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AN OUTBREAK OF TYPE E BOTULISM AMONG COMMON LOONS (*GAVIA IMMER*) IN MICHIGAN'S UPPER PENINSULA

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ABSTRACT: An epizootic of type E botulism (*Clostridium botulinum*) occurred among common loons (*Gavia immer*) along the Lake Michigan shore of Michigan's Upper Peninsula (USA) during October and November 1983. An estimated 592 dead loons washed ashore along the Garden Peninsula. Type E botulinal toxin was demonstrated in blood samples and stomach contents of dead loons, and in samples of three species of dead fish found on the Lake Michigan shore. We suspect that loons acquired botulism by ingesting sick or dead fish containing type E toxin.

Key words: *Clostridium botulinum* type E, avian botulism, type E botulism, common loon, *Gavia immer*, Michigan, epizootic, field study.

INTRODUCTION

Epizootics of type E botulism (*Clostridium botulinum* type E) in birds were first reported in 1963 and 1964 along the southern shore of Lake Michigan in Michigan and Indiana (Herman, 1964; Kaufmann and Fay, 1964). Mortality occurred primarily in common loons (*Gavia immer*) and gulls (*Larus* spp.); an estimated 7,720 and 4,920 birds died in 1963 and 1964, respectively (Fay et al., 1965). Since then, sporadic outbreaks of type E botulism have been reported (Fay, 1966; Monheimer, 1968; Graikoski et al., 1970; Stuht et al., 1977; Brand et al., 1983; Haagsma, 1987). In contrast to type C botulism, that occurs in a wide variety of birds world-wide (Rosen, 1971; Jensen and Price, 1987), reports of type E botulism in birds are largely confined to the Great Lakes area, particularly Lake Michigan, and primarily involve common loons, gulls, and diving ducks (*Aythya* spp.). During October and November 1983, an outbreak of type E botulism in common loons in Lake Michigan, near Michigan's Upper Peninsula (USA), was investigated, and the results are reported here.

MATERIALS AND METHODS

Reports of several dead loons on the Lake Michigan shore along the Garden Peninsula of Michigan's Upper Peninsula (45°40′ to 46°00′N, 86°10′ to 86°30′W) were first received by the Michigan Department of Natural Resources (MDNR) on 10 October 1983. Further reports of sick and dead loons were received through 30 October 1983, with a peak in reports on 21 to 23 October. On 25 October 1983, 11 chilled loon carcasses were submitted to the MDNR's Rose Lake Wildlife Disease Laboratory (RLWDL, East Lansing, Michigan 48823, USA) for necropsy and laboratory studies. Following initial diagnosis of type E botulism by RLWDL, 15 additional loon carcasses were similarly examined at the U.S. Fish and Wildlife Service's National Wildlife Health Center (NWHC, Madison, Wisconsin 53711, USA). Lesions observed at necropsy were recorded. Bacteriologic (Lennette et al., 1985) and virologic (Hitchner et al., 1980) studies were performed on liver, spleen and intestines. Tests for type E toxin were performed on serum obtained from hearts of dead loons using the standard mouse toxicity test (Quortrup and Sudheimer, 1943), involving intraperitoneal injection of 0.5 ml of serum each into unprotected 20 g female Swiss white mice (strain ND/4, Harlan Sprague Dawley, Inc., Madison, Wisconsin 53711, USA), and into mice protected with 0.2 ml (2 IU) type E antitoxin (Centers for Disease Control, Atlanta, Georgia 30333, USA). The test was positive for botulinal toxin when unprotected mice became sick and died with characteristic signs of botulism (rapid or labored breathing, "wasp-waist" body form, lethargy and progressive limb paralysis), and mice protected with type E antitoxin survived. Loon livers were analyzed for heavy metals by atomic absorption spectrophotometry (Perkin-Elmer Model 303, Perkin-Elmer Instruments, Norwalk, Connecticut 06856, USA) at the Wisconsin Department of Trade and Consumer Protection, Central Animal Health Laboratory (Madison, Wisconsin 53705, USA). Brain tissues

