New Dimensions in Diseases Affecting Waterfowl

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Introduction - We start off with light heart, but as we near the marsh, we stop abruptly in shock and horror. The shoreline, where only last evening we saw thousands of sleek, apparently healthy birds, is now littered with their bodies. Most of them are ducks, but here and there we see a Canada goose, a gull, an avocet, a black-necked stilt, a pelican.....

This is the way Jensen and Williams (1964) described an outbreak of avian botulism in a western marsh. It illustrates, more vividly than dry facts and body counts, the impact a disease can have on a population of waterfowl.

When we think about waterfowl diseases, we probably think first of botulism and then of lead poisoning, the result of birds swallowing spent lead shot. In fact, these are the only two diseases discussed by Kortright (1942) and Day (1949) in major books on North American waterfowl, attesting to both their importance and our years of experience with them. Botulism was probably an old problem in 1910, but the catastrophic loss that summer of millions of aquatic birds near the Great Salt Lake and in California marked the beginning of real concern about its impact on waterfowl populations (Jensen and Williams, 1964). Concern about lead poisoning of waterfowl began a few years later following the investigations of Wetmore (1919).

Today, more than 50 years after these early reports, botulism and lead poisoning are still major disease problems of North American waterfowl, but they are no longer the only ones. After years of study, we have learned much about how these two diseases occur and about their prevention and control. But now there are new pathogens and new toxic compounds that can afflict waterfowl populations. So rather than redescribe the established disease problems of the past and present, I will concentrate on some of these new wate-fowl disease problems of the present and future.

Philosophy - Before we can discuss either old or new diseases of waterfowl, we need to agree what "disease" means. I will use the term to mean physiological malfunction, not only that produced by pathogenic organisms, but from any cause. This concept of disease is not revolutionary. Perhaps Aldo Leopold, in the thirties, described it best: "game disease may have as its causative agent various living organisms, various nutritional deficiencies, chemical poisons, mechanical injuries, or combinations of these"(Leopold, 1933).

By any definition, disease problems seem to be increasing among North American wildlife. For one thing, a number of new pathogens have been identified during the past 35 years. Some of this increase may be explained away as a simple result of improved diagnostic technology and increased scientific activity, increasing the probability of discovery. But for waterfowl at least, some of the increase is probably real. Changes in movement and activity patterns and a greater use of artificially propagated birds have increased the contact between wild and domestic waterfowl in some areas. As waterfowl habitat continues to shrink under the pressure of more intensive agriculture and an expanding human population, this interchange will intensify and must eventually result in wild waterfowl sharing more of the diseases of domestic waterfowl. Another important factor in disease ecology is the steadily in-

creasing contamination of the environment with pesticides and other toxic chemicals. These not only act directly as poisons, but can interact with each other and disease organisms to produce additional, unexpected detrimental effects.

Given these conditions, it is not surprising that new waterfowl disease problems have appeared and more are expected. I cannot, of course, discuss them all, but I will try to illustrate the size and complexity of the challenge they pose by three examples: Duck virus enteritis (duck plague) as a new problem, avian influenza as a future problem, and chemical-microbial interactions as a perspective.

DVE (Duck Plague);History and Distribution - DVE (duck virus enteritis), or duck plague, as it is usually called outside the United States, is an acute, contagious, and often fatal herpesvirus infection of ducks, geese, and swans. DVE has been a disease of domestic waterfowl in the Netherlands for more than 50 years (Baudet, 1923), although it was not specifically recognized until 1942 (Box, 1942) and the virus that causes it was not identified until 1949 (Jansen and Kunst, 1949). This disease has also occurred in domestic waterfowl in Belgium, India, and England, and is suspected to have occurred in France and China (Table 1).

