

Disease Problems and Needs

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About a year ago I was appointed as the first Director of the newly established U.S. Fish and Wildlife Service's National Fish and Wildlife Health Laboratory (NFWHL). The primary orientation of the laboratory is disease problems of waterfowl and, therefore, it was both a pleasure, and appropriate that my first presentation after assuming that position was to address the First International Waterfowl Symposium. At that time I indulged in a largely philosophical discussion titled, New Dimensions in Diseases Affecting Waterfowl (Friend, 1975). Principal subject areas of that presentation were duck plague (duck virus enteritis or DVE), avian influenza, and chemical-microbial interactions. During the past year, personnel of the NFWHL have been involved in one or more important field situations dealing with each of these problems. Today then, I will briefly describe the manifestation of last year's philosophical considerations in terms of this year's disease problems, and the actions taken.

The Need for Waterfowl Disease Studies

I am often presented with the questions of why should waterfowl diseases be studied and what can be done about these problems anyhow. It is not my intent today to directly answer these questions in any detail. However, I hope this presentation will leave you with two impressions. First, waterfowl losses from disease are far greater than are generally appreciated, and unspectacular daily losses take the greatest toll of waterfowl, not spectacular epizootics. Second, prevention of problems is the most effective way of combating them, but when disease epizootics do occur, prompt, aggressive, and responsible control activities can save thousands of waterfowl that might otherwise be needlessly lost.

To appreciate the impact disease can have on waterfowl populations, one need only recall the loss from duck plague of more than 40% of the 100,000 mallards (Anas platyrhynchos) wintering at the Lake Andes National Wildlife Refuge in 1973 (Friend and Pearson, 1974). Some of the major waterfowl losses from disease during 1975 are shown in Table 1, these losses only represent a small portion of the actual losses from disease during this period.

No good estimates of annual losses from disease are available, even for diseases such as botulism. However, annual waterfowl losses from causes other than hunting are approximately twice the 20 million waterfowl harvested by North American sportsmen. There is a strong need to identify and quantify the cause of these losses so that, where possible, appropriate measures can be initiated to reduce them. Table 2 provides data from the NFWHL on causes of mortality from two samples of birds assumed to be hunting season cripples. Note that only about one-half of these deaths were due to crippling losses. Other factors such as lead poisoning, avian cholera, and aspergillosis were also involved, but would have gone undetected because the numbers of dead birds observed in these samples were considered to be "normal hunting season losses." We are now selecting a number of locations for more intensive studies of this type to better define what

proportion of the "disease iceberg" is represented by major disease epizootics and what is the identity and magnitude of the causes contributing to the non-visible portion of the iceberg (Figure 1).

1975 Waterfowl Disease Problems

Duck Plague: The 1973 Lake Andes duck plague epizootic caused major concern among waterfowl resource managers, but interest in this disease has waned somewhat in the absence of similar waterfowl die-offs since that time. Those of us who helped contain duck plague at Lake Andes view the current situation with mixed emotions. We are rewarded by the feeling that the aggressive state-federal cooperative control effort carried out at that time is a major reason for the lack of duck plague activity in North American waterfowl. However, the specter of this disease continues to haunt those of us that experienced its devastation.

Unfortunately, duck plague is not a dead issue. This disease has been active during the past year, but in a manner that has not drawn excessive attention. New locations where duck plague occurred in 1975 include the National Zoo in Washington, D.C., a private holding in Louisiana, and at least two private holdings in Canada. In addition, a second epizootic of duck plague occurred at the Palace of Fine Arts in San Francisco in 1975.

All of these duck plague outbreaks involved captive waterfowl and that its appearance in free-flying wild waterfowl may be the result of exposure to the disease from contact with captive or captive-reared and released waterfowl (either domestic, exotic, or native wild species). Therefore, it becomes imperative that proper controls are exercised to prevent the accidental introduction of duck plague into wild waterfowl populations by this means. Dr. Burger will touch on this problem in his presentation, Propagation and Release of Mallards.

The first epizootic of duck plague at the Palace of Fine Arts lagoon in San Francisco occurred in April, 1972 (Snyder et al., 1973). Despite the status of duck plague at that time as an exotic disease, and the fact that this epizootic represented the first known occurrence of duck plague west of Pennsylvania, some of the birds were spared from destruction as a result of public outcry and political pressures. After a period of quarantine, these birds were used to restock the lagoon along with other birds. This action ignored two important considerations: first, quarantine does not cure duck plague, and second, duck plague, like many other herpesvirus infections, probably involves a "carrier" state. That is, birds that survive infection may transmit the virus at a later time to susceptible birds, thereby causing additional epizootics.

It is highly probable that the source of the April 1975 duck plague epizootic was one or more duck plague "carriers" that survived the 1972 epizootic. A number of these birds were identified by leg bands during the eradication of the lagoon's entire waterfowl population in 1975. The control effort was again interfered with by a well-meaning, but misinformed citizenry. The results could have been disastrous, but the resulting court hearing ruled in favor of destruction of these birds.

