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RENAL COCCIDIOSIS IN WILD DUCKS IN SASKATCHEWAN

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Abstract: Ten of twelve species of wild ducks examined in central Saskatchewan were infected with renal coccidia. Of 261 ducks examined during autumn migration, 24.5% were infected while only 4% of 74 ducks examined during spring migration were infected. The greatest prevalence of infection occurred in female and juvenile birds. No gross lesions attributable to coccidia were found, and microscopic lesions were focal in nature. Oocysts from mallards were transmitted to captive mallards.

INTRODUCTION

Renal coccidiosis due to *Eimeria truncata* has been known to be a significant parasitic disease of domestic geese since its first description in 1890[11]. Renal coccidiosis due to *Eimeria boschadis, Eimeria somateriae* and unspecified coccidia has been reported in ducks in Europe and North America[8,9,11,12]. Other than one outbreak in common eiders *(Somateria mollissima)*[9], these parasites have not been reported to cause clinical disease in ducks.

The objectives of the present study were to determine the prevalence of renal coccidia in wild migratory ducks in Saskatchewan, to determine the pathologic changes associated with renal coccidia in these birds, and to attempt experimental transmission of the infection.

MATERIALS AND METHODS

Kidneys and wings were collected from 220 ducks of 12 species shot by hunters in the Saskatoon area in 1974, and from 41 ducks of 9 species in the autumn of 1975. Twenty-five adult mallards *(Anas platyrhynchos)* were collected by special permit in the spring of 1975, and 48 lesser scaup *(Aythya affinis)* and 1 ring-necked duck *(Aythya collaris)*, which died of unknown causes, were examined in the spring of 1976. Birds were identified to species and age by wing plumage characteristics[1]. Sex was determined by internal examination where possible, and by plumage characteristics[1] when internal organs other than kidneys were not available.

A smear of ureteral content was prepared[12] and examined microscopically. If oocysts were present on the direct smear, the ureteral content was placed in 2% potassium dichromate ($K_2Cr_2O_7$) solution for sporulation and then refrigerated. Kidneys were fixed in 10% neutral buffered formalin, processed routinely, sectioned transversely at a thickness of 6 μm, stained with hematoxylin and eosin (H&E), and examined microscopically.

Oocysts obtained from mallards in the autumn of 1974 were held in 2% $K_2Cr_2O_7$ at 4 C for a period of 9 months after sporulation. Transmission of these oocysts via gastric entubation was attempted to 45 - 1 month-old domestic Pekin and 35 - 1-2 week-old wild mallard ducklings. Oocysts obtained in the autumn of 1975 from a wild mallard were maintained in 2% $K_2Cr_2O_7$ at room temperature for 1 week and then administered to four 6-month-old wild mallards. Two of the latter birds were given a single intramuscular injection of 2 mg betamethasone at the time of infection.

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Excrement from experimentally infected ducks was collected daily for 25 days, concentrated[3], and examined microscopically. Birds were killed 25 days after inoculation, kidneys were removed, and ureteral scrapings and sections for histopathology prepared and examined as described above.

RESULTS

Of 220 ducks surveyed in the autumn of 1974, 54 (24%) were infected with renal coccidia (Table 1). Of these, 20 were positive by ureteral smears, while 42 were positive by histopathology. Coccidia were not found in tissue sections in 12 of 20 birds which had oocysts on ureteral smear, and conversely, 34 of those birds with coccidia in tissue sections were negative when checked by ureteral smear. Of the 41 ducks surveyed in the autumn of 1975, 11 (26.8%) had renal coccidia. Of the 11 positive ducks, 6 had oocysts in ureteral smears while 8 were found to be positive on histopathology. Individuals of all seven species of dabbling ducks examined were infected with renal coccidia, while infection was found in three of five species of diving ducks. Blue-winged teal *(Anas discors),* American widgeon *(Anas americana),* and gadwall *(Anas strepera),* in particular, had a high prevalence of infection. Juvenile and female birds had a significantly higher rate of infection by coccidia than did adults and males, respectively, (Table 2, 3).

TABLE 1. Renal Coccidiosis in Hunter-Killed Birds Autumn of 1974 and 1975.

