vulnerable.
Gross Lesions

The occurrence and types of gross lesions are highly variable depending on the course of the infection, the virulence of the organism, and the resistance of the host. In acute cases, obvious lesions can be completely absent. Livers often become swollen and crumbly with small reddened or pale spots if the course of the disease has been prolonged. In other infections, so-called paratyphoid nodules develop in the liver and extend into the body cavity. These are small tan-to-white granular nodules that are best seen under a microscope. In some birds, these nodules are more visible and appear as plaques or granular-abscess-like lesions seen within breast muscle and other tissues and organs. Infected songbirds often have yellow, cheesy nodules visible on the surface of the esophagus. When the esophagus is cut open, the nodules may be seen as large, diffuse plaque-like lesions or as discrete, nodular areas within the esophagus (Fig. 9.6).

An acute intestinal infection can be recognized by the reddening of the internal lining of the posterior two-thirds to one-half of the small intestine, the ceca, which are the blind pouches that extend from both sides of the beginning of the large intestine, and the colon. As the disease progresses, the intestinal lining becomes coated with a pale, tightly adher-

Figure 9.6  Lesions of salmonellosis in the esophagus of (A) an English sparrow and (B) and (C) an evening grosbeak. (A) From the surface, these lesions appear as a yellow, cheesy nodule that could be mistaken for a seed taken in as a food item. (B) When the esophagus is opened, lesions may be seen that appear as large, diffuse, plaque-like areas (C) or as a series of discrete, nodular plaques.

Figure 9.7  Necrotic, crumbly cores that appear as thick, cheesy areas are often found in the intestines of birds dying from salmonellosis.
ing, fibrinous material. In some infected birds, the intestinal ceca contain thick, crumbly necrotic cores (Fig. 9.7). Enlargement and impaction of the rectum are commonly reported in domestic ducklings.

Arthritis in the wings of pigeons is common. Domestic ducks with paratyphoid infections often have arthritis of the hips and knee joints. Small external abscesses about 1 millimeter in diameter have been described for infected pigeons and house sparrows. These abscesses appear in small bunches along the underside of the bird along the mid-to-posterior areas of the body.

**Diagnosis**

Gross lesions of salmonellosis can be similar to several other diseases, including avian cholera and colibacillosis. Diagnosis requires laboratory isolation and identification of *Salmonella* sp. from infected tissues in conjunction with pathological findings. Therefore, whole carcasses should be submitted for examination. Birds with markedly abnormal behavior patterns, such as convulsions and tumbling, often have lesions observable by microscopic examination of the brain. Isolation of salmonellae from the intestine without significant lesions and accompanying isolation of the bacteria from other tissues generally indicates that the bird was a carrier, rather than a victim, of salmonellosis.

Salmonellae are often confined to the gut. The ceca offer the greatest potential for obtaining positive cultures for most strains of salmonellae. Therefore, when whole carcasses cannot be submitted, submit the intestine as a minimum sample. The liver and heart should also be removed and submitted, if possible. Wrap each different tissue in a separate piece of aluminum foil. Place the foil-wrapped specimens in tightly sealed plastic bags, and ship them frozen to the diagnostic laboratory (Chapter 2, Specimen Collection and Preservation and Chapter 3, Specimen Shipment).

Fecal droppings can be checked for *Salmonella* sp., but these need special handling and they should not be submitted as diagnostic specimens without prior discussions with the diagnostic laboratory. Submission of whole eggs should be considered when low hatchability is encountered. Egg shells and shell membranes can also be cultured for salmonellae; this is an effective means of detecting salmonellae in eggs that have hatched, provided that the egg fragments have not been subjected to environmental conditions that would destroy the bacteria. Eggs, too, should only be submitted following consultation with disease specialists.

**Control**

Prevention of infection by pathogenic forms of *Salmonella* sp. and control of salmonellosis is warranted for wild bird populations despite the fact that *Salmonella* sp. have been isolated from a wide variety of wild bird species from many different types of habitats. Surveys have disclosed that the prevalence of salmonellae in most wild bird populations is generally low. Other studies have indicated a rapid elimination of salmonellae from the intestines of their avian host, suggesting passive, rather than active, infection in some instances. The relatively recent increase in the frequency of occurrence of large-scale salmonella outbreaks in wild birds, especially songbirds, is without precedent and it suggests that environmental contamination is an important source for infection of birds.

Landfills and waters where sewage effluent is discharged are common feeding areas for gulls, the wild bird species group with the highest prevalence of salmonella infections. Ducks and other waterbirds also feed heavily in areas of sewage effluent, and they generally have a higher prevalence of salmonellae than most land birds except for pigeons and sparrows, two species that feed in manure piles. Raptors are thought to become infected from the prey they feed upon (often small rodents such as mice).

