

PROGRESS REPORT: DUCK PLAGUE SURVEILLANCE OF AMERICAN ANSERIFORMES

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method used is direct blood film examination. This has value only when the infection is in a state of patency; latent infections often going unnoticed.

Manwell and Herman (1935. J. Parasitol., 21:415-416) and Herman et al. (1966. Avian Diseases, 10:541-547) have shown that much higher incidence of infection is observed when isodiagnosis is employed. This technique, however, is limited by the availability of susceptible hosts, time and personnel to handle the animals, and the need for extensive routine microscopic examinations.

Since the canvasback was an adult, it is impossible to know where it became infected. A comparison of the rate of infection in birds of the year moving south with that of birds returning north

would demonstrate whether the nesting grounds or wintering grounds was the site of transmission.

Apparently the parasite is well adapted to the duck host. Regardless of the level of parasitemia, no mortality was observed in Pekin ducks. Perhaps this is not true for young canvasbacks, but since studies of these young birds have not been carried out it is impossible to predict its pathogenic significance for the species.

I believe that this is the first report of a natural infection of *Plasmodium* in the canvasback duck.

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PROGRESS REPORT: DUCK PLAGUE SURVEIL-LANCE OF AMERICAN ANSERIFORMES

The first reported outbreak of duck plague on the American Continent occurred in a flock of White Pekin ducks in the concentrated duck-producing area of Long Island, New York, on January 3, 1967 (Leibovitz and Hwang, Proceedings of the 39th Annual Northeastern Conference on Avian Diseases, 1967). While some members of the order Anseriformes (ducks, geese and swans) have been shown to be experimentally susceptible to duck plague (Van Dorssen and Kunst, 1955. Tijd. Diergeneesk. 80: 1286), reports of natural infection have been limited to domestic ducks (Anas platyrhynchos domesticus), muscovy ducks (Cairina moschata) (Jansen, J. 1964. Ind. Vet. J. 41: 309-316), and geese (Anser anser domesticus) (Jansen and Wemmenhove, 1965. Tijd. Diergeneesk. 90: 811).

Subsequent to the first reported American outbreak in White Pekin ducks referred to above, a surveillance was initiated for duck plague in American Anseriformes other than White Pekins (Leibovitz and Hwang, Bull. Wild. Dis. Assoc., Jan. 1968). The following is a progress report of the continued surveillance on Long Island, representing approximately the last six months of 1967. Further attempts were made to isolate and identify the virus from wild birds during this period.

Virus Isolation and Identification

Swans, geese and ducks submitted to the laboratory for diagnosis were examined for gross lesions. Pieces of the livers and spleens of individual birds were taken for virus isolation. The tissues were homogenized, and antibiotics were added to the homogenate which was used as inoculum. Each inoculum was injected intramuscularly into newlyhatched susceptible White Pekin ducklings, and onto the chorio-allantoic membrane of duck eggs on the 14th day of incubation. Tissue samples were considered duck plague virus positive if the inoculated ducklings and duck embryos died within 7 days postinoculation, and with mortality patterns and gross lesions characteristic of duck plague virus infection. When requested, specimens of duck plague virus positive tissue samples were then submitted for confirmation to the Plum Island Animal Disease Laboratory of the U. S. Department of Agriculture.

RESULTS

Of the 36 laboratory accessions received from Long Island, 15 were positive for duck plague, representing outbreaks at five locations during November and December (see Table 1). The largest single outbreak occurred in the Flanders Bay area. A known infected White Pekin duck farm was located approximately two and one quarter miles across the bay from this vicinity. The black duck population of the Flanders Bay area at this time was estimated at 1500 by Mr. Harry Greenwald, U.S. Game Management Agent, U.S. Fish & Wildlife Service. In addition, the estimated ratio of black ducks to mallards was 4:1 respectively. Other species of waterfowl were present in this area at the time, but estimates of their number could not be made. A total of 80 black ducks, 19 mallards, one Canada goose, and one bufflehead were found dead in this area. Specimens from each of these accessions were positive for duck plague. In addition, one white-winged scoter was found dead in the same area during the outbreak but was negative for the disease.

