

KILLING WITH KINDNESS: BIRD FEEDER-ASSOCIATED DISEASES OF WILD BIRDS

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Birding is the one of the leading recreational past times in the United States. In addition to the popularity of bird viewing and species identification, millions of people actively and purposefully attract wild birds to their homes and yards with bird feeders. Commonly encountered species at bird feeders are primarily passerines, and those that are resident in an area may use feeders year-round, especially in winter when weather is harsh and other sources of food are difficult to access. Types of feeders vary from the common tube-shaped seed or nectar feeders, to platform-type structures that hang or sit on a post or on the ground. Feeders are stocked with sunflower seeds, millet, cracked corn, thistle, peanuts, sugar solution, or suet.

Bird feeders can be an important source of nutrition for birds, especially during abnormal or harsh climate or foraging conditions, yet they are man-made constructs and therefore an artificial habitat, promoting repeated congregation of multiple species of birds around a point source of food. Not only may this result in unnatural densities of birds at a given point in time, it can also lead to interactions among and between species that would normally not occur.

BIRD FEEDER-ASSOCIATED DISEASES

As a consequence, one of the impacts of the presence and use of bird feeders is their role in the transmission of disease among wild birds. Birds gathering at this common food source come into direct contact with one another in ways, or at a frequency, that would not otherwise occur in nature. Similarly, bird feeders contaminated with infectious particles or food-borne toxins enable direct and indirect contact between affected and naïve individuals, facilitating disease transmission.

While there are many infectious diseases of wild birds that may be clinically evident in birds visiting feeders, and other infectious diseases for which commingling at feeders may facilitate transmission (e.g. influenza), there are several diseases particularly associated with feeders, because the incidence and prevalence of these diseases are greatly enhanced by birds' use of feeders.

Salmonellosis

First detailed reports of the disease in wild birds were published in the 1960s, although the disease had been well known in domestic poultry since the 1800s. *Salmonella* organisms can survive for a long time in the environment in soil and in water (including salt water). *Salmonella typhimurium* DT (definitive phage type) 40 and 160 are the strains most commonly associated with 1822

mortality in North American songbirds like siskins and finches (*Carduelis* and *Carpodacus* spp), and these and other phage types have become adapted to some songbirds species that use feeders. Songbirds vary in their susceptibility to *Salmonella* infection, partly as a function of their feeding habits.

As enteric bacteria, the Salmonellae are primarily transmitted by the fecal-oral route. *Salmonella* infections are largely picked up from contaminated environments, and in fact, the bacteria is more prevalent in the intestinal tracts of wild birds living in areas where there are higher densities of people and livestock creating refuse and manure and thereby contaminating soil and water.

Infected birds are usually inapparent carriers, and will clear infections within a few weeks, shedding organisms in their feces for a short period of time during infection. Bird feeders act as mechanical vectors for the pathogen, becoming contaminated by an infected bird. Clinically affected birds show signs of acute septicemia: dyspnea, weakness, ataxia, shivering, swollen eyelids, ocular and nasal discharge, and diarrhea. Birds may die within 24 hours, may recover to become inapparent carriers, or may localize the infection to particular organs or sites (eyes, joints, air sacs). Pathogenesis involves invasion of the gastrointestinal epithelium (in the esophagus and crop of songbirds, in particular), infection of local lymphoid tissue, dissemination via circulation, and then residency in macrophages in the liver, spleen and other organs, where the organism is relatively protected from the humoral immune response.

Gross necropsy may reveal congested, swollen organs with hemorrhagic or necrotic foci, fibrinopurulent inflammation of membranes, and hypopyon. In songbirds, the crop and esophagus may exhibit multiple areas of mucosal necrosis and fibrinopurulent inflammation; ingluvitis and esophagitis may be the only evident lesion. Diagnosis is confirmed by culture (on blood or MacConkey's agar at 37°C for 24 hours); isolates can be serotyped at reference laboratories, and if possible, phage-typed for epidemiologic analysis.

Treatment during outbreaks should be aimed at reducing incidence of infection in the population. Treatment of individuals can be accomplished with antibiotics, although antibiotic-resistant strains are common. Oral therapy of inapparent carriers has proven to be ineffective in eliminating shedding.

The only known cases of salmonellosis in people acquired from wild birds have been associated with outbreaks at feeders (and not from contact with gulls or other wild species). Feeder-associated outbreaks of salmonellosis are an indicator that environmental load has increased to a level that may pose a risk to domestic animals and humans.

Mycoplasmosis

This disease emerged in wild songbirds (primarily house finches) in winter 1994 in the mid-Atlantic. The disease spread to the entire Eastern population of house finches within a few years, including in Quebec and

Ontario provinces in Canada, and into the Midwest. House finch populations in the Eastern US declined dramatically with the advent of this disease. The disease was first described in house finches in their native Western range in Montana in 2002.

Molecular analysis of *Mycoplasma gallisepticum* isolates revealed that this is a novel strain of the pathogen, unrelated to poultry strains, and its origins remain undetermined. Since first being identified, the pathogen has also been isolated from other songbird species with conjunctivitis, including American goldfinches, purple finches, pine grosbeaks, and evening grosbeaks. The organism is fragile in the environment, although it can survive for short periods in dust, litter, and feathers, making mechanical transfer possible.

Transmission in songbirds is horizontal via direct contact or aerosolization of droplets; vertical transmission has not been demonstrated in songbirds as it has been in poultry, although transmission from infected adults to nestlings has been reported. In songbirds, it appears to be highly contagious, and its impacts more severe in high-density populations. The disease peaks in the southeast in late summer and early fall, and in winter in the northeast.