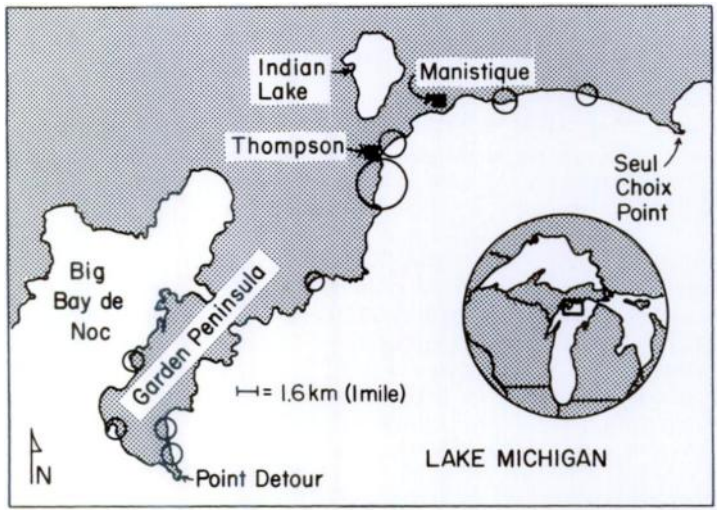

FIGURE 1. Map of the Garden Peninsula in the Upper Peninsula of Michigan, USA. Sections of Lake Michigan shoreline that were searched for loon carcasses are indicated by circles.

were assayed for organochlorine residues by gas chromatography (Hewlett-Packard Model 5840, Hewlett-Packard Co., Palo Alto, California 94303, USA) at the William C. Geagley Laboratory (Michigan Department of Agriculture, East Lansing, Michigan 48823, USA). Small fish and a crayfish (Cambarinae) found dead on the Lake Michigan shoreline and stomach contents of dead loons were processed using the extraction method described by Foster et al. (1974), and trypsinized with 0.05 ml of 1% trypsin (DIFCO Laboratories, Detroit, Michigan 48232, USA) in phosphate buffered saline/0.45 ml extract. The extract was then tested for botulinal toxin as described above. Weather data was obtained from the U.S. Forest Service (Hiawatha National Forest, Manistique, Michigan 49839, USA).

RESULTS

Field investigation

On 22 October 1983, 87 loon carcasses were counted by MDNR personnel on a 6.4 km section of shore south of Thompson (Fig. 1). Based on this and the distribution of reports, an estimated 300 loons had died by 22 October 1983.

On 1 to 2 November 1983, one of us (CJB) conducted searches on eight 1.6 km segments of shore, including two on the western shore of the Garden Peninsula, three on the eastern shore, and three on the southern shore of Lake Michigan between Thompson and Seul Choix Point (Fig. 1). Carcasses were not observed on the western shore of the Garden Peninsula. The numbers of carcasses found on each of the three segments on the eastern shore were eight, 16 and seven, respectively. On the three segments between Thompson and Seul Choix Point, one, five and five carcasses, respectively, were found. Many carcasses were partially buried in sand. One-third of the carcasses appeared fresh, and probably had died within the past 4 days. In addition, about one-third of the carcasses were partially scavenged. Canine (*Canis* spp.) and raccoon (*Procyon lotor*) tracks were abundant along the shore, suggesting that predation or scavenging had removed additional carcasses.

A mean of 6.5 ± 2.8 (± standard error) carcasses per km were found during 1 to 2 November on sampling sites along the 78 km eastern shoreline of the Garden Peninsula (from Point Detour to Thompson, Michigan) and 2.3 ± 1.3 per km on the 37 km section between Thompson and Seul Choix Point, Michigan. Thus, an estimated 592 ± 138 carcasses had washed ashore. An additional unknown number of carcasses might have been buried in sand or removed by predators or scavengers. Also, it is possible that additional loons died, but

drifted further into open water of Lake Michigan. We suspect that loons dying during this outbreak were from large rafts of birds reported by MDNR to be offshore to the southeast of the Garden Peninsula. Wind direction and velocity during the epizootic period were variable. Southerly winds would have blown carcasses to the Garden Peninsula area; however, wind direction during 7 of 18 days between 15 October and 1 November 1983 was northerly. Carcasses drifting to the south may have sunk, decomposed, or been scavenged before reaching the nearest shore. Thus, the estimated 592 loons is a minimum number.