	Species	Number Examined	% Positive
Dabbling Ducks	Mallard *(Anas platyrhynchos)*	109	13.8
	Blue Winged Teal *(Anas discors)*	38	52.6
	Northern Shoveler *(Anas clypeata)*	24	8.3
	American Widgeon *(Anas americana)*	18	44.0
	Pintail *(Anas acuta)*	18	5.5
	Gadwall *(Anas strepera)*	19	31.6
	Green Winged Teal *(Anas crecca)*	19	26.3
Diving Ducks	Redhead *(Aythya americana)*	9	44.4
	Lesser Scaup *(Aythya affinis)*	3	66.6
	Canvasback *(Aythya valisneria)*	2	50.0
	Ruddy Duck *(Oxyura jamaicensis)*	1	0
	Ring-Necked Duck *(Aythya collaris)*	1	0
	TOTAL	261	24.5

TABLE 2. Age Distribution of Renal Coccidiosis in Hunter-Killed Ducks.

Age of Birds	Number Examined	% Positive
Adult	76	14.5*
Juvenile	179	31.3*
Unknown	6	16.6

*Statistically different ($P < 0.05$), as determined by chi-square.

TABLE 3. Sex Distribution of Renal Coccidiosis in Hunter-Killed Ducks.

Sex of Birds	Number Examined	% Positive
Male	138	21.0*
Female	112	28.6*
Unknown	11	18.2

*Statistically different ($P < 0.05$), as determined by chi-square.

No oocysts were found in ureteral smears from any of 25 mallards collected in the spring of 1975, but coccidia were detected by histopathology in one bird. All 48 lesser scaup and one ring-necked duck examined in the spring of 1976 were negative by ureteral smear, but coccidia were detected on histopathology in two scaup.

Oocysts from the kidneys of all species of ducks were similar; however, only those isolated from mallards were chosen for description and transmission studies. Oocysts from mallards were unsporulated when collected. They were ovoid, slightly asymmetrical and had a micropyle contained in a "bottle neck" projection (Fig. 1). The oocyst wall was colorless and rough, making internal structures hard to visualize. It was 1 μm thick and composed of opaque outer and inner layers with a translucent middle layer. The mean dimensions of 157 oocysts were 13.5 μm by 23.7 μm (range 10-22 μm by 17-30 μm). The micropyle was 2-3 μm wide measured at the distal end of the "bottle neck".

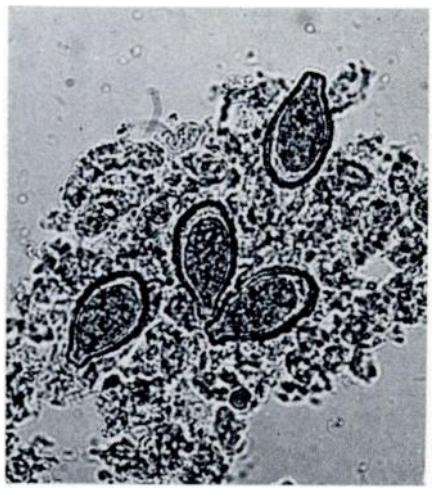

FIGURE 1. Unsporulated oocysts obtained from the ureter of a mallard. Note elongate pole containing micropyle and rough wall. Bright field x 1030.

Sporulation occurred in 19.7% of the oocysts examined. An oocyst residuum was formed. Four sporocysts were present in sporulated oocysts, each measuring approximately 7 μm by 9 μm and containing two sporozoites. A Stieda body was present. Sporocyst residua were not seen, possibly due to the rough oocyst wall which made it difficult to see internal structures.

No gross lesions attributable to renal coccidiosis were found in the kidneys of any of the infected birds. Microscopically, the only stages of the life cycle identifiable in stained kidney sections were gametocytes and oocysts. These generally were located in the epithelial lining cells and the lumen of perilobular collecting ducts and medullary collecting tracts. Oocysts were found at all levels of the urinary tract distal to these points. Generally there was very little reaction to the coccidia in affected collecting tubules. Where present, the reaction consisted of slight epithelial hyperplasia, an increase in the amount of cellular debris in the tubular lumen and mild peritubular mononuclear cell response consisting predominantly of lymphocytes and macrophages. Apparent blockage of renal tubules by a mass of oocysts occasionally was observed. In such tubules pressure atrophy of tubular epithelial cells was evident.

Occasionally, gametogony and oocyst formation were found to occur in tubular cells of the renal cortical areas. In such cases the cellular reaction was more severe and consisted of large interstitial accumulations of lymphocytes and mononuclear cells with occasional heterophils. There was obliteration of some tubules by the inflammatory response and oocysts occasionally were found among the inflammatory cells.

Cellular destruction may be associated with gametogony and oocyst formation in the renal tubules since there was an increase in the amount of cellular debris in ureters of infected kidneys; however, the distribution of lesions in infected kidneys was of a very focal nature. It was not unusual to find a single collecting tubule to be distended by oocysts while immediately adjacent tubules were entirely free.