Eliminating point sources of infection should be the focus for combating salmonellosis in wild bird populations (Fig. 9.8). Disease prevention should be practical at birdfeeding stations; the public should be educated to maintain clean feeders and to remove spilled and soiled feed from the area under the feeder. Feeders occasionally should be disinfected with a 1:10 ratio of household bleach and water as part of the disease-prevention program. In the event of a die-off from salmonellosis, more rigorous disinfection of feeding stations is necessary and station use should be discontinued temporarily.

Other potential point sources of infection include garbage, sewage wastewater, and wastewater discharges from livestock and poultry operations. The potential for contaminating migratory bird habitat with *Salmonella* sp. should be considered when wastewater is intentionally used to create wetland habitat; when existing wetlands are used to receive wastewater discharges; when agricultural fields on wildlife areas are to receive manure and slurries as fertilizer; and when development of landfill, livestock, and poultry operations are proposed in areas where contamination of environments used by migratory birds is likely. A 1995 outbreak of *S. enteritidis* in California poultry was traced to sewage treatment plant wastewater which entered a stream that bordered the poultry farm. Contamination of feral cats and wildlife by the waters of the stream was thought to be the source of entry of *S. enteritidis* in the poultry.

Control of salmonellosis in captive flocks of migratory birds is necessary to prevent major losses, especially in young birds. Control of this disease should be of continual concern whenever migratory birds and other wild birds are being propagated for release programs or are being maintained in captivity during rehabilitation. The conditions causing birds to be brought to rehabilitation and the stresses of confinement may result in apparent infections developing into systemic clinical salmonellosis that may jeopardize the well-being of the infected bird and of other birds within the facil-
Figure 9.8  (A) Sources and (B) consequences of salmonellosis in wild birds.
Strict sanitation measures need to be instituted and judiciously followed. Salmonella carriers can be identified by fecal culturing and should be destroyed. Multiple periodic fecal cultures are required to identify carrier birds because salmonellae are intermittently shed from the intestine. All birds that die should undergo necropsy and appropriate laboratory testing to determine the cause of mortality and any actions required to prevent further losses.

Infected adults should never be used for breeding. Antibiotic therapy may aid in overcoming an outbreak of salmonellosis, but antibiotic therapy will not eliminate carriers and vertical transmission via eggs could result in new outbreaks and disease spread. Storage of food in rodent- and insect-proof containers should be part of a disease prevention program. Many outbreaks in domestic poultry operations have been traced to food contaminated by rodent feces because rats and mice are common sources of salmonellae.

**Human Health Considerations**

Bacteria of the genus Salmonella are well-documented human pathogens. “Food poisoning” characterized by acute intestinal pain and diarrhea is the most common form of human infection. However, more serious forms of salmonellosis also affect humans. The general level of *Salmonella* sp. in most species of wild birds is low, but extra care with personal hygiene is warranted by people who handle these birds or materials soiled by bird feces. This consideration is not limited to situations where disease is apparent, and it extends to routine maintenance of birdfeeders, cleaning transport cages, and handling birds during banding and other field activities.

*Milton Friend*

(Modified from an earlier chapter by Richard K. Stroud and Milton Friend)

**Supplementary Reading**


Chapter 10
Chlamydiosis

Synonyms
Parrot fever, psittacosis, ornithosis, parrot disease, Louisiana pneumonitis

Cause
Chlamydiosis refers to an infection with organisms of the genus *Chlamydia* sp., which are bacteria that live within animal cells. *Chlamydia psittaci* is the species generally associated with this disease in birds. The severity of the disease differs by the strain of *C. psittaci* and the susceptibility of different species of birds. As a result, chlamydiosis may range from an inapparent infection to a severe disease with high mortality. The organism is excreted in the feces and nasal discharges of infected birds and can remain infective for several months. Infection commonly occurs from inhaling the bacteria in airborne particles from feces or respiratory exudates. Because of the organism’s resistance to drying, infected bird feces at roosts are especially hazardous.

Species Affected
Chlamydiosis was first recognized as an infectious disease affecting parrots, parakeets, and humans involved in the international parrot trade in the late 1920s to 1930s. Chlamydiosis has since become known as a serious disease of domestic turkeys in the United States, of domestic ducks and geese in central Europe, and as a common infection of domestic and feral pigeons worldwide. The feral city pigeon is the most common carrier of *Chlamydia* sp. within the United States.

Chlamydial infections have been reported from at least 159 species of wild birds in 20 orders, but most isolations have been made from six groups of birds (Figure 10.1). Psittacine birds such as parakeets, parrots, macaws, and cockatiels are most commonly identified with this disease, while among other caged birds *Chlamydia* sp. occurs most frequently in pigeons, doves, and mynahs. Waterfowl, herons, and pigeons are the most commonly infected wild birds in North America (Figure 10.2). Chlamydiosis also occasionally infects gulls and terns, shorebirds, songbirds, and upland gamebirds.

