Chapter 9

Salmonellosis

Synonyms

Salmonellosis; paratyphoid; bacillary white diarrhea (a synonym for pullorum disease); pullorum disease[1], fowl typhoid[1]

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 lifelong 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 fertilizer 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.*

Characteristic	***Salmonella pullorum***	***Salmonella gallinarum***	***Salmonella typhimurium***
Common name	Pullorum disease	Fowl typhoid	Salmonellosis
Natural hosts	Chickens (primary), turkeys	Chickens, turkeys	Wide range of vertebrates; not restricted to birds.
Age susceptibility	Mortality usually confined to the first 2–3 weeks of age.	Generally infects growing and adult birds; disease also infects young due to egg transmission.	All ages affected; more common in young and often in association with concurrent disease agents.
Transmission	Infected hatching eggs followed by spread from infected chicks to uninfected chicks that hatch.	Infected carrier birds most important; egg transmission of secondary importance.	Contaminated environment resulting in ingestion through food and water; egg transmission can also occur.
Relative occurrence in wild birds	Rare in free-ranging species; not maintained within wild populations.	Uncommon in free-ranging species; not maintained within wild populations.	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.
Other naturally infected avian species	Ducks, coots, pheasants, partridges, guinea fowl, sparrows, European bullfinch, magpies, canaries, hawk-headed parrot.	Ducks, swans, curlews, pheasants, quail, partridge grouse, guinea fowl, peafowl, wood pigeon, ring dove, rock dove, owls, rooks, jackdaws, sparrows, blackbirds, goldfinches,ostrich, parrots.	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.
Current geographic occurrence	Rare in most advanced poultry-producing areas.	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.	Worldwide due to wide range of species infected.
Relative human health significance	Occasional infections following massive exposure (contaminated food); prompt recovery without treatment.	Rare and of little public health significance.	One of the most common causes of food-borne disease in humans.

Table 9.2 *Pathways for transmission of* ***Salmonella*** *sp. in birds.*

Type of transmission	Means	Consequences/processes
Vertical (from parent to offspring)	Through contaminanted eggs from infected female; embryo may be infected or surface of egg becomes infected as it passes down oviduct.	Infection of hatchlings at age of greatest susceptibility. Infected hatchlings become source of infection for other hatchlings.
Horizontal	Bird-to-bird contact	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.
	Contaminated environments	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.
	Contaminated feeds	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.
	Inapparent infections	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.

Table 9.3 *Examples of reported environmental persistence for* ***Salmonella*** *sp. in different substrates. [—, no data available.]*

Substrate	Temperature				Serovar
	11 °C	25 °C	38 °C	Ambient	
Poultry feed	18 months	16 months	40 days	—	***S. typhimurium***
Poultry litter	18 months	18 months	13 days	—	***S. typhimurium***
Soil from vacated turkey pens	—	—	—	6–7 months	Unspecified paratyphoid form
Urban garden soil	—	—	—	280 days	***S. typhimurium***
Hatchery fluff	—	—	—	5 years	Unspecified paratyphoid form
Avian feces	—	—	—	28 months	Unspecified paratyphoid form
Reptilian feces	—	—	—	30 months	Unspecified paratyphoid form
Manure	—	—	—	36 months	Unspecified paratyphoid form

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.

