Diseases of birds - how and why some birds die

A propaedeutic survey of the most prevalent diseases afflicting North American birds

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Diseases, and the parasites of birds, many of which cause diseases, are always present in wild birds and are often widespread. Some exist within or on a bird almost indefinitely without killing it—the disease organisms may lack virulence or an opportunity to spread when the bird population is low. At other times, under certain ecological conditions, and with large concentrations of birds, the disease agents may cause sudden detectable illness, followed by death among vast numbers, for example from botulism among wild waterfowl.

A normal bird in relatively good health can, or often does, support a large number of parasites. In 1959, Austin L. Rand (Chic. Nat. Hist. Mus. Bull. 30) pointed out that a city pigeon, flying or walking about, is not a single organism, but may have an active community of parasites living on and in it. It is a living zoo that may be inhabited by at least 71 or more different kinds of animals and plants—among the external parasites, 2 kinds of ticks, 8 of mites, 1 fly, 1 bug, and 6 lice, and of internal parasites, 9 roundworms, 18 tapeworms, 3 flukes, at least 8 blood protozoans. Pathologist Robert M. Stabler, has listed for me a dozen or more kinds of protozoans one might find in a pigeon: 2 fungi, 9 bacteria and 4 viruses which can cause infectious diseases. Viruses are not living things as bacteria are. (They cannot make their own enzymes and proteins.) Diseases—those particular forms of destructive processes in the body—are generally caused in birds by their internal parasites which in this discussion, owing to limitations of space, shall be limited to a brief survey of some of the better known or most spectacular diseases caused by bacteria, fungi, and viruses, some of which are communicable to man.

SOME BACTERIAL DISEASES

Bacteria can cause destruction of birds in two ways (1) by producing toxins and (2) by destruction of a bird's tissues. Perhaps the best-known bacterial disease of wild birds is botulism, a form of food poisoning, in which the bacterial organism, Clostridium botulinum type C, a natural inhabitant of the soil, which thrives on decaying plants and dead animals, produces a toxin highly poisonous to birds. Wild ducks and pheasants are especially affected (paralyzed) by it. (The lack of reports of type C botulism in people suggests that man may be fairly resistant, especially in comparison with types A, B, and E.)

Botulism was first reported in wild ducks in the western United States as early as 1876 and was originally called "western duck sickness" but it occurs throughout North America and has been reported in the Old World. In 1910 the tremendous mortality among ducks and other birds around the Great Salt Lake first attracted wide attention to the disease in North America; in 1925 an estimated million birds, mostly waterfowl, died of botulism at a lake in Oregon; 1-3 million died at the Great Salt Lake in 1929, and hundreds of...
thousands periodically elsewhere. The greatest waterfowl mortality in North America has been at the breeding grounds in the western United States and western provinces of Canada and the incidence is greatest in warm weather.

A typical sequence in an outbreak starts with Clostridium botulinum type C in the soil. In years of heavy rains or melting snows, excess water floods agricultural lands below the hills and mountains, making broad shallow expanses with large amounts of underlying decaying organic matter, with an accompanying rapid growth of C. botulinum. The high temperatures of July and August quickly warm the shallow waters and underlying ooze. A large flight of ducks moves into the area, begins feeding on the underwater plants and invertebrates. Within a few days, the ducks lose their power of sustained flight, and as the poison progresses, they cannot fly or walk; their neck muscles become so weakened that they lie with their heads and necks on the ground or in the water. They die then, either from predation, drowning, respiratory failure, dehydration, or exposure. Even the rare Peregrine Falcon has been affected; some of those that fed on afflicted ducks in Malheur and Bear River National Wildlife Refuges prior to 1950 were found stricken with botulism.

Although there is some disagreement about how the birds get the toxin, many investigators believe that ducks get it from eating submerged infected aquatic insects or crustaceans, from submerged grain or other plants containing the toxin, and from maggots (larvae of blowflies) in the decomposing bodies of birds that have died of the poison.