Distribution
Among free-living birds, avian chlamydiosis has been found worldwide in the feral pigeon, in gulls and fulmars on islands of coastal Great Britain, in waterfowl and shorebirds in the Caspian Sea, and in herons, waterfowl, gulls, and doves in the United States. Infected parrots and parakeets have been found throughout the tropics and Australia.

Figure 10.1 Relative occurrence of reported chlamydiosis in the most frequently infected groups of birds. (Adapted from Burkhart and Page, 1971.)
Seasonality

Individual cases may occur at any time because of healthy carriers and latent infections within bird populations. Shipping, crowding, chilling, breeding, and other stressors have been attributed to active shedding of the infectious agent among captive birds with latent infections. Groupings of wild birds together in flocks, such as during spring and fall migrations, may facilitate the transmission of chlamydiosis. In caged birds, the onset of disease following exposure to *C. psittaci* occurs across a broad range of time from as quickly as 3 days to as long as several weeks. Young birds are more susceptible than adults, and the disease can spread rapidly among colonial nesting birds.

Field Signs

Signs of infection depend on the species of bird, virulence of the strain of *Chlamydia sp.*, the physiological condition of the bird as influenced by stressors, and route of exposure to the organism. Chlamydiosis in wild birds is often inapparent and infected birds can serve as asymptomatic carriers. Infection may also result in an acute, subacute, or chronic form of disease. *C. psittaci* can cause severe, acute disease that may be rapidly fatal in highly susceptible species. Birds often become weak, stop eating, and develop purulent (fluid containing pus) discharges of the eyes and nares. Birds tend to become motionless, remain in a fixed position, huddled up with ruffled feathers (Fig 10.3). Birds may have diarrhea, sometimes rust-colored because of the presence of blood, and respiratory distress is common. Feces from birds that stop eating are often dark green. In an outbreak of chlamydiosis in gulls, primarily fledglings died and the birds that were found dead were typically thin. Captive snowy and American egrets with chlamydiosis exhibited weakness, abnormal gait, ruffled feathers, diarrhea, and rapid weight loss; the birds generally died 1–2 days after the onset of signs. In other species of egrets, the infection may be inapparent even though the organism can be isolated from swabs of the cloaca or respiratory tract.

Feral pigeons exhibit many of the same signs; however, their diarrhea is likely to be more frequently tinged with blood. Mortality rates in young pigeons are often very high. Purulent discharges from the eyes of a very sick pigeon should cause the observer to think first of chlamydiosis. Sudden death without any signs of illness has been reported among captive cage birds (Java finch, parrots) and among wild parrots in Australia where king parrots were reported to have fallen out of trees and died within minutes.

Gross Lesions

The most common anatomical change in infected birds is an enlargement of the spleen or splenomegaly or of the liver or hepatomegaly or both, up to three-or-four times normal size (Figure 10.4). During an outbreak of chlamydiosis in gulls, splenomegaly was noted in each of nine birds exam-
ined and hepatomegaly was noted in four of the nine. Pericarditis, which is an inflammation and thickening of the pericardial sac that surrounds the heart (Figure 10.5), is a striking lesion sometimes seen with acute or subacute chlamydiosis. The air sacs may be thickened and the lungs are often congested, appearing darker than normal.

**Diagnosis**

Diagnosis is based upon the isolation of *Chlamydia* sp. from tissues of infected birds. Whole birds should be submitted. When this is not possible, selected tissues should be collected (Chapter 2, Specimen Collection and Preservation and Chapter 3, Specimen Shipment). The lungs, spleen, liver, and affected air sacs are the preferred tissues for microbial examination. Because *C. psittaci* is also a human pathogen, care must be taken in handling carcasses and tissues.

Diagnosis cannot be based on gross lesions alone because the lesions of some other diseases are similar. Chronic avian cholera infection can produce similar gross lesions in gulls, avian malaria can cause enlarged spleens, and early stages of aspergillosis can produce somewhat similar changes in the lungs and air sacs.

**Control**

*Chlamydia* sp. are present in the tissues, feces, discharges from the eyes and nares, and may also be present on plumage of infected birds. When the excreta and discharges dry, the resulting material can become airborne. Infection may be transmitted by direct contact with affected birds, or by inhaling dried bird fecal material or respiratory exudates that contain *Chlamydia* sp. organisms. Sick birds should be collected and euthanized and carcasses should be picked up. The removal and incineration of carcasses will help reduce the amount of infective material in the area. However, the level of human activity in the area should be carefully considered because it may cause redistribution of birds that could result in the spread of infection to new areas.

**Human Health Considerations**

Chlamydiosis can be a serious human health problem, infecting more frequently those who work with birds. The close association between parrots and this disease in humans prompted the United States and most nations of Western Europe to outlaw the importation of parrots and parakeets from 1930 to 1960. Individuals who work in areas in which there is a strong possibility of inhaling airborne avian fecal material should consider wearing a mask or respirator. Dry, dusty areas with bird droppings can be wetted down with a 5 percent solution of household bleach, or a commercial disinfectant. Working with large numbers of birds in dusty, closely confined areas should be avoided as much as possible.