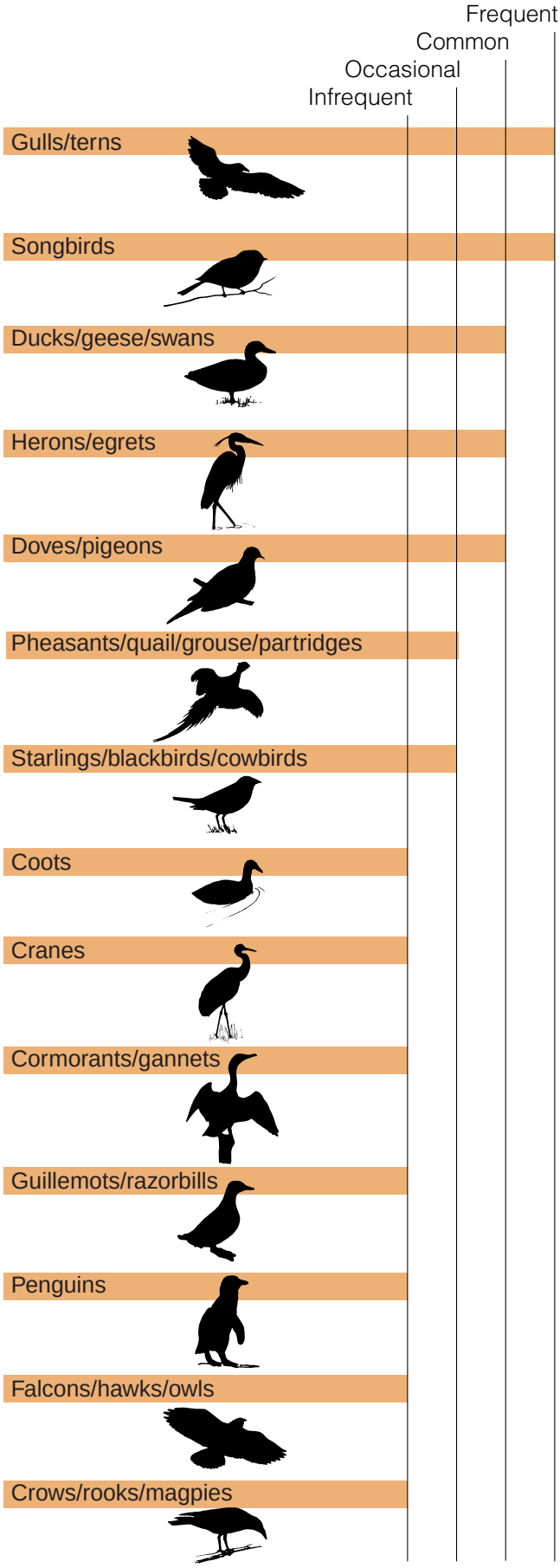


Figure 9.1 *Relative rates of isolation of* ***Salmonella*** *sp. in free-ranging wild birds.*

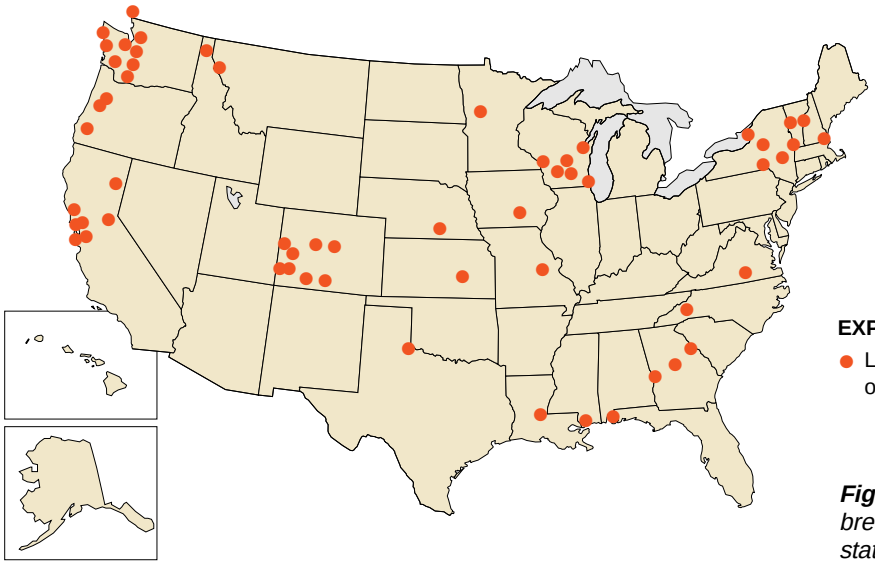


Figure 9.2 *Locations of reported outbreaks of salmonellosis at birdfeeding stations within the United States. (From National Wildlife Health Center database.)*

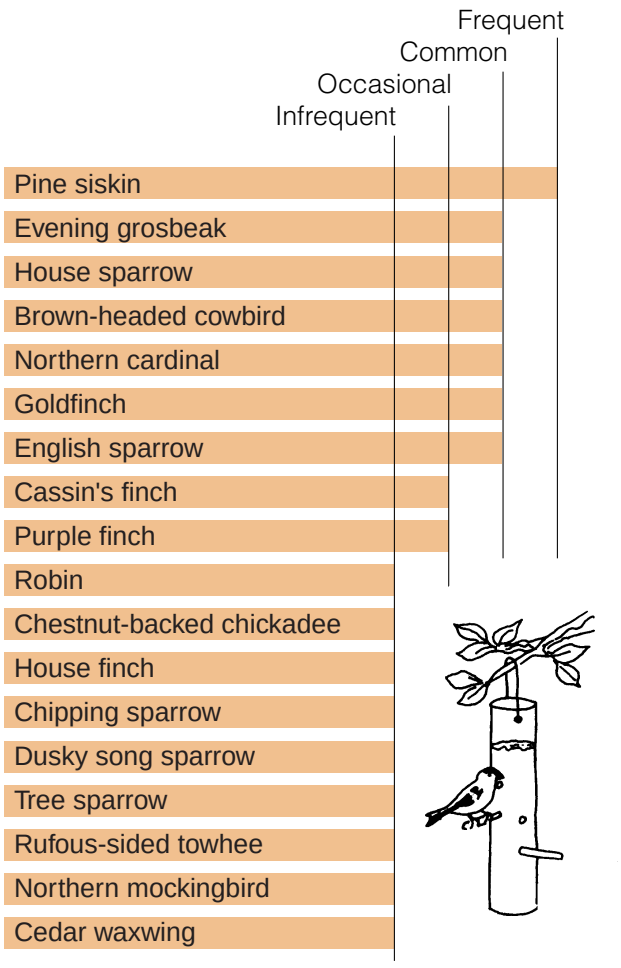


Figure 9.3 *Relative occurrence of species found dead from salmonellosis outbreaks at birdfeeding stations within the United States.*

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 be 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.