DIVING DUCKS, owing to their preference for deep waters, are ordinarily less affected; the duck most involved is the Pintail, followed by the Greent Winged Teal, which are the most numerous species during the "botulism season" of late summer in the Bear River region of Utah. Others affected in lower numbers are Mallard, Gadwall, Blue-winged Teal, Cinnamon Teal, American Wigeon, Northern Shoveler, Redhead, and Ruddy Duck. Grebes, geese, mergansers, and coots suffer less from botulism than the dabbling ducks. Great Blue Herons, American Bitterns, and gulls are affected, and shorebirds may sometimes suffer large losses to botulism. Shorebirds most stricken are the American Avocet, Black-necked Stilt, and occasionally, the Least Sandpiper. Common Loons, grebes, ducks, gulls and other birds have been noted on the shores of the Great Lakes, stricken with type E botulism. About 7700 loons and gulls died in 1963 and 4900 in 1964.

On some of its western wildlife refuges, the U.S. Fish and Wildlife Service, after years of research, has developed methods of combating environmental conditions favorable to botulism. The Service has built levees and dikes, and has cut channels, all for control of water movement and prevention of shallow flooded areas, and with considerable success in saving waterfowl and other birds from large losses to botulism.

T HE NOTORIOUS DISEASE, "Parrot fever," psittacosis, or ornithosis, now called chlamydiosis (klah-mid-ih-oh'sis) was attributed until recently to a virus, but the disease-causing organism is now classified as a bacterium. According to one authority, chlamydiosis has become an occupational disease among breeders of pigeons, poultry farmers, and employees of poultry processing plants. It may also infect persons who band birds, and those who own and handle parrots. Fortunately, human mortality from the disease has been reduced by use of the wide-spectrum antimicrobial drugs, especially tetracycline.

Investigators who took serological tests of 34 bands of wild birds in Connecticut, New York, New Jersey, and Pennsylvania, reported in 1957 that 33 tested were negative for ornithosis (chlamydiosis) and the single infected bander was a youth with little experience in banding birds who might have picked up the disease elsewhere. The investigators concluded that most species of native wild North American birds are not a hazard to man or a reservoir for the disease.

The bacteria can be transmitted from a bird to man in three ways (1) from inhaling airborne bacteria (2) by ingesting bacteria from a bird's excreta or from its nasal discharges when handling sick or latently infected birds, and (3) through skin-piercing bites of contaminated birds.

Of all known hosts, the Rock Dove is the most common and consistent source of infection, but even though it is a potential reservoir of human infection there is little evidence to date, according to two eminent authorities on the disease, that there is transmission of chlamydiosis from pigeons to wild birds.

Infections, worldwide, have been reported in 139 species of wild birds, which, in North America, include the Great Egret, Snowy Egret, Glossy Ibis, Ring-necked Pheasant, Willet, Sandpiper, Laughing Gull, Gull-billed Tern, Common Tern, Least Tern, Royal Tern, Black Skimmer, Ringed Turtle Dove, tattie, and the Painted Bunting. Another authority includes among infected North American species, the Northern Fulmar, Herring Gull, Black-billed Magpie, Starling, and House Sparrow, and among cagebirds, besides members of the parrot family, ricebirds, finches, and canaries, which are highly susceptible to chlamydiosis, and after having been affected, die within a short time.

Signs of acute infection with symptoms of severity, developing rapidly in individual birds, start with an exudate from the bird's eyes or nostrils, a lack of appetite, and inaction, followed by diarrhea with gray or bloody feces. Apparently little can be done for infected free-living wild birds but treatment of captives with antibiotics in their food and water, and repeated injections has helped reduce them as reservoirs of infection for wild birds. Recent evidence suggests that intrafl ock transmission of chlamydiosis bacteria may be through nestmates and bird lice.

A VIAN CHOLERA, also called fowl cholera and avian pasteurellosis, is not caused by a bacterial poisoning as in botulism but by a bacterial infection in which the tissues of a bird are invaded and destroyed by the bacterium, Pasteurella multocida. Domestic ducks, geese, chickens, turkeys, and fowl of all kinds are susceptible, also pigeons, sparrows and other wild birds that visit poultry yards. In North America, avian cholera can be epizootic in wild birds and it attacks largely swans, geese, ducks, coots, and gulls. It has also been reported in birds of prey in zoos and in pheasants and quail in captivity. Marsh Hawks, Short-eared Owls, meadow mice, and a weasel were infected in an outbreak in California in 1958; the bacterium has also been isolated from the Common Flicker, Starling, an American Robin, and Common Grackle.

