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EXPERIMENTAL DUCK PLAGUE IN BLUE-WINGED TEAL AND CANADA GEESE

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ABSTRACT: Ten adult blue-winged teal (*Anas discors*) and six Canada goose (*Branta canadensis*) goslings were inoculated with liver tissue from a natural case of duck plague in a wild mallard (*Anas platyrhynchos*). Four additional teal were placed in contact with the inoculated ducks. Inoculated teal died 63.5–68 hr after inoculation; two of the contact teal died 161–162 hr after exposure. Three of the goslings died 119–133 hr after inoculation, the others were killed when moribund 90–133 hr postinfection. The clinical course of disease was extremely rapid in both species. Signs were limited to sudden onset of profound weakness, ataxia, tremors and terminal convulsions. The only consistent gross lesion in the teal was a small dark spleen; half the teal also had inconspicuous foci of epithelial necrosis in the distal esophagus and in the cloaca. Goslings had more severe lesions, with focal hepatic necrosis visible in all, and mucosal necrosis over the intestinal lymphoid tissue, and intestinal hemorrhage in three birds each. Microscopic lesions in both species were similar to those reported in mallards. Duck plague might be overlooked at necropsy of blue-winged teal, because of the paucity of gross lesions.

Key words: Duck plague, pathology, ducks, geese, diagnosis, *Anas discors*, *Branta canadensis*.

INTRODUCTION

Duck plague (DP) represents an enigma in wild waterfowl. Characteristics of the causative herpesvirus including long-term persistence of infection in carrier birds with periodic shedding of virus (Burgess et al., 1979; Burgess and Yuill, 1983); transovarial transmission (Burgess and Yuill, 1981); ease of transmission by a variety of routes (Dardiri, 1971; Spieker, 1978) and relative hardiness of the virus in surface water (Wobeser, 1981) all suggest that infection could become widespread in nature. Since its recognition in North America in 1967 (Leibovitz and Hwang, 1968) outbreaks have occurred at widely dispersed localities among captive waterfowl, but although wild waterfowl shared ponds with captive birds in most of these instances, DP has been reported only in free-flying birds on four occasions (Leibovitz, 1968; Dardiri and Butterfield, 1969; Friend and Pearson, 1973; Wobeser and Docherty, 1987). Brand and Docherty (1984) did not isolate the virus from any of almost 5,000 wild waterfowl sampled across the United States and concluded that DP is not widespread currently.

All species of Anatidae are probably susceptible to infection, but the response to infection varies with the strain of virus, species, age and perhaps sex of the bird (Van Dorssen and Kunst, 1955; Dardiri and Butterfield, 1969; Leibovitz, 1969, 1971; Spieker, 1978). Because of this variability, DP might occur over a broad spectrum from inapparent infection to severe, rapidly fatal disease and it is possible that the disease could go unrecognized in nature. Most reports of the pathology of DP deal with mallards (*Anas platyrhynchos*), domestic ducks derived from mallards, or muscovy ducks (*Cairina moschata*). Leibovitz (1969) warned that gross lesions were "maximal" in mallards and might be considerably less obvious in other species. Spieker (1978) found the blue-winged teal (*Anas discors*) to be the most susceptible among six native species he inoculated with DP, but natural cases have not been reported in this species. The Canada goose (*Branta canadensis*) was one of the less susceptible species among those tested by Spieker (1978). However, natural cases have been reported in this species by several authors (Leibovitz, 1968; Friend and

Pearson, 1973; Bernier and Filion, 1975; Montgomery et al., 1981). This report describes experimental infection of adult blue-winged teal and Canada goose goslings with DP obtained from a spontaneous case in a free-flying mallard.