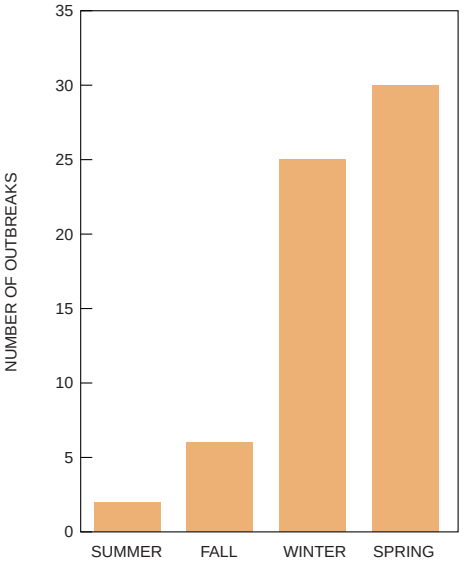


Figure 9.4 *Seasonal occurrence of salmonellosis outbreaks at birdfeeding stations within the United States.*

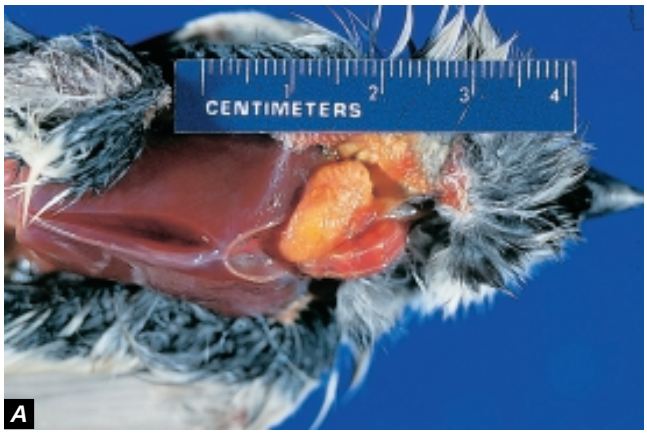
Photos by Milton Friend

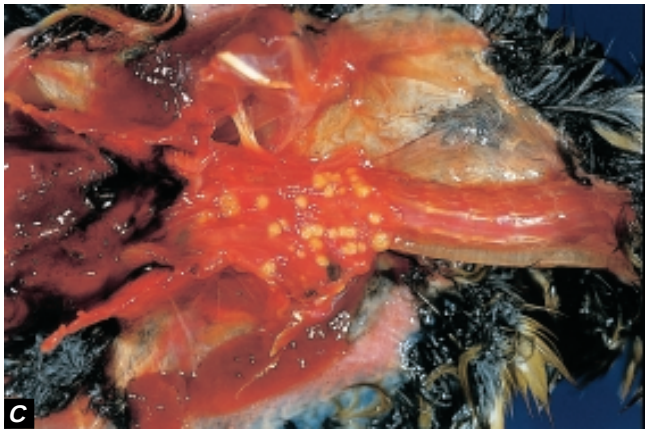
Figure 9.5 ***(A)*** *Salmonellosis can cause large-scale losses of colonial nesting birds.* ***(B)*** *Young birds are especially vulnerable.*

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-





Photos by J. Christian Franson

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.*

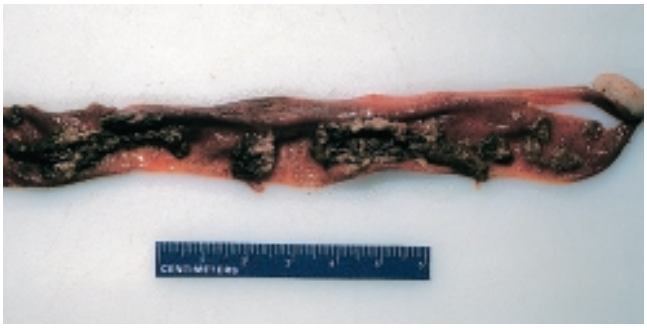


Photo by James Runningen

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. enteriditis* 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. enteriditis* 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 inapparent infections developing into systemic clinical salmonellosis that may jeopardize the well-being of the infected bird and of other birds within the facil-