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**Figure 10.3** Classic appearance of an immature little blue heron with severe chlamydia infection.

**Figure 10.4** Enlarged spleen (top) and liver (bottom) of a ring-billed gull affected with chlamydiosis. (From Franson and Pearson, 1995. Reprinted with permission from the Journal of Wildlife Diseases).

**Figure 10.5** Pericarditis in a ring-billed gull that died of chlamydiosis (From Franson and Pearson, 1995. Reprinted with permission from the Journal of Wildlife Diseases.)
Outbreaks have occurred among poultry slaughterhouse workers and there have also been several severe cases among wildlife biologists. These biologists were thought to have become infected from handling snow geese, common egrets, snowy egrets, white-winged doves, and ducks.

Before the availability of antibiotics, chlamydiosis was fatal in about 20 percent of the human cases. Today, such fatalities are rare. However, persons working with birds should inform their physicians of that fact to help avoid potential situations where early signs of chlamydiosis could be overlooked or dismissed.

J. Christian Franson
(Modified from an earlier chapter by Louis N. Locke)

Supplementary Reading
Chapter 11

Mycoplasmosis

Synonyms
Chronic respiratory disease, infectious sinusitis, house finch conjunctivitis

Cause
Mycoplasmosis is caused by infection with a unique group of bacteria that lack cell walls but possess distinctive plasma membranes. Mycoplasma are also the smallest self-replicating life-forms, and they are responsible for a variety of diseases in humans, animals, insects, and plants. These bacteria can cause acute and chronic diseases in hosts that they infect, and they are also implicated with other microbes as causes of disease when the immune system of the host has become impaired through concurrent infection by other disease agents or through other processes. This chapter focuses on mycoplasmal infections of birds, the most significant of which are caused by Mycoplasma gallisepticum (MG), M. meleagridis (MM), and M. synoviae (MS). Only MG is of known importance for wild birds.

Species Affected
Until recently, mycoplasmosis has not been considered an important disease of wild birds. During late winter 1994, eye infections in house finches caused by MG were first observed in the Washington, D.C. area. Since then, mycoplasmosis has rapidly spread throughout much of the eastern range of the house finch. Mycoplasmosis has also appeared in wild populations of American goldfinch within the eastern United States. Clinical or observable disease caused by MG has not previously been found in wild passerine birds in the United States despite a long history and common occurrence of MG in poultry wherever poultry are raised. Molecular studies of isolates from the songbirds shows that those isolates are similar but that they are distinctly different from isolates obtained from poultry.

M. gallisepticum is a known pathogen of upland gamebirds raised in captivity, and it has been isolated from ducks and geese. Studies of mycoplasmosis in Spain have resulted in isolation of MG from free-ranging peregrine falcons, and isolation of MG from a yellow-naped Amazon parrot is further evidence of a diverse host range that can become infected by this organism (Table 11.1). Strain differences of MG exist and differ in their ability to cause clinical disease. Also, isolates of the same strain can vary widely in their ability to cause clinical disease in different species. This variance in the ability to cause clinical disease is, in part, shown by the greater numbers of birds that have antibody to MG than by the presence of mycoplasmosis in species and popu-

Table 11.1  Reported occurrence of selected avian mycoplasmas of poultry in selected wild avian species. [Frequency of occurrence: ● frequent, ○ common, ◆ occasional, ○ infrequent or not reported. Square symbol indicates free-ranging species. All other reports are natural infections in captive-reared birds.]

<table>
<thead>
<tr>
<th>Type of bird</th>
<th>Mycoplasma sp.</th>
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<tbody>
<tr>
<td></td>
<td>M. gallisepticum (MG)</td>
<td>M. meleagridis (MM)</td>
<td>M. synoviae (MS)</td>
<td>M. gallinarum</td>
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<tr>
<td>Chicken</td>
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<td>○</td>
<td>●</td>
<td>●</td>
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<tr>
<td>Domestic turkey</td>
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<td>○</td>
<td>●</td>
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<tr>
<td>Pigeons</td>
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<td>○</td>
<td>●</td>
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<tr>
<td>Peafowl/guinea fowl</td>
<td>○</td>
<td>○</td>
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<td>○</td>
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<tr>
<td>Pheasants/quail/partridge</td>
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<tr>
<td>Wild turkey</td>
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<tr>
<td>Ducks/geese</td>
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<tr>
<td>Parrots</td>
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lations tested. The isolates of MG from wild songbirds do not cause significant disease in chickens.

Chickens and turkeys are commonly infected with MG, and direct contact of susceptible birds with infected carrier birds causes outbreaks in poultry flocks. Aerosol transmission via dust or droplets facilitates spread of MG throughout the flock. Transmission through the egg is also important for poultry, and MG is thought to spread by contact with contaminated equipment. The highly gregarious behavior of house finches and their use of birdfeeders likely facilitates contact between infected birds or with surfaces contaminated with the bacteria. Infected finches are thought to be responsible for spreading this disease because they move between local birdfeeders and to distant locations during migration.