MATERIALS AND METHODS

The inoculum used for infection was liver tissue from a wild mallard found dead of DP near Saskatoon in 1984 (Wobeser and Docherty, 1986). Tissue held frozen at −70 C for approximately 12 mo until used for the trials was prepared as a 10% suspension in phosphate-buffered saline containing 1,000 I.U. penicillin and streptomycin/ml. The amount of virus within this tissue suspension was not determined because of lack of a suitable tissue culture system at the time of the experiment. Fourteen blue-winged teal (seven of each sex), approximately 13 mo old, were used. These birds had been hatched the previous year from eggs collected in the wild and reared indoors together with ducks of other species also hatched from eggs collected in the wild. Seven Canada goose goslings, approximately 7 wk old, obtained as day-old hatchlings from Brooks Wildlife Centre, Brooks, Alberta and reared in an isolation room were used.

The teal were divided equally among groups on the basis of sex; six received 0.1 ml inoculum by intramuscular (IM) injection; four received 1.0 ml inoculum per os via a soft plastic tube inserted into the upper esophagus, and the remaining four birds were not inoculated, but were placed in direct contact with the infected birds. The teal were placed either two or three (two inoculated, one contact) birds to a 80 × 80 cm cage with a wire screen floor. Six of the Canada geese were given 0.1 ml inoculum by IM injection and placed in an isolation room. The seventh gosling was held separately, necropsied and tissues collected for control purposes. Feed and water were supplied to all birds ad libitum.

Birds were observed regularly and clinical signs were recorded. Blue-winged teal were necropsied as soon after death as possible (usually within 1 hr); three of the geese were necropsied shortly after death, the other three goslings were killed when moribund and necropsied. Tissues collected at necropsy were fixed in 10% neutral buffered formalin, processed routinely, sectioned at 5 μm and stained with hematoxylin-eosin for histologic examination. Liver tissue was cultured aerobically for bacteria at 37 C on 5% sheep blood agar and MacConkey agar.

RESULTS

Clinical observations

All of the inoculated teal died. The median time to death (range) for birds given inoculum via IM injection and per os was 66.5 (63.5–77.5) and 75 (72–86) hr post-inoculation (PI), respectively. Two of the in-contact teal died 161 and 162 hr after being placed in contact with the inoculated birds. The other two in-contact teal remained healthy and had no gross or microscopic lesions when killed 15 days after exposure. Clinical signs were very limited in the teal and the average time from first observed clinical abnormality to death was 4.6 hr (0–14.5). The first sign observed in most birds was elevation of the feathers on the dorsum of the head and neck, followed by general elevation (ruffling) of the body feathers. Seven of the 12 teal were ataxic and in all 12 there was lethargy that progressed rapidly to inability to rise from a sitting position. Two birds convulsed immediately prior to death and one of these had opisthotonus terminally. Clear serous fluid exuded from the bill of one teal immediately after death.

The Canada geese survived longer after infection than did the teal. Three goslings were found dead 119, 119.5 and 133 hr PI. The other three goslings were killed when in a moribund state and unable to stand at 90, 110, and 113 hr PI. Clinical signs were limited to the sudden onset of profound weakness that progressed rapidly to inability to stand or raise the head, with head and neck tremors. One bird regurgitated a few ml of bloody fluid 3 hr prior to its death. None of the goslings was observed to have clinical signs for more than 3 hr prior to death.

Gross pathology

Grossly visible lesions were minimal in the blue-winged teal (Table 1) and no difference was noted among birds infected by various routes. The only change present in all birds that died was a small, extremely

FIGURE 1. Distal esophagus of blue-winged teal with duck plague. Tiny 0.5–1.0 mm raised white foci of epithelial necrosis are present in the mucosa.

dark spleen, usually with a maximum diameter of about 4 mm. Ten of the teal had clear or bile-stained fluid in the esophagus and proventriculus. The alimentary system was empty in all. Superficial necrosis of the epithelium of the mucosa of the esophagus and cloaca was present in three and six teal, respectively. This change was limited, even in the most severely affected teal (Fig. 1). One bird had a single tiny petechial hemorrhage in the mucosa over an intestinal lymphoid aggregation, but this was not visible from the serosal surface and there was no grossly visible necrosis of the mucosa overlying the "annular bands" in any of the teal.