The first accession was received from the Flanders outbreak on November 17. Hunting season started on November 4 and dead ducks observed in the area as early as the 5th or 6th of November were attributed to normal hunting mortality. However, the mortality persisted from this time until the receipt of the first accession. Local observers reported that while mortality was persistent, the number of dead birds increased during the accession period. The last accession from the Flanders outbreak was received on December 5, 1967; accordingly, the accession mortality extended over an 18day period, but the history suggests a 30-day die-off period for this single episode.

During the Flanders outbreak, dead ducks were found floating among the vegetation in the shallow water along the entire length of three creeks that feed into Flanders Bay. As seen from the

surface of the water, the back of the head, extended neck, partially flexed wings, back, and extended feet, with the plantar surface of the web uppermost, were visible. The beak and the face were submerged. Some of the male mallards evidenced prolapse of the penis. When the birds were picked up and suspended by their feet, a dark brown tarry fluid flowed from the mouth.

Among the mortality of the Flanders outbreak, three banded black ducks were found. Two had been banded previously at Flanders Bay; one had been banded at Winterport, Maine.

On the 26th of November a single moribund adult female greater scaup was found on a road near the Village of Bellport, Long Island, N.Y., approximately three miles distant from the nearest duck farm and ten miles from the nearest known infected duck farm. Necopsy revealed hemorrhages and injuries. The specimen was positive for duck plague.

On the 19th of December, a wild adult female mallard duck was found dead on a pond maintained for a collection of ornamental captive waterfowl. Upon necropsy the bird evidenced heavy parasitism; however, no lesions of duck plague were found. Duck plague virus was recovered from this bird. On the same premise during the first surveillance period, duck plague virus had been isolated from muscovy ducks.

On the 20th of December, four dead plague-positive black ducks were found in the vicinity of East Moriches, Long Island, N.Y. The finders reported that dead black ducks had been observed for some time in this locality. Neither the total black duck population nor the total mortality could be estimated in this outbreak. The lesions observed in these black ducks conformed to those previously seen in the same species during the Flanders outbreak. Known infected duck farms were located within a one-half mile radius of this outbreak.

On the 21st of December a sample of

three mallard ducks was received from the vicinity of Southaven County Park on Carman's River, L. I., N. Y. Two of the ducks were mutilated by scavengers and were not satisfactory for examination. The remaining mallard had lesions of duck plague and the diagnosis was confirmed by virus isolation. These birds were found dead approximately 200 yards from a White Pekin duck farm that had been under continued surveillance and was negative for the disease up to and including the time of this report.