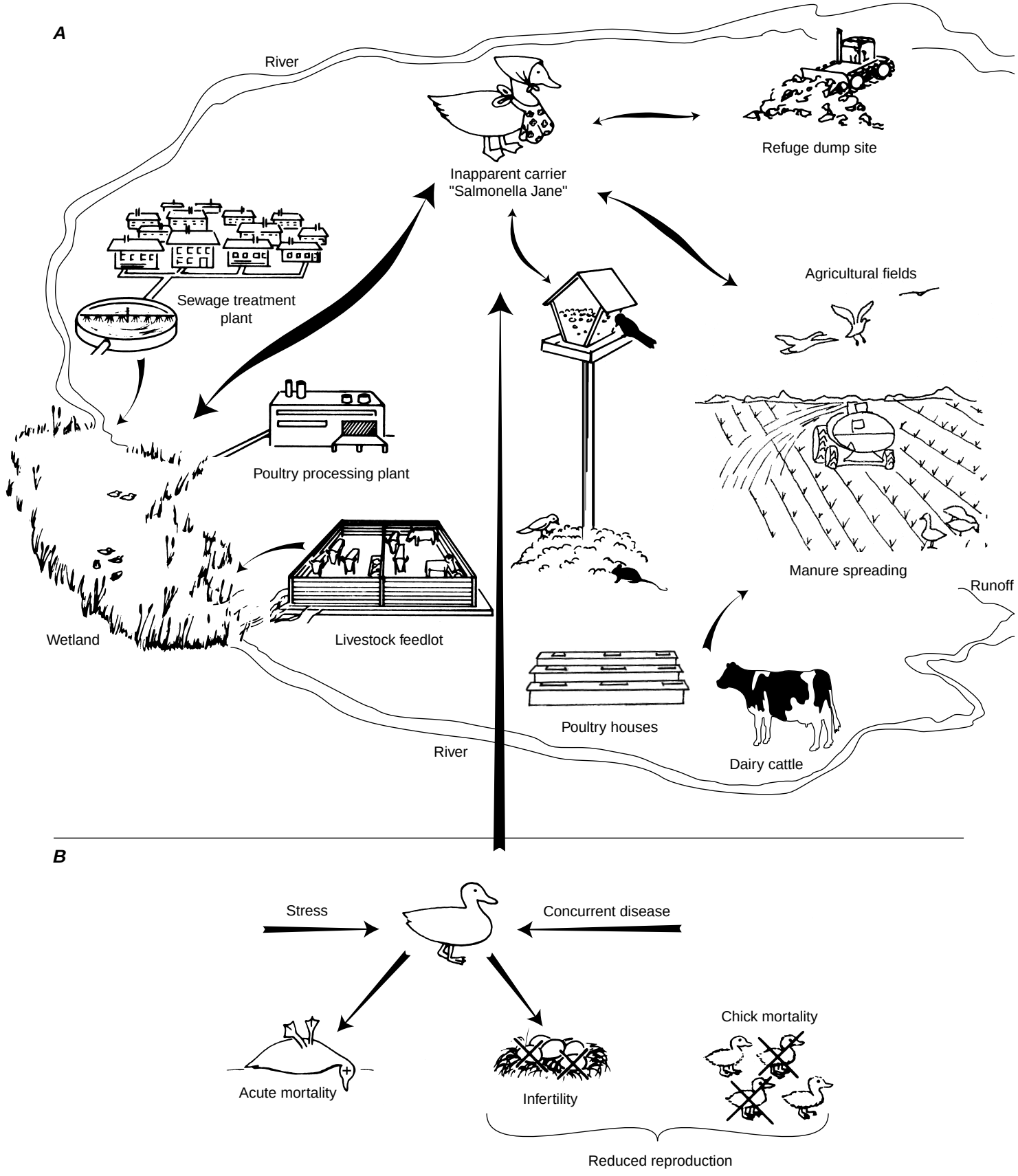


Figure 9.8 ***(A)*** *Sources and* ***(B)*** *consequences of salmonellosis in wild birds.*

ity. 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.

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

Supplementary Reading

Gast, R.K., 1997, Paratyphoid infections, *in* Calnek, B.W., and others., eds., Diseases of poultry (10th ed.): Ames, Iowa, Iowa State University Press, p. 97–121.

Snoeyenbos, G.H., 1994, Avian salmonellosis, *in* Beran, G.W., and Steele, J.H., eds., Handbook of zoonoses (2d ed., Section A): Bacterial, rickettsial, chlamydial, and mycotic: Boca Raton, Fla., CRC Press, p. 303–310.

Steele, J.H., and M.M. Galton, 1971, Salmonellosis, *in* Davis, J.W., and others, eds., Infectious and parasitic diseases of wild birds: Ames, Iowa, Iowa State University Press, p. 51–58.