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AN EPIZOOTIC OF AVIAN BOTULISM IN A PHOSPHATE MINE SETTLING POND IN NORTHERN FLORIDA

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Abstract: Type C botulism was determined to be the cause of an epizootic among waterfowl and shorebirds in a phosphate mine settling pond in northern Florida during May and June of 1979. Several hundred birds, the most common of which were American coots (Fulica americana), wood ducks (Aix sponsa), common gallinules (Gallinula chloropus), and northern shovelers (Anas clypeata) were afflicted over about a three-week period. A second smaller outbreak occurred in the same pond in early December of 1979. This is apparently the first time that botulism has been reported in waterbirds of Florida.

INTRODUCTION

Outbreaks of botulism have resulted in massive losses among waterfowl and other wild birds.^{4,7} The disease is especially common in western North America, but also has been reported from Central America, South America, Europe, and South Africa.⁸ Few reports of outbreaks in the southeastern United States have been found, although Kalmbach and Gunderson⁵ and Richardson *et al.*,⁶ reported the disease in Virginia and Georgia, respectively. The present paper is concerned with two outbreaks of type C botulism in waterfowl and shorebirds in northern Florida.

HISTORY

On 27 May 1979 four wood ducks (Aix sponsa) were found dead on a phosphate mine settling pond in Hamilton County, northern Florida. Three of these were hens found on eggs in artificial nesting boxes, the fourth was a drake found in the shallow waters of the pond. The ducks had been dead for several days and were moderately decomposed, but at necropsy were found to contain large numbers of maggots in their proventriculi and gizzards. The total body weights, condition of the pectoral muscles and the presence of body fat indicated that the birds were in good flesh and that the disease was acute. On 30 May and 1 June additional dead and moribund birds were found, the majority of which were American coots (Fulica americana), wood ducks (Aix sponsa), common gallinules (Gallinula chloropus), northern shovelers (Anas clypeata), and blue-winged teal (Anas discors) (Table 1). A number of the moribund birds had partial paralysis of their wings and legs, limberneck, and swollen lower eyelids. Many of the carcasses were floating in shallow water and contained hundreds of maggots. The area where the

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TABLE 1. Species of waterfowl and shorebirds found dead or moribund during May and June, 1979, in northern Florida.

Species	Number
American Coot (Fulica americana)	48
Wood duck (Aix sponsa)	26
Common gallinule (Gallinula chloropus)	13
Northern shoveler (Anas clypeata)	9
Blue-winged teal (Anas discors)	6
Ring-necked duck (Aythya collaris)	3
Black-necked stilt (Himantopus mexicanus)	2
Snowy egret (Egretta thula)	1
Semipalmated sandpiper (Calidris pusillus)	1
Mallard (Anas platyrhynchos)	1
TOTAL	110

afflicted birds were found was part of a larger impoundment of about 100 ha in size and was very shallow (no greater than 0.8 m at the deepest point and often much less) and contained gradual sloping edges. In some areas the pond bottom was completely exposed. Emergent vegetation was extensive and covered approximately 40% of the surface of the pond several months prior to the outbreak. At the time of the mortality the vegetation had been reduced by flooding to about 5% of the pond's surface. This vegetation was dominated by cattails (Typha sp.) and primrose willow (Ludwigia peruviana). Air temperatures were high during this period, often reaching up to 30 C. The above observations suggested that botulism was the cause of the morbidity and mortality. As a measure to control the disease, personnel of the mining company raised the water level on the pond. On 9 June only five dead wood ducks and one ring-necked duck were found and by 15 June, no further mortality was observed. The outbreak involved an estimated several hundred birds over the three-week period.

During early December, a second outbreak occurred on the same area as the first outbreak. Small numbers of moribund and dead coots and a few northern shovelers were involved. Signs were similar in the afflicted birds. Ambient temperatures were abnormally high for that time of year.

In the above outbreaks the numbers of afflicted birds appeared to be roughly proportional to the numbers of birds at risk, i.e. there did not seem to be differences in species susceptibility.

PATHOLOGIC AND **MICROBIOLOGIC FINDINGS**

Necropsies were performed on 11 birds which were obtained alive and showed signs of botulism intoxication. These included three blue-winged teals, two common gallinules, two American coots, one ring-necked duck, two shovelers, and one wood duck.

All of the birds were in good body condition and contained large amounts of body fat. No gross lesions were seen in any of the birds. Specimens of liver were collected from each bird and cultured individually on McConkey's agar and on 5% sheep blood agar. Contents of the large intestine of each bird were cultured on McConkey's and Eosin-Methylene Blue (EMB) media and in Selenite enrichment medium. S No pathogenic bacteria were found.