Lesions occurred with a greater prevalence and were more severe in the Canada geese than in the teal (Table 1), and all goslings had conspicuous necrosis and hemorrhage in the liver (Figs. 2, 3). Three of the six birds had necrosis of the mucosa over the intestinal lymphoid discs (Fig. 4) and the content of the upper intestine was bloody in three. The spleen in all inoculated geese was smaller than that of the control bird and so dark that it resembled a blood clot. Foci of necrosis were visible on the cut surface of the spleen in two birds. An adherent white membrane was present on the mucosa of the distal esophagus in two goslings (Fig. 4). Pathogenic bacteria were not isolated from any of the experimental birds.

TABLE 1. Frequency of occurrence of selected gross lesions in adult blue-winged teal and Canada goose goslings infected experimentally with duck plague virus.

Species	Blue-winged teal	Canada goose
Sample size	12	6
Esophagus		
Excess clear or bile-stained mucus	10	5
Petechial hemorrhage	4	0
Focal epithelial necrosis	3	2
Heart		
Epicardial petechiae	2	1
Liver		
Focal necrosis	0	6
Petechiae	3	2
Ecchymoses	0	2
Small intestine		
Bloody content	0	3
Petechiae (mucosa)	3	0
Colon		
Petechiae (mucosa)	5	0
Focal epithelial necrosis	1	0
Cloaca		
Mucosal hemorrhage	3	0
Epithelial necrosis with adherent membrane	6	2
Spleen		
Small, dark	12	6
Necrotic foci	0	2
Intestinal lymphoid aggregates		
Hemorrhage	1	3
Necrosis of overlying epithelium	0	3
Bursa of Fabricius		
Fibrinous core	NA*	1
Mucosal hemorrhage	NA	1

* Not applicable.

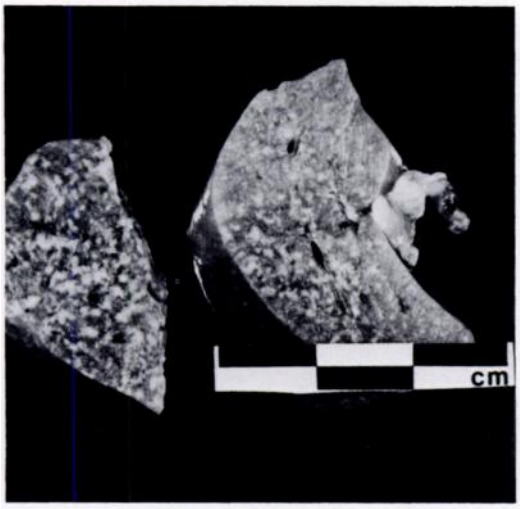

FIGURE 2. Extensive hemorrhage in the liver of a Canada goose gosling with duck plague.

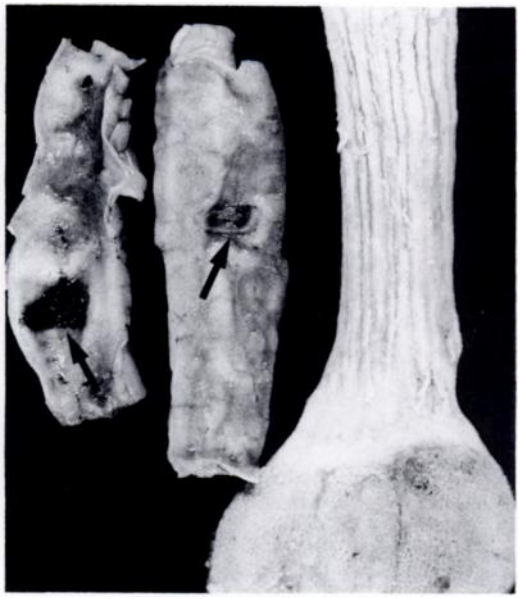

FIGURE 4. Canada goose gosling with duck plague. The epithelium overlying the intestinal lymphoid disc is necrotic with hemorrhage (arrows). There is accumulation of white necrotic debris on the mucosa of the distal esophagus.