*M. meleagridis* causes an egg-transmitted disease of domestic turkeys, and it appears to be restricted to turkeys. Clinical disease has not been documented in wild turkeys, and reports of infection in other upland gamebirds have not been confirmed. Airborne transmission and indirect transmission by contact with contaminated surfaces also happen. *M. synoviae* has a broader host range than MM. Chickens, turkeys, and guinea fowl are the natural hosts. Several other species have been naturally infected, and others have been infected by artificial inoculation. Transmission is similar to that for MG, except that MS spreads more rapidly.

Many other avian mycoplasmas have been designated distinct species, some of which are identified in Table 11.2. The number of mycoplasma species identified from birds has increased rapidly during recent years and it will continue to grow. For example, *M. sturni* was recently isolated from the inner eyelids (conjunctiva) of both eyes of a European starling that had the clinical appearance of MG infection in house finches. Enhanced technology is providing greater capabilities for studying and understanding the biological significance of this important group of microorganisms. Too little is known about mycoplasma infections in wild birds to cur-

### Table 11.2 Primary hosts of some mycoplasma species isolated from birds. [—, no data available.]

<table>
<thead>
<tr>
<th>Mycoplasma species</th>
<th>Primary host</th>
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<tr>
<td></td>
<td>Chicken</td>
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<tr>
<td><em>M. gallisepticum</em></td>
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<tr>
<td><em>M. synoviae</em></td>
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<tr>
<td><em>M. iowae</em></td>
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<td><em>M. gallopavonis</em></td>
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<tr>
<td><em>M. cloacale</em></td>
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<tr>
<td><em>M. gallinarum</em></td>
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<tr>
<td><em>M. gallinaceum</em></td>
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<tr>
<td><em>M. pullorum</em></td>
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<tr>
<td><em>M. iners</em></td>
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<tr>
<td><em>M. lipofaciens</em></td>
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<tr>
<td><em>M. glycolphilum</em></td>
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<tr>
<td><em>M. columbinasale</em></td>
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<tr>
<td><em>M. columbinum</em></td>
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<tr>
<td><em>M. columborale</em></td>
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<tr>
<td><em>M. anatis</em></td>
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<tr>
<td><em>M. anseris</em></td>
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<tr>
<td><em>M. imitavis</em></td>
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<tr>
<td><em>M. sturni</em></td>
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<tr>
<td><em>M. buteonis</em></td>
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<tr>
<td><em>M. falconis</em></td>
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<tr>
<td><em>M. gypis</em></td>
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</table>
rently assess the significance of these organisms as a disease factor, although the house finch situation clearly illustrates the potential for clinical disease to occur. Of added significance is the suppression of reproduction through lowered egg production that commonly affects poultry. Reproduction has also been suppressed during natural MG infections of captive chukar partridge, pheasants, peafowl, and other species and during experimental studies with MM in wild turkey. Preliminary studies at the National Wildlife Health Center (NWHC) with *M. anatis* isolated from a wild duck resulted in reduced hatchability of mallard eggs inoculated with that isolate and decreased growth of the infected hatchlings.

Mycoplasmas have been recovered from domestic or semi-domestic ducks since 1952, but the bacteria have not been reported from wild North American waterfowl before a 1988–1990 waterfowl survey by scientists from the NWHC. *M. anatis* has more recently been isolated from wild shoveler ducks and coot and from a captive saker falcon during surveys conducted in southern Spain. The finding of *M. anatis* in three different major groups of wild birds (Falconiformes, Gruiformes, Anseriformes) demonstrates how the ability of a single strain to infect different avian groups could facilitate interspecies transmission.

**Distribution**

Avian mycoplasmas cause disease in poultry and other captive-reared birds worldwide. The current reported distribution of mycoplasma-caused conjunctivitis in wild songbirds roughly corresponds with the distribution of the eastern house finch population (Fig. 11.1).

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**Figure 11.1** Reported geographic spread of house finch inner eyelid inflammation (conjunctivitis) since the initial 1994 observation. (Data adapted from reports in the scientific literature and personal communications between the National Wildlife Health Center and other scientists.)
Seasonality

Because mycoplasmas in poultry are commonly transmitted through the egg and are present in carrier birds, there is no distinct seasonality associated with disease in those species. Observations of house finch conjunctivitis are most frequent when birds are using birdfeeders during the colder months of the year.