Clinical signs include severe eyelid swelling, conjunctivitis, and oculonasal discharge that dries to become crusts. Lesions impair vision and may cause total blindness; some birds present with generalized debilitation and depression and weight loss. The organism attaches to epithelial cells of the respiratory tract, causing cell damage as well as eliciting a host inflammatory response that contributes to clinical disease. Histologically, chronic lymphoplasmacytic inflammation of the ocular mucosa and submucosa and upper respiratory tract is characteristic, accompanied in some cases by keratitis and/or tracheitis. Asymptomatic or chronic infections are possible with all mycoplasmas, which can change surface antigens and thereby immunoregulate their host's response.

Diagnosis can be accomplished by serology (serum plate agglutination), and/or confirmed by culture. The organism is fastidious, requiring selective media and then plating, with optimal growth at 37°C (Frey's medium with swine serum is used most commonly). Differential diagnoses in songbirds include bacterial infections, head trauma, or avian poxvirus.

Treatment of infected individuals can be accomplished with tetracycline or tylosin: infected house finches treated with enrofloxacin and ophthalmic gentamicin showed resolution of clinical signs but remained culture or polymerase chain reaction (PCR) positive for *M. gallisepticum* for months after treatment. That said, rehabilitators are urged not to bring infected birds into their facilities because of the risk posed to other birds; as well, there is a risk of releasing wild birds that have been treated for *M. gallisepticum* because they may be inapparent carriers.

Songbird mycoplasmosis does not appear to be zoonotic, and to date, transmission of the songbird strain

of *M. gallisepticum* to domestic poultry has not been documented.

Avian Pox

Avian pox virus is one of the oldest-known diseases of birds, and has been documented in nearly 10,000 avian species. Characterized by proliferative lesions on the skin on the head, legs or toes, or mucous membranes of the mouth and upper respiratory tract, it is transmitted primarily by direct contact with the virus on abraded skin. It can also be transmitted, however, via biting insects, or by contact with exfoliated scabs and other fomites on contaminated surfaces like bird feeders. Poxvirus is extremely hardy in the environment and can remain viable on man-made surfaces for long periods of time.

Lesions are usually readily apparent and fairly typical: discrete wart-like lesions on unfeathered parts, or necrotic lesions in the mouth or upper respiratory tract. Differential diagnoses include candidiasis, capillariasis, trichomoniasis, or ectoparasites (mites) or bacterial infections. Histologically, the virus causes epithelial proliferation (hypertrophy, hyperplasia) and infected epithelial cells stack to form the characteristic pox lesion. Intracytoplasmic inclusions (Bollinger bodies) can be seen on hematoxylin and eosin (H&E)-stained histosections. Diagnosis is confirmed via virus isolation in chicken eggs, or PCR.

There are several strains of the virus, which vary in their virulence across species. Birds that recover from infections are usually immune for life. While clinical disease is self-limiting, recovery is prolonged, and lesions can be quite debilitating for the individual bird. Treatment is aimed at reducing or removing lesions and sterilizing the area to kill virus and minimize risk of secondary bacterial infection, and providing supportive care.

Trichomoniasis

Trichomonas gallinae is a protozoal parasite that invades the mucosa of the upper gastrointestinal tract, causing proliferative caseous lesions, or "cankers," in the oral cavity and upper gastrointestinal tract that can interfere with feeding and breathing. While columbiformes and raptors are considered most susceptible, the disease has been diagnosed in several passerines species as well, and is associated with heavily used, contaminated bird feeders. Transmission is by ingestion of organisms in contaminated food or water. Suspect birds with clinical evidence of disease can be screened using wet mounts, and the diagnosis confirmed with culture and PCR. The disease is not zoonotic.

Mycotoxiosis

Considered to be primarily a disease of waterfowl, toxin-producing micro-organisms in moldy grains, cereals, and seeds can lead to disease in birds visiting feeders. Molds produce mycotoxins such as aflatoxin (produced by *Aspergillus* species and one of the most well-studied mycotoxins) under the right environmental

conditions. While aflatoxin levels in poultry feeds are regulated, studies of mycotoxin levels in commercially-available bird seeds reveal significant levels in some batches of feed. Mycotoxicosis can be clinically inapparent to severe (peracute death), and acute (typically liver failure) to chronic (poor weight gain, immunologic or reproductive disruption, carcinogenesis). Clinical signs of acute aflatoxicosis include apparent blindness, weakness, inability to fly or stand, or unconsciousness. The effects of low-dose chronic exposure to mycotoxins may have more impact on wild birds than acute exposure causing epizootics of severe disease.

PREVENTING DISEASE TRANSMISSION AT BIRD FEEDERS

Keeping bird feeders clean and using fresh food, unused portions of which are changed out regularly, are keys to preventing disease transmission at bird feeders. Feeders should be cleaned at least once a week with 10% bleach solutions. The ground under and around

feeders should be swept to remove droppings and unused feed. Water in drinkers or baths should be changed daily.

The type of feeder used has implications for disease transmission as well. Tube feeders that do not accumulate bird droppings the way that platform feeders do may be beneficial in this regard, although studies have shown that tube feeders promote direct contact between birds competing for the limited access points.

To prevent zoonotic potential, people should wear rubber gloves when cleaning. Keeping cats indoors during feeder-associated outbreaks is important for the health of the cats and their owners. During outbreaks of mycoplasmosis or salmonellosis, people should be urged to stop using bird feeders until the epidemic resolves.

RECOMMENDED READING

1. Thomas NJ, Hunter DB, Atkinson CT (eds): *Infectious Diseases of Wild Birds*. Ames, IA: Blackwell Publishing; 2007, 484 pp.