The first known outbreak of DVE in North America occurred during January 1967 in the concentrated White Pekin duck-producing area of Long Island, New York (Leibovitz and Hwang, 1968a). Unlike outbreaks reported from Europe and Asia, this one involved not only domestic flocks but wild waterfowl (Leibovitz, 1968; Leibovitz and Hwang, 1968a,b). The first positive case in wild birds was a wild mute swan (*Cygnus olor*) found dead on January 30, 1967, in the marshes opposite the duck farm where the outbreak started; additional cases were detected in captive waterfowl nearly 5 months later in muscovy ducks (*Cairina moschata*) being raised by an aviculturist about 40 miles away (Leibovitz and Hwang, 1968a).

Despite control efforts by the U.S. Department of Agriculture and other agencies, DVE persisted in the White Pekin flocks on Long Island until November 1968 (Walker et al., 1969). Apparently, free-flying wild waterfowl became infected through exposure to infected White Pekins in fall 1967, and in November 1967 there was an outbreak in the Flanders Bay area of Long Island, about 2 miles from a known infected White Pekin duck farm (Leibovitz, 1968). The Flanders Bay outbreak lasted about 30 days (November 5 - December 5) and killed approximately 5% each of the estimated 1,500 black ducks (*Anas rubripes*) and 375 mallards (*Anas platyrhynchos*) present on the bay. In addition, DVE was found in a Canada goose (*Branta canadensis*) and a bufflehead (*Bucephala albeola*) on the bay and in a few black ducks, mallards, and a greater scaup (*Aytha marila*) at other locations on Long Island as late as January 1968 (Leibovitz, 1968). Since then, DVE has appeared sporadically in domestic and captive waterfowl on Long Island, upstate New York, Pennsylvania, and Maryland (Walker et al., 1969, and USDA records). Therefore, it should probably now be considered as enzootic in this general geographic area.

In April 1972, DVE appeared in the far west. In San Francisco, California, captive birds began dying in a 6-acre lagoon near the Palace of Fine Arts (Snyder et al., 1973). By June, the entire resident population of 46 muscovy ducks and smaller numbers of other waterfowl species had died. Examination of tissues from the muscovies confirmed DVE.

At this point, DVE outbreaks in wild waterfowl species had involved small numbers of birds, many of them captive. But in January 1973, a severe outbreak began among a large population of free-flying wild birds in a major flyway. Wintering waterfowl began dying in large numbers at the Lake Andes National Wildlife Re-

fuge in southeastern South Dakota. At the peak of the outbreak, daily mortality exceeded 1,000 birds. Despite the best efforts of control teams, more than 40% of the 100,000 mallards and 3% of the 9,000 Canada geese wintering on the refuge had died by the time the outbreak ended in late March (Friend and Pearson, 1974; Friend, Pearson, and Berlinger, in manuscript) and every waterfowl species on the Refuge had suffered some mortality. Of the 60,000 survivors that left Lake Andes when spring migration began, approximately 30% has been exposed to DVE and were potential carriers of the virus.

So far, there has not been a second catastrophic outbreak in wild waterfowl, but a small one has occurred in a significant location. In April 1973, DVE erupted among black ducks in a commercial game farm in Wisconsin (Jacobsen et al., in manuscript). Thus, within 6 years after its first discovery in North America, DVE has spread to wild waterfowl in all four of the continent's major flyways.

Species Susceptibility - All species of ducks, geese, and swans should be considered susceptible to DVE until more is known about this disease. Experimental studies by Van Dorssen and Kunst (1955) indicated that European teal (*Anas crecca*) and pintails (*Anas acuta*) did not die when exposed to DVE, but two drake pintail-mallard hybrids were found dead of DVE during the Lake Andes outbreak (Friend, Pearson, and Berlinger, in manuscript).

Impact - In early DVE outbreaks in the Netherlands, mortality was high, nearly 100% in many flocks; since 1959, it has dropped somewhat but is still substantial (Jansen, 1968). In the United States, total mortality during outbreaks in domestic ducks has been highly variable, ranging from 5% to 100% (Leibovitz, 1972). It has also been variable in the two major outbreaks in wild waterfowl. Losses of mallards and black ducks on Flanders Bay were approximately 5% of the populations at risk; losses of mallards at Lake Andes were more than 40%.