In the 1940s and 1950s severe outbreaks of avian cholera caused great mortality among waterfowl and other birds. An estimated 40,000 swans, geese, ducks, coots, and some shorebirds died
of the disease in the San Francisco Bay area of California in the winter of 1948-1949. More than 60,000 waterfowl perished of the disease in the Muleshoe National Wildlife Refuge in Texas during the winter of 1956-1957.

In January 1964 more than 1100 Lesser Snow Geese and "Blue" Geese died of avian cholera at Squaw Creek National Wildlife Refuge, Missouri in one night; in the winter of 1965-1966, an outbreak of the disease over thousands of square miles of California, from Salton Sea north to Tule Lake near the Oregon border, destroyed more than 70,000 birds. Waterfowl losses from the disease fluctuate however, and in some years in Muleshoe Refuge, for example, only a few sick or dead birds are noted.

Although avian cholera breaks out regularly on the Pacific coast of North America and elsewhere, it was reported on the Atlantic coast for the first time in June 1963 on Goose Island, Maine where more than 70% of the nesting Common Eider females died. No males were found dead because they had apparently moved out of the area to their molting grounds. Their summer molt usually precedes that of the nesting females. In the same area Herring and Great Black-backed gulls also were found dead from the same infection.

According to two authorities on the disease, the bacteria of avian cholera are in both the nasal and anal excretions of infected birds. When waterfowl are crowded together, the infected birds can quickly contaminate the environment. Sick birds are rarely seen during an outbreak of the disease but dead ones may lie about among the living, healthy-appearing waterfowl; the few that recover become chronic carriers of the disease, in which it may continue over a long time, or is characterized by slowly progressing symptoms. A specialist in studies of avian cholera reported that infections of Pasteurella multocida have been noted in man but were usually local and the source of the infections had generally not be determined.

Avian tuberculosis, rarely reported in wild birds, is a contagious disease known worldwide among domestic chickens. It is dissimilar to tuberculosis in man, which is also dissimilar to that of cattle. Avian tuberculosis is caused by a living bacterial parasite,—the tubercle bacillus, Mycobacterium avium, also known as Mycobacterium tuberculosis. It is most frequent in the North Temperate Zone, is present in most European countries and in the United States. All species of birds are susceptible and swans, geese, ducks, turkeys, and peacocks are infected more frequently than wild birds.

In tuberculosis of birds, the bacilli attack the liver and spleen more than the lungs and commonly enter the body through the alimentary tract. Among wild birds, pheasants seem markedly susceptible and the disease has been reported in the House Sparrow. Most infected birds are emaciated and some develop skin growths around the eyes, wing joints, or on the legs.

Avian tuberculosis has been reported in North America in the American Kestrel, Barn Owl, Common Crow, Common Raven, and Brown-headed Cowbird, and one investigator reported that two American Avocets in California died from the disease. Avian tuberculosis has also been observed in wild Whistling and Trumpeter swans, Mallard, Pintail, Green-winged Teal, American Wigeon, Northern Shoveler, Redhead, Ruffed Grouse, a Starling, and a Swainson's Thrush.

Some authorities believe that the tubercle bacilli in wild birds may come from close contact at feeding places with infected poultry, and that predatory birds may contract it from their prey, for example, Golden Eagles in Scotland that have preyed on Wood Pigeons infected with tuberculosis. Gulls in Europe have been infected from the effluence in sewers emptying into rivers where gulls gather in large numbers.

Salmonellosis (sal-moh-neh-LOW-sis), also called paratyphoid infections, bacillary white diarrhea, and fowl typhoid, is a disease caused by bacteria of the genus Salmonella. All bacteria in the genus are considered to be potential pathogens for man and other animals, however, some members of the Salmonella differ widely in their host adaptations and in the signs and symptoms in the host. They usually occur as intestinal infections which may result in enteritis, diarrhea, and terminates in septicemia and death in most animals. According to one authority, the reported incidence of Salmonella infections in the general wild bird population is extremely low, therefore wild birds are not a great threat as a natural reservoir and sources of infection for man and/or his domestic animals.