Field Signs

Mycoplasma infections in poultry are generally more severe than those reported for house finches, the only wild bird for which any substantial field observations of clinical disease have been made. The prominent field signs are puffy or swollen eyes and crusty appearing eyelids (Fig. 11.2). A clear to somewhat cloudy fluid drainage from the eyes has been reported for some birds. Birds rubbing their eyes on branches and birdbuffer surfaces have also been reported. Other observations of infected birds include dried nasal discharge, severely affected birds sitting on the ground and remaining at feeders after other birds have departed, and birds colliding with stationary objects due to impaired vision. The European starling recently diagnosed to have been infected by *M. sturni* had similar clinical signs and was apparently blind.

Initial field signs observed during a natural outbreak of MG in a backyard gamebird operation included foamy eyes, excessive tearing, and severely swollen sinuses in chukar partridge and ring-necked pheasant, along with reduced egg production. As the disease progressed, severe depression, lethargy, and weight loss preceded respiratory distress and death. Eye inflammation was the only sign observed in Indian blue peafowl that became infected.

A captive saker falcon from Spain infected with *M. anatis* displayed signs of respiratory illness in addition to involvement of the eyes. Irregular breathing, wheezing, and a mucous discharge from the nose and beak were seen in this bird along with anorexia or loss of appetite. These signs are typical of mycoplasmosis in poultry.

Gross Lesions

Mycoplasmosis lesions in wild birds reflect the observed field signs. Infected house finches typically have a mild to severe inflammation of one or both eyes and the surrounding area including swollen, inflamed eyelids; a clear to a cloudy, thickened discharge from the eye; and drainage from the nares of the bill (Fig. 11.2). Chukar partridge and pheasant naturally infected with MG have had moderate to severe swelling of the eyelids, mild to moderate tearing, swelling of one or both of the sinuses near the eyes, and moderate to large amounts of cheesy discharge within the sinuses.

Diagnosis

Mycoplasma are among the most difficult organisms to grow from clinical specimens because of their fastidious
nature, intimate dependence upon the host species they colonize, and slow growth on artificial media. The greatest success in isolating MG from house finches has been when tissue swabs were obtained from live trapped, freshly killed, or fresh dead birds. There has been limited success from frozen carcasses. When mycoplasma is suspected, contact with a disease diagnostic laboratory is recommended to obtain guidance on how to handle specimens. If field conditions permit, selective media provided by a diagnostic laboratory should be inoculated with swabs from the inner eyelids, sinus, the funnel-shaped area at the back of the sinuses where they split right and left (choanal cleft), and trachea of suspect birds and shipped to the laboratory with the freshly killed or dead birds from which those swabs were made. If birds can be submitted, they should be chilled, rather than frozen, and immediately transported to a qualified disease diagnostic laboratory.

Control

Routine cleaning and disinfection of birdfeeders with household bleach is recommended to prevent mycoplasmosis and other diseases that can be transmitted at birdfeeders. A 10 percent solution of household bleach applied weekly for feeders with high bird use will reduce the potential for contaminated surfaces to transmit disease. Close observation of birds using feeders and the prompt reporting of suspect cases of mycoplasmosis to authorities will provide the opportunity for early intervention based on timely diagnosis and for initiating an appropriate disease-control strategy specific to the location and population involved. Special consideration needs to be given to the fact that house finch conjunctivitis is a new and emerging disease problem that has been documented in two additional species of songbirds. One of these included a case where a blue jay being rehabilitated in a cage previously occupied by an infected house finch became infected. That case demonstrates the need for adequate cleaning and disinfection of cages used in wildlife rehabilitation. Birds that survive infection can become disease carriers that serve as a source for initiating new outbreaks. Also, aerosol and egg transmission of mycoplasmosis is common for poultry. Similar transmission is likely for wild birds and must be taken into consideration during the rehabilitation of wild birds infected with mycoplasmosis.

The potential for interspecies transmission of MG from poultry to upland gamebirds being reared in captivity for sporting purposes must also be considered. This same consideration exists for raptors that may be fed poultry carcasses and waste.

Human Health Considerations

None. Mycoplasmas that infect birds are not known to be hazards for humans.

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Supplementary Reading


Disease in free-ranging birds is caused by many other pathogenic bacteria in addition to those illustrated within this section. These other diseases are currently considered less important because of their infrequent occurrence, the small numbers of birds generally lost annually, or because they primarily result from infection by opportunistic pathogens and they require concurrent disease processes for them to become apparent. The following brief highlights about the more important of these diseases are included to acquaint readers with their existence and provide some basic information about their ecology.