Another potential source of waterfowl losses from DVE is a drop in egg production. This has not been assessed for wild birds, but during DVE outbreaks among Long Island White Pekin flocks, egg production by mature breeders dropped 25-40% (Walker et al., 1969).

Control - By now, any hopes of eradicating DVE from the United States are probably unrealistic. However, its impact on wild waterfowl can be greatly reduced if new outbreaks are detected early and reported promptly, and if waterfowl managers take every precaution to minimize the potential for outbreaks on their areas.

To prevent DVE, the environment of susceptible birds must be kept free of the virus (Leibovitz, 1972). However, DVE virus is probably not maintained for prolonged periods in physical elements such as soil, water, or air, or in other vertebrate or invertebrate animals, but within the waterfowl themselves. Therefore, a priority need is to identify DVE infections in captive and domestic waterfowl and eliminate these foci of infection by destroying infected flocks and disinfecting their surroundings. To accomplish this, regulatory agencies will have to cooperate closely and will need the full support of sportsmen's groups, game breeders, aviculturists, and the domestic duck industry. In addition, whenever possible waterfowl managers should eliminate direct or indirect contact between domestic, captive, and wild waterfowl by (1) keeping these three types of birds from using common areas, particularly common swimming or drinking ponds, (2) discouraging employees of waterfowl management areas and game farms from keeping domestic waterfowl flocks, and (3) prohibiting the use of common shipping crates, vehicles, incubators, feeders, waterers, and other equipment used to rear or move waterfowl.

A second source of infection is DVE carriers among our wild waterfowl. For the present, we must assume that this virus acts

like other herpesviruses and that survivors may carry it in latent form until they are "stressed" and become capable of transmitting it. Therefore, waterfowl, especially exotic species, must not be moved from one area to another without proper precautions. Before either wild-trapped or farm-reared birds are introduced, their serum should be tested and certified as negative for DVE, and they should be held in isolation from resident flocks for at least 30 days. Presently, no mechanisms are available to carry out either of these actions, but they must be developed. Each year tens of thousands of game-farm waterfowl are released on shooting preserves, for retriever trials, and in waterfowl stocking programs, and few if any precautions are taken to prevent the introduction of DVE or other diseases along with these birds. The time is long overdue for waterfowl managers to correct this situation.

DVE can be transmitted by direct contact between infected and susceptible birds or indirectly through contamination of a common environment such as a shallow pothole. Stagnant or slow-moving, shallow bodies of water contaminated with the virus were believed to be an important vehicle for the transmission of DVE in domestic duck outbreaks and the Lake Andes outbreak. Obviously, waterfowl density is also important. Although DVE outbreaks have occurrec during every season of the year, they are usually associated with inclement weather, which may stress carrier birds enough for them to begin transmitting virus. Thus, late winter is probably the most hazardous time for DVE in most waterfowl areas because the risk of an outbreak increases with increasing concentrations of waterfowl per unit area, with increasing lengths of time they are concentrated, with decreasing water area available to the birds, and with increasing frequency of inclement weather. Waterfowl managers should be aware of these contributing factors and clearly should try to maintain the highest possible environmental quality on their management areas, particularly in winter.

Finally, waterfowl managers can assist greatly be becoming familiar with the signs of DVE and notifying the proper authorities immediately if they suspect this disease on their area. Color brochures describing how to recognize DVE are available for both domestic ducks (U.S. Department of Agriculture, 1969) and wild waterfowl (Friend and Pearson, 1973).

Avian Influenza - If little is known about DVE in wild waterfowl, even less is known about avian influenza. I know of no outbreaks of avian influenza among wild waterfowl, but viruses of this group have recently been isolated from free-flying birds, and the group almost certainly contains future pathogens of our waterfowl populations.