Microscopic pathology

The extent and uniformity of microscopic changes in the blue-winged teal was surprising in view of the paucity of gross lesions. In the liver there was marked vacuolation of hepatocytes, particularly in the periportal regions, together with focal necrosis of hepatocytes. There was also necrosis of bile duct epithelium and peri-

FIGURE 3. Multifocal necrosis of the liver of a Canada goose gosling with duck plague.

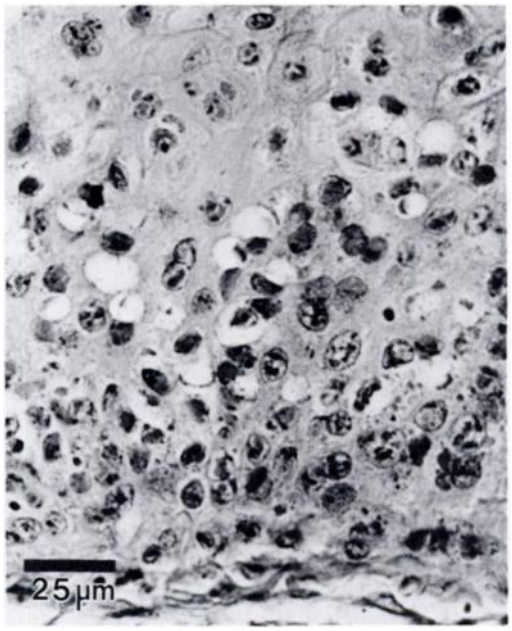

FIGURE 5. Vacuolation, necrosis and intranuclear inclusion bodies in epithelium of cloaca of blue-winged teal with duck plague. H&E.

