AVIAN CHOLERA IN A BALD EAGLE FROM OHIO^{1, 2}

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ABSTRACT

Avian cholera (*Pasteurella multocida*) infection was diagnosed in an adult male bald eagle (*Haliaeetus leucocephalus*) collected in Ohio. Brain levels of organochlorine pesticides were found to be well below the reported lethal levels; the brain contained 10.7 ppm p,p'-DDE, 0.4 ppm p,p'-DDD, 1.2 ppm dieldrin, 1.1 ppm heptachlor epoxide, and 40.0 ppm polychlorinated biphenyls (PCB).

Avian cholera (*Pasteurella multocida*) has been recognized as a cause of losses among wild waterfowl in California (Rosen and Bischoff, 1949; Rosen, 1971) and Texas (Quortrup *et al.*, 1946) since 1944. More recently the disease has been reported among waterfowl in both the Mississippi (Vaught *et al.*, 1967) and Atlantic Flyways (Gershman *et al.*, 1964; Klukas and Locke, 1970; Locke *et al.*, 1970). Carcasses of ducks that have died of avian cholera frequently are scavenged by gulls and other predators with subsequent transmission of the disease to the scavengers (Rosen, 1971). Bald eagles were recently reported to have contracted avian cholera in this manner in northern California (Rosen, 1972).

This paper reports a case of avian cholera in a bald eagle (*Haliaeetus leuco-cephalus*) from Ohio, which was submitted to the Patuxent Wildlife Research Center's laboratory in conjunction with a pesticide monitoring program (Dustman *et al.*, 1971). Of 180 bald eagles that have been autopsied at the Patuxent Wildlife Research Center since 1960, only three appeared to have been infected with avian cholera. Unfortunately, a complete bacteriological study was not done on the first two of these three. These were an adult female found dead in Otoe County, Nebraska, in February, 1965, and an adult female found in Missouri in 1967. The Ohio case reported here is apparently the first bacteriologically proven case of avian cholera in a bald eagle in the Midwest. The eagle, an adult male, was found in a sickened condition some 200 yards from a well-established eagle nest near Sandusky, Ohio, June 1969 by Mr. Thomas M. Stockdale. The eagle was taken indoors, but died before any treatment could be initiated. The carcass was frozen and subsequently shipped to our laboratory for autopsy and chemical analysis.

The eagle weighed 3.18 k (7 lbs.) and the breast muscles were reduced in size; the sternal keel was prominent and only small amounts of subcutaneous and abdominal fat were present. There was hemorrhage in the posterior portions of both lungs; ecchymotic hemorrhages were present in the liver, which weighed 96.5 gms. The spleen was enlarged, the cross section measuring $3.0 \times 2.5 \text{ cms}$. There was serosanguinous fluid in the pericardial sac, and scattered whitish necrotic foci were present in the myocardium. The kidneys were mottled with petechiae. The stomach was empty except for some unidentified fragments of green plant. There was an old, partially healed, penetration-type wound in the tip of the sternum.

Material from the liver and blood from the heart were streaked onto 5% sheep-blood agar plates. After 24 hours of incubation at 37° C, dewdrop-like, non-hemolytic colonies of a gram-negative cocco-bacillus appeared on both plates. On the liver plate, a second bacterium, a non-hemolytic streptococcus, was also isolated. This streptococcus was regarded as an agonal invader.

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The gram-negative bacterium was found to be a cocco-bacillus, which in many cases appeared to form short chains. The organism produced acid but no gas in dextrose, mannitol, sucrose, fructose, sorbitol, galactose, mannose, and xylose. No acid was produced in lactose, maltose, arabinose, salicin, or erythritol. The isolate was indole-positive, produced no change in litmus milk, and formed nitrites from nitrates. On the basis of these morphologic and biochemical characteristics, the bacterium was identified tentatively as *Pasteurella multocida*. A culture of this isolate was submitted to the National Animal Disease Laboratory, Ames, Iowa, where its identity as *Pasteurella multocida* was confirmed.

Upon the completion of the necropsy, the carcass remainders (carcass except for skin, feet, wings, liver, and gastrointestinal tract) and the brain were analyzed for organochlorine pesticide residues according to Mulhern *et al.*, 1970. Analysis for polychlorinated biphenyls (PCB) was by thin-layer chromatography as described by Mulhern *et al.*, 1971. Mercury analysis was for total mercury by atomic absorption spectrophotometry. In this procedure, the sample is digested by refluxing with sulfuric and nitric acid, and is oxidized with hydrogen peroxide. Hydroxylamine hydrochloride and stannous chloride are added to the digest to reduce the mercury (II) ions to mercury metal. The sample is aerated, and the mercury measured in the air stream passing through a gas cell. The results of this chemical analysis, which are summarized in Table 1, show that all values are well below the lethal levels which have been reported in the literature (Stickel *et al.*, 1969; Stickel *et al.*, 1970).

Chemical	Parts per million, wet weight	
	Carcass	Brain
p,p'-DDE p,p'-DDD p,p'-DDT Dieldrin Heptachlor Epoxide Dichlorobenzophenone Polychlorinated Biphenyls Mercury	$12.4 \\ 4.8 \\ 0.2 \\ 3.3 \\ 0.3 \\ 0.8 \\ 160.0 \\ 1.8$	$ \begin{array}{r} 10.7 \\ 0.4 \\ \hline 1.2 \\ 1.1 \\ 0.4 \\ 40.0 \\ N.D.* \end{array} $

 TABLE 1

 Chemical analysis results on bald eagle with avian cholera

*N.D.=not determined.

Bald eagles in the Upper Mississippi and Ohio River Valleys feed to a large extent on sick or hunter-crippled waterfowl. If extensive outbreaks of avian cholera, such as have been reported in California and more recently on the Chesakepake Bay, occurred in the Mississippi and Ohio areas, the disease could pose a serious threat to the bald eagles there. To date there have been no reported outbreaks of avian cholera among waterfowl in Ohio; however the disease has been reported in waterfowl in Missouri and Tennessee, and recently Locke and Banks (1972) reported an outbreak which occurred among cedar waxwings at Steubenville, Ohio, during June 1968. There is no evidence to suggest that this bald eagle case is related to this localized outbreak among cedar waxwings. The source of this eagle infection is unknown.

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