TABLE 1. Results of Duck Plague Virus Isolation

8/17/67		Location	Isolation
8/17/67	CYGNINAE		
	Mute swan (Cygnus olor)	Kingston, R.I.	
11/27/67	" " "	Yaphank, L.I.	_
12/11/67	" " "	Oakdale, L.I.	
12/13/67	" " "	E. Moriches, L.I.	
12/27/67	" " "	Oakdale, L.I.	_
	ANSERINAE	January 2.2.	
11/3/67	Canada goose (Branta canadensis)	Quogue, L.I.	
12/5/67	" " " "	Flanders, L.I.	+
12/20/67	" (B. c. maxima)*	,, ,,	<u> </u>
7/15/67	Richardson's goose (Branta canadensis		
// 1// 0/	hutchinsii)*	Islip, L.I.	
11/1/67		1511p, L.1.	_
8/24/67	Canada goose (B. c. minima)* Emperor goose (Philacte canagica)*	,, ,,	
			_
	ANATINAE		
8/4/67	Mallard duck (Anas platyrhynchos		
	platyrhynchos) *	Southampton, L.I.	
8/21/67	Mallard duck (Anas pl. pl.)	Riverhead, L.I.	-
9/18/67	,, ,, ,, ,, ,, ,, ,, ,, ,, ,, ,, ,, ,,	W. Islip, L.I.	
11/13/67	" " " " *	Flanders, L.I.	_
11/24/67	" " " " op	" "	+
12/1/67	" " " "	Southaven, L.I.	į.
12/2/67	,, ,, ,, ,,	Riverhead, L.I.	
12/5/67	" " " " ¢¢	Flanders, L.I.	+
12/19/67	" " " "	Islip, L.I.	i
12/21/67	,, ,, ,, ,, ,,	Southaven, L.I.	<u> </u>
10/4/67	Hybrid (Mallard X Black) duck	Flanders, L.I.	<u>.</u>
11/17/67	Black duck (Anas rubripes) **	,, ,,	
11/18/67	" " " " **	" "	<u> </u>
11/20/67	" " "	Sayville, L.I.	<u>.</u>
11/20/67	,, ,, ,, ,, _{**}	Flanders, L.I.	
11/24/67	,, ,, ,, ,,	, L.I.	ᆂ
12/5/67	n n n n to	" "	<u>.</u>
12/12/67	" " " "	Oakdale, L.I.	
12/20/67	,, ,, ,, ,, ,,	E. Moriches, L.I.	
12/20/67	" " " "	E. Moricnes, L.I.	
1/12/68	Blue Winged Teal (Anas discors)*	Islip, L.I.	T
1, 12,00	AYTHYINAE	1511p, L.1.	_
12/8/67	Canvasback (Aythya valisineria)	Jamaica, L.I.	_
11/26/67	Greater Scaup (Aythya marila)	Bellport, L.I.	+
11/28/67	Bufflehead (Bucephala albeola)	Flanders, L.I.	+ +
12/8/67	" " "	Jamaica, L.I.	
12/4/67	White-winged Scoter (Melanitta deglandi)	Flanders, L.I.	_

^{*} Reared in captivity

^{**} Specimens submitted to and confirmed by Plum Island Animal Disease Laboratory, Greenport, N. Y.

Discussion and Conclusions

During its brief history in Europe and Asia, duck plague has been presented principally as a disease of domestic ducks. Although many outbreaks have occurred on open water inhabited by wild ducks, and many species of wild Anseriformes have been shown to be experimentally susceptible to infection, appraisal of the importance or incidence in wild Anseriformes is lacking. This dearth is probably the result of the imbalance in disease detection between wild and domestic waterfowl. The continuous maintenance and replacement of large populations of young susceptible domestic ducks, under constant surveillance by experienced personnel in concentrated duck producing areas, provides an extremely sensitive system for monitoring the presence of anatine diseases. In contrast, knowledge of diseases of wild waterfowl is often dependent upon chance observation of more obvious "die-offs". Field conditions offer obstacles such as delayed reports of mortality, inability to recover adequate numbers of affected birds, decomposition, mutilation of carcasses by predators and scavengers, and poor transportation and preservation of specimens en route to laboratories.

It is also difficult to believe that this disease is geographically limited in importance to a small duck producing area of Europe (Netherlands and Belgium) and the remainder of that continent is free of the disease.

As an unreported disease on the American Continent prior to January 3, 1967, duck plague can be considered an emerging exotic infection, until evidence is presented to establish or refute its prior presence. On Long Island, duck plague has spread rapidly from one farm to another in this concentrated duck producing area, and represents a new disease experience to this relatively old industry.