Chemical-Microbial Interactions: During last year's presentation I cited lead poisoning as a predisposing factor resulting in aspergillosis in Canada geese and the hypothesis that environmental pollutants may play a key role in the occurrence of avian cholera (Friend, 1975). During November 1975, I investigated a die-off of snow and blue geese (Anser caerulescens) on a small state waterfowl refuge in northeastern South Dakota. Laboratory analysis confirmed field observations that both lead poisoning and avian cholera were principal causes of this mortality. Of the 141 geese examined, 18% died of lead poisoning, 52% died of avian cholera, and 10% had both lead poisoning and avian cholera. The high percentage of lead poisoned birds in the sample suggests that lead may have been the "stress factor" that caused the avian cholera outbreak. Aspergillosis was also present in some of the dead geese. This aspect of the effects of lead shot has not received any study, but may represent a major interaction between a chemical compound, lead, and an infectious agent, Pasteurella multocida.

A strong state-federal cooperative control effort helped contain the avian cholera epizootic within the local site of the outbreak, a small state waterfowl refuge. Approximately 10,000 geese were represented in the population using the state refuge, while more than 100,000 susceptible geese were at risk at the Sand Lake National Wildlife Refuge, approximately 8 miles away. To contain the epizootic, it was necessary to initiate a temporary ban on hunting in the area surrounding the state refuge, undertake an intensive cleanup of dead carcasses on the infected area, thoroughly burn approximately 4 tons of geese, and finally disperse all birds from the infected area once the geese from the Sand Lake National Wildlife Refuge had departed that area. This highly successful operation resulted in the loss of approximately 2,500 geese (at least half of which were dead before the die-off was recognized and response initiated). Without the control efforts losses could have easily been 10 times as great within the immediate area, and new epizootics could have been created as the geese migrated south, moving from refuge to refuge.

Avian Influenza: During last year's presentation I indicated that direct evidence of influenza virus activity in wild waterfowl is of recent origin, and that the first virus isolations from wild waterfowl occurred in 1972 (Slemons et al., 1974). I also indicated that with the large amount of influenza virus activity appearing in wild waterfowl, it would only be a matter of time before genetic recombination of non-lethal strains might result in a lethal mutant that could kill large numbers of waterfowl. Very recently, a lethal strain of avian influenza did appear, but fortunately not in waterfowl. This epizootic occurred among chickens in Alabama and represents the first occurrence of a lethal influenza virus in chickens in the United States since 1929. I bring this to your attention because more than 50 percent of the thousands of chickens involved died. We are now one step closer to the possibility of a similar occurrence in waterfowl.

Personnel of the NFWHL have sampled waterfowl and blackbirds in the vicinity of this epizootic in Alabama and are undertaking cooperative studies with scientists at the University of Wisconsin to determine if a similar type of influenza virus is circulating among wild birds. It is imperative that we learn

much more about the role of waterfowl in the world distribution of these viruses and the hazards these viruses pose for our waterfowl populations. The urgency for this is dramatized by the recent influenza problem in chickens, and by the fact that scientists at the University of Wisconsin are isolating influenza viruses from 5% of wild ducks being sampled (Slemons and Easterday, 1976).

Conclusions

Two interrelated factors that contribute to the success or failure of dealing with a particular die-off are: (1) early diagnosis and initiation of adequate control activities, and (2) attitude towards disease problems. Die-offs caused by infectious agents build very rapidly, and without early diagnosis and rapid response to a die-off, the disease often spreads from a localized area where it can be easily contained, to a larger area where containment becomes much more difficult. Also, losses increase proportionately to the time required for the die-off to end or be controlled. This situation is often aggravated by apathy towards disease problems based on an acceptance of losses from disease being a natural phenomenon that cannot be controlled. However, this attitude should not be acceptable to either resource managers or sportsmen. Waterfowl diseases can be controlled and losses from disease can be prevented.

Disease problems will not be solved by rhetoric; they will only be solved if resource managers and sportsmen are willing to support the various activities and research necessary to deal with disease prevention and control. Sportsmen and field biologists can be of great assistance by reporting the occurrence of mortality when they observe it and not assume that cause X or Y was responsible. Reports can be made to either local, state or federal conservation personnel. Inquiries on waterfowl disease problems can also be made directly to the National Fish and Wildlife Health Laboratory.

TABLE 1. Some major waterfowl die-offs during 1975.

General location	Time of die-off	Approximate losses	Species lost	Principal cause
Back Bay, Virginia	February	25,000	coot & ducks	avian cholera
Phelps County, Nebraska	April	25,000	geese & ducks	avian cholera
Rice Lake, Minnesota	July-August	11,000	ducks	avian botulism
Bear River NWR, Utah	August-September	13,000	shorebirds, ducks, & geese	avian botulism
Renzienhausen Slough, South Dakota	November	2,500	snow geese	avian cholera, lead poisoning
Lake Procter, Texas	November	1,000	ducks	aflatoxicosis
Great Salt Lake, Utah	November	5,000	ducks, grebes, & gulls	erysipelas
Sacramento NWR, California	December	8,000*	ducks & geese	botulism, avian cholera
Sacramento-San Joaquin River Delta, California	December	5,000*	ducks, geese & swans	avian cholera

Note that these die-offs occurred from coast-to coast and throughout the year.

* Die-off has continued into 1976.

TABLE 2. Actual causes of mortality among "hunting season cripples".

Species	Sample size	Gunshot	Number of deaths due to:					Unknown but not gunshot
			Gunshot secondary infection	Aspergillosis	Avian cholera	Lead poisoning	Other disease conditions	
Canada geese	39	15	6	8	0	3	4	3
Snow and blue geese	24	11	0	0	4	9	0	0
Totals:								
Number	63	26	6	8	4	12	4	3
Percent	100	41	9	13	6	19	6	5