⁵ Difco Laboratories, Detroit, Michigan, USA.

Parasitologic findings were unremarkable. No cases of heavy helminth parasitism were noted. The proventriculus of the ring-necked duck had a round nodule (10 mm in diameter) which contained four specimens of *Echinuria uncinata*. This bird also was infected with *Leucocytozoon simondi*. Two of the three blue-winged teals were infected with *Haemoproteus nettionis* and one with *Plasmodium vaughani*. No *Sarcocystis* infections were detected.

TOXICOLOGIC STUDIES

Contents of the gizzards of one of the affected wood ducks and one of the bluewinged teals were force-fed to two 2-dayold White Pekin ducklings. These two ducklings were observed for five days and never showed signs of intoxication.

Four other 2-day-old White Pekin ducklings each were force-fed 10 maggots (*Sarcophaga* sp.) from the decaying carcass of an American coot. All four of the ducklings showed signs of intoxication (ataxia, weakness, head bobbing, muscle spasms) and died within 2.5 to 6 h after feeding.

Serum samples from eight typically affected birds (a wood duck, a bluewinged teal, three shovelers, a ringnecked duck, an American coot, and a common gallinule) were utilized in the mouse-protection test to determine the presence of Clostridium botulinum toxins. Two of the shovelers were from the December outbreak; the other six ducks sampled were from the May-June outbreak. One ml of serum from each of the eight birds was mixed with 0.25 ml of Clostridium botulinum Type C monovalent antitoxin[®] and incubated in a 37 C water bath for 30 min. Two 20-30 gm Swiss Webster mice were each injected intraperitoneally with 0.5 ml of each of the serum-antitoxin mixtures and two additional mice received 0.5 ml of untreated serum as controls. All the mice receiving untreated serum from each of the eight birds underwent progressive paraylsis, respiratory distress, and death within 96 h. None of the mice receiving serum-antitoxin mixtures showed any signs of distress and all were alive seven days later.

DISCUSSION

The ecological conditions, the observed clinical signs in the affected birds and the results of the mouse-protection test described above, clearly indicated that the cause of these outbreaks was Type C botulism. To our knowledge, this is the first published report of avian botulism in Florida. In 1972 a botulismlike outbreak (enterotoxemia) in Lake Okeechobee was reported by Jasmin *et al.*,³ but was determined to be caused by *Clostridium perfringens* Type C. In that study, botulism was suspected, but mouse-protection tests proved otherwise.

Many of the ecological conditions usually accompanying avian botulism were associated with the present outbreak. One unique aspect of this case was the fact that it occurred in Florida. Other than reports from Georgia⁶ and Virginia,⁵ no published reports of botulism in the southeastern states have been found. Smith⁸ pointed out that botulism is rare in the tropics, but offered no explanation of this phenomenon. This distribution may be related to soil and water alkalinity. Many of the large epizootics have occurred in areas of alkaline waters in western North America,⁵ but the full significance of alkalinity to botulism outbreaks is not well understood.1

An additional unique feature of the outbreaks in question was their occurrence on a phosphate mine settling pond. These settling ponds are diked impoundments used for the retention and dewatering of a waste colloidal slurry formed during the physical separation of

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⁶ Center for Disease Control, Atlanta, Georgia, USA.

phosphate from the mined matrix. In Florida these ponds cover approximately 24,000 ha.² The solid portion of the slurry which is retained in the settling ponds is made up of fine-grain particles of phosphate, clay minerals, and quartz.9 These particles are known collectively as slimes and have high capacities for water adsorption and retention. This property causes the slimes to settle out of suspension and to dewater very slowly. Stabilization of a pond's substrate may take more than 20 years and during this time water levels may fluctuate widely (from 1.0 cm to 3.0 m) depending on the rates of inflow and drainage. Supernatant water is drawn off the pond surface periodically and occasionally the water is completely drawn off, exposing the

slimes and speeding up stabilization through evaporation. These drainage periods apparently enhance plant growth.¹⁰ This phenomenon of changing water levels and rich vegetation, in concert with other ecological conditions (high ambient temperatures, presence of susceptible birds) would lead to conditions favorable for a botulism outbreak. Once mortality began and the carcasses became fly-bown, maggots containing high levels of botulinum toxin could accelerate the epizootic. The above sequence of events seems to have occurred in the outbreaks reported herein and illustrates a potentially serious hazard which must be dealt with when phosphate mine settling ponds are managed for wildlife.

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