In addition to the renal coccidia, a number of other parasites were present also in the kidneys of these ducks. Unidentified trematodes were observed in histologic sections of renal veins and ureters. Trematode ova (surrounded by mononuclear cells and occasionally by heterophils) commonly were encountered in the renal parenchyma. Cestodes, subsequently identified as immature *Cloacotaenia megalops* (R. S. Freeman, 1975, pers. comm.) occasionally were encountered in the ureters.

None of 45 domestic pekin and 35 wild mallard ducklings given oocysts which had been stored for 9 months passed oocysts during the 25 day observation period, nor did any of these birds have oocysts present in the ureteral smears prepared at necropsy; however, 6 of the mallard ducklings had oocysts in their kidneys when examined by histopathology. Of 4 mallards given oocysts collected in the fall of 1975, 1 of the 2 steroid-injected birds passed oocysts from days 20 to 23, inclusive, post-exposure. Oocysts were passed in greatest numbers on days 21 and 22. The other steroid-injected and the 2 non-steroid injected ducks did not pass oocysts and no histopathologic evidence of infection was found in any of the birds.

DISCUSSION

Renal coccidiosis had been reported previously in greater scaup *(Aythya marila)*, pintails (*Anas acuta*), and mallards in North America.[8,12] This report increases by eight the number of North American species known to be infected.

The two species of renal coccidia described from ducks are *E. boschadis* in wild mallards in Sweden[11], *E. somateriae* in common eider in Denmark[2], and the long-tailed duck *(Clangula hymenalis)* in Sweden[11].

Oocysts isolated from mallards in this survey were variable in size, but most closely resembled the published description of *E. boschadis*[11], with the exception that the wall was rough in the present oocysts rather than finely granular as described for *E. boschadis*[11]. Oocysts described here differed from the descriptions of *E. somateriae*[2,7,11], in that those of the latter species were smaller, had a smooth wall, and had an oocyst residuum. The organism isolated from mallards in this survey resembles *E. truncata*[7], although the range of measurements is more variable. The results of previous attempts at transmission of *E. truncata* between geese and ducks have been inconclusive[4,6,10] so that the possibility of this organism being *E. truncata* cannot be ruled out.

Microscopic examination of kidney sections revealed a much higher prevalence of infection than did microscopic examination of ureteral smears. Due to the focal nature of the infection within an individual kidney, not all infections will be revealed by either histopathology or ureteral smear examination alone, and some infections were possibly missed even when both techniques were used.

The significance of renal coccidiosis as a disease of wild ducks is difficult to assess. There has been only one report of disease in ducks due to renal coccidiosis[9]. In that case, *E. somateriae* produced mortality in eider ducklings.[9] No gross lesions were found in any of the birds examined in the present study and the microscopic lesions of renal coccidiosis were focal in nature. The inflammatory response consisted almost entirely of

lymphocytes and macrophages. Heterophils were seldom seen, and when present were only found in small numbers. Tubular obliteration was seen in cortical areas affected by coccidia. It appears that the damage was insufficient to cause clinical disease. However, with the exception of the lesser scaup, the birds examined represented a sample of clinically normal birds, and may only represent birds with minimal infections or perhaps the survivors of earlier disease.

There was no difference in the prevalence of infection with renal coccidia between northward migrating birds and southward migrating adult birds; however, there was a significantly lower prevalence of infection in northward migrating ducks than in all southward migrating ducks (adult and juvenile). If renal coccidia were easily spread among adult birds, one might anticipate a high prevalence of infection in northward migrating birds, due to concentration of birds on southern wintering grounds. The low prevalence of infection of adult birds suggests that there may be some age-related or acquired "resistance" to renal coccidia. The higher prevalence of infection in female ducks may be important for the spread of coccidiosis to juveniles on the breeding grounds since juvenile ducks would have more contact with females than males.

The relatively poor results of attempted transmission in this study may be due to several factors. The 9-month storage of oocysts likely resulted in decreased viability. Sporulation rate was only about 20% for oocysts recovered from mallards, and while this was low, it should not have reduced the chance of transmitting infection. Although coccidiostat-free feed was used for experimental ducks, the feed was mixed commercially. Residual amounts of coccidiostats in commercial feeding mixing equipment may contaminate coccidiostat-free feeds mixed in such equipment (J. R. Allen, 1975, pers. comm.).

The presence of oocysts in the kidneys of experimentally infected ducks killed at 3 weeks post-infection, and the passage of oocysts only after 20 days in one bird suggest that this coccidium of mallards has a long prepatent period, in contrast to *E. truncata* which has a 5 to 6 day prepatent period[4].

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