History and Distribution - Avian influenza viruses are of the same species as those causing influenza in man. Most human influenza epidemics have high infection rates but low mortality rates, leaving many survivors. But epidemics keep on occurring because "new" influenza strains appear periodically. Occasionally, the appearance of a highly virulent strain causes catastrophic losses of life. For example, some 20 million people died during the 1918-1919 influenza pandemic. The situation appears to be similar with avian influenza. Most of the strains identified, though highly infective, produce little mortality. But there is at least one exception--fowl plague virus, now known to be one of the most highly pathogenic members of the type-A influenza viruses.

Fowl plague virus has been reported in many countries throughout the world (Easterday and Tumova, 1972) and may be enzootic in areas of the Middle and Far East (DeLay et al., 1967). Fowl plague has not been reported in the United States since 1929 (Easterday and Tumove, 1972), but a large number of other influenze viruses have recently been found in birds, especially since 1963. Begin-

ning in 1956, influenza viruses have been isolated from chickens in Scotland and Italy; turkeys in England, Canada, and the United States; pigeons in Germany; captive quail (Coturnix coturnix japonica), pheasants (Phasianus colchicus), and chukar partridge (Alectoris chukar) in Italy; and domestic ducks in Italy, England, Yugoslavia, Germany, the United States, and the USSR (Easterday and Tumova, 1972). More recently, influenze type-A viruses have been isolated from a variety of captive exotic birds in the United States (Slemons et al., 1973a,b).

The first identification of an influenza virus in free-flying wild birds was made during a sudden mass die-off of common terns (Sterna hirundo) in South Africa in 1961 (Rowan, 1962; Becker, 1966). The outbreak was similar to human influenza epidemics: observations along a 1,000-mile stretch of coast showed that the birds were affected virtually simutaneously and were either dead or immune within a matter of days. The second isolation of an influenza virus in wild birds was made in 1971 from a shearwater (Puffinus pacificus chlororhynchus) nesting off the Austrailian east coast (Downie and Laver, 1973). Even though influenza has never been reported in Australian domestic birds (Downie and Laver, 1973), recent serum samples from shearwaters and other Australian pelagic birds have contained high levels of influenza antibodies similar to those produced in man during the 1957 influenza pandemic (Dasen and Laver, 1970; Laver and Webster, 1974).

Influenza in Waterfowl - Direct evidence of influenza in wild waterfowl was first found in 1966, when antibodies were detected in serum samples from Canada geese in Missouri, Wisconsin, Michigan, and South Dakota and from snow geese (Chen hyperborea) in Missouri (Easterday et al., 1968). In a 4-year survey, 1966-1969, Winkler et al., (1972) detected influenza antibodies in 4.7% of 1,401 Canada geese from eight different waterfowl refuges. The percentage of reactors increased each year, particularly in the two refuges with non-migratory flocks (Winkler et al., 1972).

More recently, influenza viruses themselves have been isolated from free-flying wild waterfowl in both southern California (Slemons et al., 1974) and the east coast (Rosenberger et al., 1974). In California study, 41 type-A influenza virus isolates were recovered from nine species of ducks killed by hunters between October 6 and December 3, 1972 (Slemons et al.,1974). This large number of both virus isolates and duck species (Table 2) provides strong evidence that ducks are involved in the natural history of type-A influenza viruses.

The presence of many strains of influenza virus circulating simultaneously in a bird population should provide suitable conditions for genetic recombinations, which is believed to be the source of "new" strains. Thus, there is the possibility of highly virulent strains like fowl plague virus evolving from this increasing pocl of genetic material. Perhaps of equal concern is the popular theory that new strains of human influenza may be derived by genetic recombination from influenza viruses infecting lower mammals or birds, and that various bird populations might actually serve as a "breeding ground" for these viruses (Downie and Laver, 1973; Easterday, 1973; Easterday and Tumova, 1972; Slemons et al., 1974).