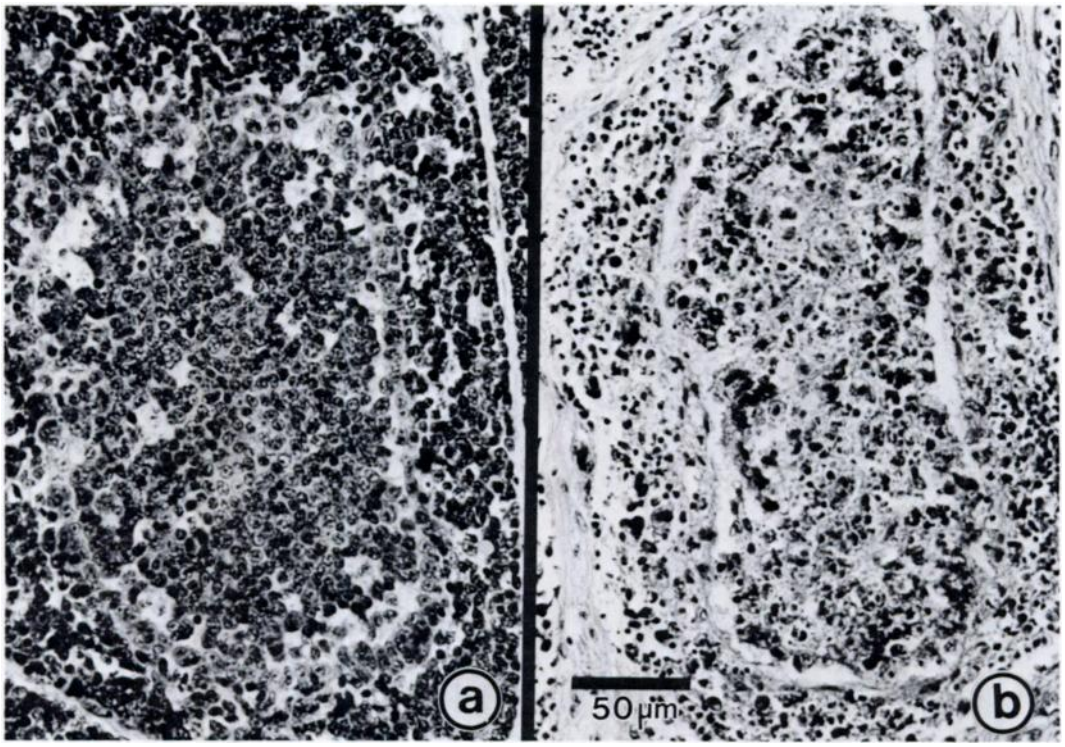

FIGURE 6. Comparison of lymphoid follicles from bursa of Fabricius from normal (a) and duck plague infected (b) Canada goose goslings. There is total necrosis of lymphoid elements in the latter. H&E.

portal accumulation of lymphocytes, with intranuclear inclusion bodies in hepatocytes and bile duct epithelial cells. There was necrosis of lymphocytes in the spleen, and in most teal all that remained of the lymphoid tissue was fragmented nuclear debris. Inclusion bodies were uncommon in the spleen. Lymphoid tissue in the submucosa of the alimentary tract at the esophageal-proventricular junction, intestinal annular bands and in the cloaca, as well as that in the thymus, was undergoing necrosis and cytolysis. Cells of Hassal's corpuscles in the thymus contained intranuclear inclusion bodies in most teal. There were many necrotic, unidentifiable cells in the lamina propria of the small intestine, with necrosis of epithelial cells and intranuclear inclusion bodies in cells in some crypts. Changes in the stratified squamous epithelium of the distal esophagus and of the cloaca were similar in type, with focal vacuolation, cellular swelling, intercellular edema and cell necrosis apparently occurring first in the suprabasilar layers and proceeding to total necrosis with ulceration, adhesion of necrotic debris and bacterial colonization of the adherent membrane and ulcerated surface. Intranuclear inclusion bodies were prominent in epithelial cells at these sites (Fig. 5).

The microscopic lesions in Canada geese were similar to those in the teals, but in general were more severe, particularly in the liver, in which necrosis ranged from focal to massive with widespread hemorrhage. There was no recognizable lymphoid tissue in the spleen, and in the thymus the lymphoid tissue was represented by a mass of pyknotic nuclei surrounding necrotic epithelial tissue of Hassal's corpuscles. There was marked necrosis of lymphoid tissue in the bursa of Fabricius and in the most severe cases all elements

TABLE 2. Reported occurrence of selected gross lesions in waterfowl with duck plague.

	Mallard (*Anas platyrhynchos*)			Black duck (*A. rubripes*)			Canada goose (*Branta canadensis*)			Blue-winged teal (*A. discors*)	Gadwall (*A. strepera*)	Redhead (*Aythya americana*)	Wood duck (*Aix sponsa*)
Author[a]	L	P	S	L	J	M	L	S	M	S	S	S	S
No. examined	15	76	15	45	55	20	1	9	4	5	5	6	9
Hemorrhage													
Esophageal-proventricular junction	++[b]	often	+	+	NQ[c]	NR[d]	—	—	—	—	—	+	—
Intestine (free blood)	+++	+++	+	+	NR	—	—	++	—	—	+++	—	—
Heart	+++	+++	+	+++	NQ	++	P[e]	++	++	++	++++	+	—
Bloody discharge													
Oral	NQ	++	+	NQ	—	—	—	—	—	++	++	—	—
Cloacal	NR	++	+	NR	—	—	—	++++	—	++	++++	—	—
Epithelial necrosis													
Esophagus	+++	NQ	+++	++	NQ	+++	—	+	+++	+	+++	—	—
Cloaca	+++	NQ	—	+	NQ	+++	P	+	+++	—	+	+	—
Necrosis and/or hemorrhage													
Intestinal lymphoid aggregates	++++	++++	+	++++	—	+	P	++	—	—	+	++	—
Liver	++++	++++	+++	+++	—	++	P	+	+++	—	+	+	—
Dark spleen	++++	++++	+	++++	NQ	—	P	—	—	++	—	++	—