**Erysipelas**

Erysipelas is caused by infection with the bacterium *Erysipelothrix rhusiopathiae*. This disease is primarily associated with swine and domestic turkeys, but it has been diagnosed in many groups of birds (Fig. 12.1) and in mammals. The causative agent has also been isolated from the slime layer of marine and freshwater fish and from crocodiles. Erysipelas is found worldwide. Little is known of the ecology of this disease in birds. Most reports of erysipelas in free-ranging birds involve individuals or small numbers of birds, but major die-offs can occur. The largest recorded die-off killed an estimated 5,000 birds, primarily eared grebes, during 1975 on the Great Salt Lake, Utah. Small numbers of waterfowl (green-winged teal, northern shoveler, and common mergansers) and a few herring gulls also died. Erysipelas has also been diagnosed as the cause of a die-off of brown pelicans in southern California during the late 1980s. Other free-ranging birds diagnosed with erysipelas include hawks, crows, raven, wood pigeon, starling, doves, finches, and European blackbird. The causative bacterium is able to survive in the environment for prolonged periods of time, and it was isolated from grebe carcasses approximately 18 weeks after their death during the Great Salt Lake mortality event. The bacteria probably are transmitted through ingestion, such as when gulls feed on carcasses, or entry of the organism through cuts and abrasions. Humans are susceptible to infection. Most human cases involve localized infections resulting from entry through a cut in the skin. Human cases have been fatal when the disease progressed to an infection of the blood and spreads throughout the body (a septicemic infection).

*Figure 12.1*  Reported occurrences of erysipelas in birds.
New Duck Disease

*Pasteurella anatipestifer* causes an important disease of domestic ducks that has infrequently caused the deaths of wild birds. This disease has also killed domestic turkeys and chickens and captive-reared pheasants, quail, and waterfowl. Major mortality events from infection with *P. anatipestifer* have occurred in free-ranging black swans in Tasmania and in tundra swans in Canada. New duck disease has also been diagnosed as the cause of mortality in small numbers of other free-ranging birds, including lesser snow geese. In the domestic duck industry, mortality primarily involves birds 2–3 months old. The swans that died in Tasmania and Canada were primarily young-of-the-year, which is consistent with mortalities of captive wild waterfowl. Birds can die within 24–48 hours after the onset of clinical signs of listlessness, a droopy appearance, fluid discharges from the eyes and bill, greenish diarrhea, and variety of nervous system disorders. The most prominent lesion seen during postmortem examination is a fibrinous covering on the surface of various organs such as the liver and heart (Fig. 12.2).

Necrotic Enteritis

Necrotizing enteritis is caused by an enterotoxemia or toxins in the blood produced in the intestine resulting from infections with *Clostridium perfringens*. This disease is found throughout much of the world where poultry are produced, and it is often an important cause of mortality for adult domestic breeder ducks. Sporadic cases have been diagnosed in waterfowl collections and in wild mallards, black ducks, and Canada goose. A die-off in Florida involved mallards and other wild ducks along with several species of shorebirds and wading birds. Wild ducks are also reported to have died from this disease in Germany.

During recent years, increasing numbers of small die-offs have been detected in snow geese, Canada geese, and white-fronted geese in Canada and the United States. An abrupt change in diet associated with seasons and bird migrations are thought to disrupt the intestinal microflora and allow *C. perfringens* to proliferate in the intestine. The toxins produced by these bacteria are the cause of death. The onset of death is generally rapid and without obvious clinical signs. Severe depression is sometimes observed in chickens along with reluctance to move, diarrhea, and ruffled feathers. Lesions generally appear as a mixture of dead cellular materials and plasma debris, tan-yellow in color, that covers much of the lower region of the intestine of affected waterfowl (Fig. 12.3).

Ulcerative Enteritis

Quail are highly susceptible to infection by *Clostridium coliunum*, the cause of ulcerative enteritis or “quail disease.” Outbreaks of this disease in free-ranging wild birds are rare, but outbreaks have been reported for California quail in Washington State. This acute bacterial infection is charac-
characterized by sudden onset followed by rapid spread through the flock. Outbreaks have been reported worldwide wherever game birds are raised in captivity under crowded conditions. In addition to upland game species such as grouse, quail, pheasant, and partridges, outbreaks have been reported in chickens, pigeons, and robins. Mortality in young quail can reach 100 percent of the flock. Gross lesions vary and depend upon how long the bird lives following infection. Ulcers within the intestine originate as small yellow spots or infected areas with hemorrhagic borders and progress to circular forms that may join together as large areas of dead tissue that resemble thickened mucous membranes with raised edges (Fig. 12.4). Liver lesions include yellow areas of tissue death or necrosis along the edges of the liver and scattered grey spots or small yellow circumscribed spots within the liver itself that sometimes are surrounded by a light yellow halo effect.

**Staphylococcosis**

All avian species are susceptible to staphylococcal infections, and *Staphylococcus aureus* is the most common cause of disease. An often observed form of infection is a lesion that appears as an inflammation of the skin of the foot or pododermatitis, that is commonly referred to as “bumblefoot” (Fig. 12.5). Staphylococcal bacteria are ubiquitous, normal inhabitants of the skin and mucous membranes, and the bacteria require a break in those protective layers for infection to occur. Captive birds are more commonly found infected than free-ranging birds. Abrasions from rough surfaces where birds perch or stand may contribute to the occurrence of this disease. Studies in Spain with free-ranging imperial eagles demonstrated that staphylococcal infection can be transferred from humans to chicks being handled for banding. Infection was common in nestlings handled without latex gloves, whereas infection was rare in those birds handled with gloves. Mallard and redhead duck, bald and golden eagle, and ferruginous hawk have been among the species submitted to the National Wildlife Health Center (NWHC) that have been diagnosed with this condition.