In Europe, an investigator reported in 1968 that he had known of previous infections only in pigeons by the bacterium, Salmonella typhimurium, but in the bitter cold winter of 1967-1968, near Lausanne, Switzerland, he observed three cases of Salmonellosis in House Sparrows, one in a Grasshopper Warbler, Locustella naevia, six in the Brambling, Fringilla montifringilla, three in the Bullfinch, Pyrrhula pyrrhula, and fourteen in the Greenfinch, Chloris chloris. He speculated that the disease was transmitted from infected to uninfected birds when large groups of them assembled to feed.

The discovery of the incidence of Salmonella infections in birds in continent-
al Europe in 1968, was paralleled in the United States by a group of investigators who reported in 1966 the incidence of 12 birds infected by *Salmonella typhimurium* in Massachusetts and in Rhode Island. Most of the victims were Brownheaded Cowbirds, although Herring Gulls, House Sparrows, and White-throated Sparrows were also infected.

As long as birds are not subjected to stress from crowding, for example, when feeding, little harm results, but when food for them is scattered on the ground and attracts large numbers, hundreds may die of the *Salmonella* infection. To prevent the ground from heavy contamination by the droppings of infected birds, seeds should be placed in feeders above the ground.

In Marion County, Florida from 1971 through December 1973, birds died from Salmonellosis at the feeding station of Mrs. Howard Pearl near Salt Springs. The incidents, reported in the *Florida Field Naturalist*, involved infected Ground Doves, Blue Jays, Tufted Titmice, Brown Thrashers, House Sparrows, Red-winged Blackbirds, Common Grackles, Cardinals, Chipping Sparrows, and White-throated Sparrows. The heaviest mortality of birds was in late winter and early spring, and many of them had subcutaneous lesions (morbid changes in diseased or injured parts) in the pectoral (breast) region. Cultures taken from the birds with lesions were positive for *Salmonella typhimurium*.

By March 10, 1974, all species of birds that had been infected earlier at Mrs. Pearl’s feeders were devoid of any lesions and were otherwise in good condition, and cultures from them were negative for *Salmonella*; it was thought that the decline of the infection among the remaining birds had resulted from the die-off of infected birds. For prevention, it was recommended, as a matter of course, that all those who feed birds regularly should avoid feeding them on the ground, and should occasionally clean and thoroughly disinfect the above-ground feeders.

### SOME FUNGAL DISEASES

**Aspergillosis** (as-per-jill-OH’-sis), a locally common or chronic infection usually fatal to birds, is caused by a fungus, or mold, *Aspergillus fumigatus*. The fungus grows in damp or wet bird seeds and in the hulls of straws and other residues in bird-seed mixtures when they are exposed to dampness and open air; also in the litter used in mew houses where falconers keep their birds of prey, and in birds’ nesting materials.

Birds usually breathe in the spores of this mold while they are feeding, and the fungus lodges in their lungs and air sacs, eventually causing avian pneumonia and bronchitis. Among propagators of game birds it is called “brooder pneumonia.” The fungus spores are widely distributed in nature, and ground-feeding birds pick it up through contaminated feed and litter. It is a serious disease of wild birds, especially of waterfowl and game birds and those brought into captivity. It is often fatal to birds of prey kept by falconers; it has created some problems in keeping penguins in zoos and for Whooping Cranes kept at the Patuxent Wildlife Research Center, Laurel, Maryland.

Some of the wild birds afflicted are loons, swans, geese, ducks, hawks, Bald Eagles, grouse, quails, owls, gulls, ravens, crows, thrushes, House Sparrows, grackles, cowbirds, and juncos; at least 48 species, either wild or captive, have been reported to be hosts to aspergillosis. The disease progresses through stages. In the beginning, afflicted birds gasp and wheeze, then begin to mope or to sit about with their feathers fluffed. Among seed-eating birds, crowded at feeders, the sick ones may continue to feed and to stand feebly in the bird feeder while doing so. One of the last signs of the illness may be severe diarrhea, after which individual birds afflicted usually fall over and die. There seems to be no cure for wild birds suffering from aspergillosis.

The best remedy is prevention: to protect birds at feeding stations by buying only clean bird seed and to be sure that the mixed grains and seeds in the feeders are not allowed to get moldy. If one has more than one bird feeder or bird bath in the garden, their use should be rotated...
so as to allow them to be periodically scrubbed and disinfected with a disinfectant like Lysol before putting them back into use. If an increasing number of birds show signs of illness, feeding should be stopped for at least a week or two until all of the afflicted birds have died or moved elsewhere. This must be done even in cold, severe winter if the lives of many healthy birds are to be protected.