[a] Author: L = Leibovitz (1969), P = Proctor et al. (1975), S = Spieker (1978), J = Jacobsen et al. (1976), M = Montgomery et al. (1981).
[b] ++ = 25–50% of birds had lesion (+ = <25%, +++ = 50–75%, ++++ = >75%), — = no birds had lesion.
[c] NQ: present, not quantified.
[d] NR: not reported.
[e] P = present in single bird examined.

within follicles were necrotic (Fig. 6). Esophageal, intestinal and cloacal lesions were similar to those in blue-winged teal. There was total destruction of lymphoid elements of the intestinal lymphoid discs with necrosis of the overlying intestinal mucosa and adherence of fibrino-necrotic debris at these sites. Three of the goslings had degeneration and/or necrosis of pancreatic duct epithelium, with intranuclear inclusion bodies in duct cells.

DISCUSSION

These experimental infections confirm the variability in expression of DP reported in different species of waterfowl (Table 2). Blue-winged teal were the most susceptible among eight species of waterfowl tested by Spieker (1978) and lesions in the five teal he infected with the Lake Andes Strain of DP virus were limited to petechiae on the heart (two birds), small dark spleen (two birds), hemorrhage in the colonic mucosa (three birds) and hemorrhage and epithelial necrosis at the esophageal-proventricular junction in one bird. Gross lesions were even more limited in teal in the present experiment, and many of the lesions usually associated with DP in mallards and domestic ducks, such as focal hepatic and splenic necrosis, and necrotizing hemorrhagic changes associated with the intestinal lymphoid tissue were not found in teal in either study.

This was probably not a function of the virus strains used in the experimental infections, as mallards infected naturally with both the Lake Andes strain and the virus used in this experiment had "classical" lesions of DP (Friend and Pearson, 1973; Wobeser and Docherty, 1987). The inability to quantify the infective dose of virus used in the present trial limits comparison with other studies, and is a recognized shortcoming of the research. Similarly, virus isolation could not be attempted from tissues of the experimentally infected birds. Virus was isolated from Pekin ducklings and a mallard that died with lesions typical of DP after inoculation with material from the same source (Wobeser and Docherty, 1987).

Had the blue-winged teal in this experiment been presented as naturally-occurring field cases to the diagnostic laboratory, DP would likely not have been considered as a differential diagnosis in most of the birds, because of the paucity of gross lesions. Even in the most severely affected teal the changes were subtle and could be overlooked easily, particularly if the distal esophagus and the cloaca were not examined carefully.

The lesions in the Canada geese goslings were consistent with those reported by others (Table 2) and should have lead to a consideration of DP, had the birds been found dead in the wild and submitted as diagnostic cases.

The results of this and earlier studies (Table 2) suggest that DP could go unrecognized in many wild waterfowl, notably blue-winged teal and wood ducks (*Aix sponsa*). This would be true particularly if the examiner expected to find "pathognomonic" gross lesions such as hepatic necrosis and hemorrhagic or necrotizing lesions of the intestinal lymphoid tissue in all birds. Because of variability of this disease, it is important that an adequate sample of birds be examined in every outbreak situation; that necropsies be complete and include examination of all lymphoid organs, the distal esophagus, and the cloaca; and that histology and attempted virus isolation be done on a subsample of birds whenever possible. Microscopic lesions appear to be more consistently present than are gross lesions. Sections of distal esophagus, cloaca, liver and lymphoid organs should always be examined. If chronic or "mild" forms of DP, as described in Europe by Gaudry (1974) and Kapp et al. (1982), were to occur in wild waterfowl, recognition would be even more difficult.

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