The current Long Island surveillance period represents approximately the last half of 1967. The number of infected

birds, species and locations increased during this period. The infection was detected in mallards, black ducks, a bufflehead, a greater scaup, and a Canada goose. Of these losses, the greatest loss was in black ducks, and secondly, in mallards. In the Flanders outbreak mortality appeared related to the population density of a given species in an infected area rather than specific species susceptibility. Necropsy findings in affected mallards conformed to the description of the classical disease in domestic ducks. Lesions in black ducks and the Canada goose were distinctive, and differed from those described in the literature. No detectable lesions of the disease were found in the Aythyinae (bufflehead and greater scaup), and death in these birds was attributed to gunshot wounds and injuries. The greatest incidence of the disease in wild Anseriformes occurred during November and December. The latter represents a normal migratory period for waterfowl on Long Island. If this disease is an emerging exotic infection in wild Anseriformes, then it is probable that there will be a future geographical extension from the known loci of infection. This extension would then conform to the migratory patterns of susceptible Anseriformes. The disease would tend to be expressed by the movement of susceptible birds into infected areas. Recovered or carrier birds could establish new infective environments. In a sense, the population density of many species of waterfowl is comparable with commercial duck production, and it is likely that mortality similar to domestic outbreaks could be experienced. If this disease is not new to the American Continent, then outbreaks should occur in a random fashion.

The need is urgent to extend this surveillance into other areas to determine the distribution of the disease. The lesions should be characterized for each susceptible species so that duck plague can be recognized with greater rapidity.

The importance of this disease should be established.

Acknowledgements

We appreciate the efforts of Mr. Harry Greenwald of the U. S. Fish and Wildlife Service, and Mr. John Kruzan and Mr. Carl Helms of the New York State Conservation Department, whose sincere interest made this study possible. We are grateful to he Plum Island Animal Disease Laborators of the

U. S. Department of Agriculture for their assistance in confirming a portion of these diagnoses.

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VISCERAL GOUT IN A CAPTIVE COOPER'S HAWK

A male Cooper's hawk (Accipiter cooperii) was taken into captivity by a falconer as a nestling (the primary feathers of the wings and tail were half grown). The bird refused food for the first 36 hours after capture but ate well thereafter. Diet during the period of captivity consisted of chicken hearts and an occasional small mammal or bird.

Approximately two weeks after capture, the bird's left leg seemed weak; the following day it refused to bear weight on the limb. A vitamin-mineral supplement* was added to the diet: 1/4 teaspoon was given daily for a week, then 1/8 teaspoon once a week for 5 weeks. The leg functioned normally a week after therapy was begun.

The hawk's adult weight was maintained at 10 ounces by controlled feeding based on daily weighing. It never progressed far enough in its training to be flown or exercised daily. Two months after capture, the hawk was accidentally exposed to a rain shower. During the next 24 hours, the bird lost approximately 1 ounce in weight, refused food, rapidly became listless, and died. Necropsy was performed within one hour after death.

When necropsied, the bird's general body condition was good and the plummage was excellent. "Hunger streaks"

or "shock marks" were seen on the mid-shafts of wing and tail feathers. These are transverse defects of the shafts and barbs of feathers, usually due to stress during feather growth; in this case they were probably due to the bird refusing food for 36 hours following capture.

The pericardium and air sacs appeared to be "dusted" with a pale yellow powder. The epicardium and myocardium were heavily mottled with small white spots. Many chalky streaks and plaques were seen beneath the fascia of the muscles on the ventral side of the neck.

Both kidneys were pale and slightly enlarged, with numerous white nodules on the external and sectioned surfaces.

Three pale yellow concretions were found in the cloaca. They were irregular in shape, ½ inch in diameter, and crumbled easily. Cloacal fluid was thick, chalky, and felt gritty.

We observed no other gross abnormalities.

Tissues were fixed in 10% formalin, sectioned at 6 microns, and stained with hematoxylin and eosin. Histologically, multiple small, focal lesions were observed throughout the myocardium. The lesions consisted of a central zone of radiating sheaves of eosinophilic crystallike material (Figure 1) morphologically consistent with sodium urate crystals (Allen, *The Kidney*, Grune and Stratton, 1951). This zone was surrounded by mononuclear and multinu-

^{*} Vitatone; Fort Dodge Laboratories, Inc., Fort Dodge, Iowa.