Septicemic staphylococcosis or staphylococcal blood poisoning can also occur, generally in birds that are immuno-compromised or whose immune systems are not fully functioning. These types of infection can result in sudden death. Lesions associated with this form of infection generally consist of congestion of internal organs, including the liver, spleen, kidneys, and lungs, accompanied by areas of tissue death (Fig. 12.6). Bald eagles, American kestrels, red-tailed hawks, a duck, a mute swan, and herring and ring-billed gulls are among the species submitted to the NWHC for which septicemic staphylococcal infections have been diagnosed.

*S. aureus* can also cause serious disease in humans both as a wound infection and as a source of food poisoning. Good sanitation procedures should always be followed when han-
dling animals, and protective gloves should be worn when handling wildlife found dead.

**Tularemia**

Tularemia is primarily a disease of mammals, but natural infections by *Francisella tularensis* have caused die-offs of ruffed grouse and other grouse species. A variety of avian species have been found to be susceptible to infection as a result of serological surveys that have detected antibody against tularemia, experimental studies to determine susceptibility, and by cause-of-death assessments for birds submitted for necropsy (Table 12.1). The strains of *F. tularensis* that caused natural infection of ruffed grouse are of low virulence for humans despite ruffed grouse becoming infected by the same tick (*Haemaphysalis leporispaulstris*) that causes highly virulent tularemia in snowshoe hare.

Ticks are the primary source for disease transmission in natural cases of tularemia in upland gamebirds such as grouse and pheasants; ingestion of diseased birds and rodents is the primary source of disease transmission to raptors, gulls, and other scavenger species. Tularemia is infrequently reported as a cause of disease in wild birds. Ruffed grouse in northern climates have been the primary focus for reports in the scientific literature. The primary lesion seen is multiple, discrete spots scattered throughout the liver tissue (Fig. 12.7).

**Table 12.1** Avian species reported to be susceptible to infection by *Francisella tularensis*.

<table>
<thead>
<tr>
<th>Upland game species</th>
<th>Other birds</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ruffed grouse</td>
<td>Gulls and terns</td>
</tr>
<tr>
<td>Sharp-tailed grouse</td>
<td>Raptors (such as hawks and eagles)</td>
</tr>
<tr>
<td>Sage grouse</td>
<td>Scavengers (such as shrikes)</td>
</tr>
<tr>
<td>Ptarmigan</td>
<td></td>
</tr>
<tr>
<td>Blue grouse</td>
<td>Ducks and geese</td>
</tr>
<tr>
<td>Bobwhite quail</td>
<td></td>
</tr>
<tr>
<td>Pheasant</td>
<td></td>
</tr>
</tbody>
</table>

**Figure 12.7** The numerous, small, yellow and white spots on the liver of this beaver that died of tularemia are similar to the appearance of liver lesions in ruffed grouse.
Other

Colibacillosis, which is caused by infection with *Escherichia coli*, is one of several additional bacterial diseases occasionally encountered in wild birds. Avian strains of *E. coli* are generally not considered important causes of infection for humans or species other than birds. *E. coli* is a common inhabitant of the intestinal tract, but it often infects the respiratory tracts of birds, usually in conjunction with infection by other pathogens. These infections result in disease of the air sacs, and the infections are referred to as chronic respiratory disease. Lesions commonly associated with this disease include pericarditis or inflammation of the transparent membrane that encloses the heart and perihepatitis or inflammation of the peritoneal covering of the liver. These conditions make the coverings of the heart and liver look like a white or yellow mass that somewhat resembles the icing of a cake (Fig. 12.8). The livers of infected birds often appear swollen, dark in color, and may be bile stained (Fig. 12.9). Unhygienic hatcheries and other areas where young waterfowl and gamebirds are being held are often heavily contaminated with *E. coli*, and this results in infections causing acute mortality.

Figure 12.8  (A) Pericarditis and perihepatitis in a bird with colibacillosis. (B) Infection results in the liver being encased in a translucent covering.
Figure 12.9  Swollen bile-stained liver in a bird with colibacillosis.

Similar to the other sections of this Manual, the bacterial diseases discussed are not comprehensive of diseases of wild birds. The similarities in clinical signs and gross lesions displayed in illustrations in this section emphasize the need for cause-of-death evaluations by qualified animal disease laboratories. Also, the environmental persistence and human health impacts noted for some of these pathogens emphasize the need to consider personal and environmental protection when handling dead birds. Assumptions that the cause of death is due to a pathogen of minor importance could have serious consequences if highly virulent infections are involved.

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Supplementary Reading