Impact - It is not known whether influenza is new in waterfowl or whether the recent evidence of it simply reflects increased scientific efforts and better detection techniques. As Slemons et al. (1974) pointed out, to isolate influenza viruses from wild birds, the birds must be available for sampling and the samples must be taken while they are shedding recoverable amounts of infectious virus. Since these conditions are most often encountered during obvious outbreaks, we can probably assume that influenza

viruses are even more widespread in waterfowl than the records indicate.

Although high mortality rates are probably an exception in avian influenza outbreaks, the 1961 tern die-off and the mass die-offs characteristic of fowl plague outbreaks suggest what could happen to waterfowl populations if a highly pathogenic strain appeared. Besides mortality, there is the possibility of impaired reproduction. In domestic turkeys, at least, a sudden and marked drop in egg laying is characteristic of influenza infections, and some survivors never completely recover normal egg production (Easterday, 1973).

Perspective - Whether or not avian influenza viruses have been pathogens of wild waterfowl in the past, they are certainly potential pathogens of the future. As with DVE, mortality and reduced egg production are two probable results.

Since influenza viruses require close contact for transmission (Easterday and Tumova, 1972), their spread undoubtedly depends on waterfowl density. The apparent increase in influenza virus activity in wild waterfowl populations during the past 2 years may well reflect new waterfowl movement and activity patterns that are more conducive to transmission of these viruses. Therefore, it is imperative that we learn as much as possible of the distribution and types of avian influenza viruses in waterfowl and other migratory birds, determine the effects of sublethal infections in waterfowl, and gain a better understanding of the interrelationships between different type-A influenza isolates and the transmission of influenza viruses in nature.

Chemical-Microbial Interactions - Like viruses, toxic chemicals can cause a disease state. Others in this symposium will address the direct impact of chemicals on waterfowl survival and reproduction. But in nature, birds are rarely exposed just to chemicals alone. I want to briefly discuss what may happen when they are exposed both to chemicals (pesticides, lead shot, etc.) and to potentially pathogenic organisms (Bacteria, viruses, fungi, etc.).

Interactions between disease-causing agents is not a new concept, but these interactions are so complex that they are seldom studied and almost never well documented. Like man, waterfowl are exposed to a vast range of parasites, from viruses and bacteria to worms and mites, and to a wide variety of chemical pollutants. Because many of the chemicals are new in the environment, the mammal or bird may not have totally adequate body defense mechanisms for handling them. One possible result is that chemicals, in amounts too small to be noticeably toxic, can nevertheless interfere with well-established host-parasite relationships and result in clinical disease.

Examples - One interaction I have repeatedly observed is a high incidence of aspergillosis in lead-poisoned Canada geese. Aspergillosis is caused by the fungus *Aspergillus fumigatus*. Although its spores are common practically everywhere, adult birds rarely exhibit the disease unless they receive a massive exposure, as from moldy grain or hay, or they undergo prolonged physiologic stress (O'Meara and Witter, 1971). Apparently, ingested lead provides enough stress for some geese to develop aspergillosis. Once their spore densities reach high enough levels, the infected birds may then infect others in the flock.

Aspergillosis and lead poisoning are old problems. With recent experimental work, we can now add interactions between disease-causing agents and modern pesticides. In one series of studies, exposure to sublethal levels of DDT, dieldrin, and a polychlorinated biphenyl (three of our most common environmental pollutants) dramatically increased the susceptibility of mallards to duck virus hepatitis (Friend and Trainer, 1970, 1974a,b). In another series,

sublethal levels of parathion significantly impaired the function of the salt glands in mallards (Friend and Abel, 1975; Verhage et al., 1975). These glands, which protect the body's vital osmotic balance by excreting excess salt, must work properly if waterfowl are to survive in marine or alkaline environments. Their impairment by pesticides is not only a threat in itself; since salt-gland malfunction can contribute to losses from avian botulism (Cooch, 1964), impairment could result in another detrimental chemical-microbial interaction.