Histoplasmosis (HISS-toh-plaz-MOH'-sis) is a respiratory disease of man caused by the airborne spores of a fungus, Histoplasma capsulatum. It is not a fungal disease of birds, nor are they carriers of the fungus, but soil enriched with the feces of Starlings, Red-winged Blackbirds, and grackles, for reasons not fully known, give the fungus a competitive growth advantage over the myriads of other soil fungi. The fungus grows especially in the droppings under the winter roost of enormous numbers of blackbirds. After the soil inhabited by Histoplasma capsulatum dries, the spores are scattered by air currents, grow again in suitable soil or infect persons who happen to inhale the spores.

In 90% of people infected, the disease is benign; they show no apparent symptoms but the remaining 10% develop what appear to be colds and other viral respiratory ailments; most of these clear up spontaneously. Persons with severe infections require hospitalization and treatment with a few antifungal agents of which amphotericin B seems the best.

SOME VIRAL DISEASES

Viruses are submicroscopic infectious agents and are capable of rapid multiplication only in living cells. Encephalitis (en-sef-ah-LIE'-tis) viruses (arboviruses) cause diseases that include equine encephalomyelitis which also infects man, birds, small mammals, and reptiles. Arbovirus (arthropod-borne virus) gets its name from its carriers—arthropods such as mosquitoes and ticks.

Eastern encephalitis was first reported in birds in 1938 from a sick Ring-necked Pheasant in Massachusetts and the virus disease was also isolated from a sick Rock Dove during the 1938 epidemic, which involved both man and horses. Two different viruses which cause encephalomyelitis in horses in North America, because of their geographic distribution, are called respectively, eastern encephalitis virus (EEV) and western encephalitis virus (WEV). The eastern form ranges in North America from Wisconsin south to Texas and east to the Atlantic coast where it is particularly prevalent; the western form until 1953 was thought to be limited in North America mostly west of the Mississippi River but isolations of WEV have been made from House Sparrows and Brown-headed Cowbirds in New Jersey. Besides EEV and WEV, another arbovirus in the United States is St. Louis encephalitis virus which, according to Dr. Harald Norlin Johnson, an authority on arboviruses, has also been found in wild birds. The virus was so-named because it was first isolated during an epidemic of encephalitis in St. Louis in 1933. Others of this group of arboviruses, with different names, are endemic in other parts of the world.

Although eastern encephalitis virus causes a severe and usually fatal disease in people and horses, there is little evidence that it is harmful to native North American birds. However, it occurs in introduced species which may be good sentinel hosts for detecting its presence. Fatal infections of EEV have now been reported in domestic white Peking ducklings, Ring-necked Pheasants, Chukar Partridges, and House Sparrows. In some parts of New Jersey it is considered impractical to try to raise pheasants without prophylactic vaccination of them against EEV. According to authorities, antiencephalitic vaccines have been developed for the protection of man and horses and mules.

The arbovirus is a tiny, intracellular parasite, 20-200 microns in diameter. In nature, mosquitoes and ticks receive the virus when they suck the blood of a bird or other animal infected with an arbovirus, and they can then infect the blood of other susceptible animals when they bite them. Thus the infected birds for example, do not spread the virus caused disease directly but are reservoirs or carriers of arboviruses.

EEV is transmitted by several species of mosquitoes and when EEV successfully invades the central nervous system of infected birds, they become lethargic, show incoordination and paralysis, and adopt abnormal postures of the head and neck, with tremors, head retraction and circling.

Control (destruction) of native wild birds that may possibly become infected and serve as reservoirs for the disease, and hosts for causing infection among mosquitoes and other blood-sucking arthropods, is difficult and impractical. In the words of an authority on the disease, destroying birds for these reasons "could not be defended as sound conservation practice." Furthermore, many other animals besides birds are reservoirs for the encephalitis viruses.

Avian pox, sometimes called bird pox, foot pox, fowl pox, "foot disease," contagious epithelioma, and avian diptheria, is well-known to bird banders. It is caused by a virus that is confined to birds. The disease produces warty protuberances on the feet or head of a bird and also infects its throat and