Indirect evidence for a pesticide-microbial interaction already occurring under natural conditions is provided by recent records of avian cholera (pasteurellosis) in wild waterfowl. This disease, again an old probelm in domestic birds, is caused by the bacterium Pasteurella multicida. It was first identified in wild waterfowl at Lake Nakuru, Kenya, in 1940 (Hudson, 1959) and in North American waterfowl at the Muleshoe National Wildlife Refuge in Texas in 1944 (Quortrup et al., 1946). Avian cholera has since become established as a constant problem of wild waterfowl in both the Texas panhandle and the San Francisco Bay area of California. No other areas share this dubious distinction. Since agricultural chemicals are used intensively in both of these general areas, it has been postulated that exposure of waterfowl to these chemicals results in carrier birds "breaking" with the disease or becoming more susceptible to the bacteria (Friend and Trainer, 1970).

Perspective - The use of chemical agents in habitat manipulation has become nearly as widespread as their use in agriculture. Waterfowl managers need to consider the subtle effects of these chemicals wherever and whenever they are to be used. Those investigating disease outbreaks in waterfowl should not stop when they have found one causative agent but should consider the possibility of multiple contributing factors. In short, we must learn to consider the full spectrum of disease-causing agents and their complex interrelationships if we hope to control existing disease problems and prevent new ones.

Prognosis - Present - We all know that environmental quality can be important to health. Crowding of people or animals facilitates the spread of contagious and infectious diseases and creates stresses that can lead to the onset of latent diseases. Poor quality habitat results in scanty and often deficient food supplies that can lead to poor general health and nutritional diseases. Chemicals, even if they may not by themselves poison healthy individuals, can add further stress, disrupt body defense mechanisms, and alter well-established host-parasite relationships to bring on still further diseases.

All of these conditions are now affecting North American waterfowl as their habitat shrinks and its quality deteriorates.Too often the lands set aside for wildlife are those too poor for agriculture or undesirable for other uses. Too often the water that goes to wetlands for waterfowl has been used and reused many times and contains a variety of agricultural chemicals, including pesticides, when it reaches the birds. We should not be surprised that disease problems have increased. Perhaps we should be surprised, however, that little effort has been expended to properly identify these problems, and even less to solve them.

Advances in microbiology, immunology, virology, and chemical analysis have improved our capability to identify pathogens and harmful chemical agents that may have escaped detection in the past, and the medical and environmental sciences have accumulated an unparalleled body of knowledge to deal with such problems. We have been slow in applying these advances to the broad area of wildlife disease. Like the village in "Fiddler on the Roof", we have "Tradition", and it takes us time to change our traditional

ways of approaching problems. What I'm afraid of is that we do not have the time.

Future - The variety and complexity of diseases affecting North American waterfowl is already staggering. And it can be expected to keep on growing. Established diseases of domestic waterfowl are probably next on the list, but "new" pathogens, chemicals, and nutritional diseases will also appear.

We do not have to be overwhelmed and defeated by this growing threat to our waterfowl resources. If we accept the idea that we can do something about it, our management approaches will change. A general increased awareness of waterfowl disease will result in better surveillance and early detection of disease problems. Improving technology will provide greater capability for detecting disease probelms and effectively dealing with them. Vaccination of waterfowl will become feasible and will be applied both to prevent the introduction of diseases and to limit the impact of outbreaks. Birds will not be introduced into new areas until they have been tested and certified as free of serious infectious diseases. Finally, managers will be aware of how diseases are related to waterfowl density, activity and movement patterns, species diversity, food supplies, and chemicals, and will regulate these factors to limit the potential for disease outbreaks and to help contain outbreaks when they occur.

This is not a wild dream. Much of the necessary technology is already available, and new techniques are perfected every day. Whether we use our growing knowledge to protect waterfowl depends on our attitude. If we go on believing that wildlife disease is a natural phenomenon, an act of God over which we have no control, we have lost the battle. We can win only if we believe that disease can be not only controlled but prevented, and if we accept the responsibility for doing both.