Carcass examinations

Of the 15 loon carcasses examined at NWHC and eight at RLWDL, 11 (48%) were males; testis lengths ranged from 10 to 13 mm. Among the 12 females, 10 had well developed oviducts and follicles measuring up to 1 mm diameter; they were undeveloped in the remaining two. The bursa of Fabricius was not present in any of the 23 birds examined, indicating that they were after-hatching-year birds. Three distinct plumages were evident; however, we were unable to determine age by plumage characteristics described by Palmer (1962).

Type E botulinal toxin was detected in serum from seven of eight loons tested at NWHC, and in pooled samples from two and six loons tested at RLWDL. Tests for botulism were not performed on other loons examined because of poor post mortem condition. Stomachs of 16 of the 23 loons examined contained food items including fish bones and flesh, presumably of fish. Type E toxin was present in two of three pools of stomach contents (n = 7 birds). Toxin was not detected in stomach contents of a single loon whose serum was negative for the toxin. We do not know the cause of death in this bird, but it is possible that tests on blood and stomach contents provided a false negative diagnosis, since type E toxin is relatively unstable and may have been denatured by proteolytic enzymes produced by other organisms (Bott et al., 1966).

None of the 23 loons examined had lesions suggestive of other infectious, toxic or traumatic disease processes. Also, significant pathogens were not isolated from spleens (virology), livers (aerobic and anaerobic bacteria) or intestines (aerobic bacteria, including salmonellae). All examined loons appeared in good to excellent body condition based on fat deposits and muscle development. Several of the loons had wet and frothy lungs, suggesting that death was from drowning. This observation is consistent with botulism, since flaccid paralysis of neck muscles in birds sick from botulism makes them unable to hold the head above water (Rosen, 1971). Concentration (wet weight basis) of nine metals in livers were not within ranges known to be toxic in other birds, including arsenic (<0.5 ppm), thallium (<0.6 ppm), lead (<1.2 ppm), copper (4.0 ppm), and zinc (24.0 ppm); cadmium, chromium, and nickel were not detected. Total mercury concentrations ranged from 0.6 to 18.0 ppm, and were below levels associated with mercury intoxication (>20 ppm) in other birds (Fimreite, 1974). Residues of 1,1-dichloro-2,2-bis-(p-chlorophenyl)ethylene (DDE) and polychlorinated biphenyls (0.14 and 0.55 ppm, respectively) in brains of eight loons were also below toxic levels (Stickel et al., 1970; Eisler, 1986).

Botulinal toxin in fish and crayfish

Carcasses of ninespine stickleback (*Pungitius pungitius*), freshwater burbot (*Lota lota*), alewife (*Alosa pseudoharengus*), American smelt (*Osmerus mordax*), and a single unidentified crayfish found on the shore were tested for type E toxin. Toxin was detected in a single burbot and in separate pooled samples of alewife and smelt. Although such carcasses are commonly found along the shore of Lake Michigan, major concentrations were not observed,

and numbers of dead fish did not appear to be unusually large.

DISCUSSION

The consistent occurrence of type E toxin in blood from loons examined, and the lack of evidence for other mortality factors, strongly suggest that the majority of birds died from type E botulism. Although post mortem formation of toxin could have occurred in some carcasses, it is unlikely, since sick loons with signs of botulism were observed by MDNR. Mercury levels found in this study were not above toxic levels (20 ppm), and also were below the mean of 43.0 ppm in livers from five apparently healthy loons in northwestern Ontario (Fimreite, 1974). However, they were above those levels found in surveys of other fish-eating birds in Wisconsin (Kleinert and Degurse, 1972). Mercury intoxication was suspected to have contributed to emaciation in sick and dead loons found during a large-scale epizootic in Florida during winter 1983 (R. K. Stroud and R. E. Lange, NWHC, unpubl. report); 14 of 22 loons had liver mercury levels exceeding 20 ppm, while seven of the remaining eight had between 15 and 20 ppm. The significance of elevated mercury levels in loons examined in the present study is unknown, but mercury poisoning was not the likely cause of death. The source of mercury exposure and its effects in loons deserve further investigation.

Recurrent outbreaks of type E botulism in loons have been investigated since the mid-1960's (Fay et al., 1965; Fay, 1966; Monheimer, 1968; Brand et al., 1983), but little is known about the epizootiology of this disease. Spores of *Clostridium botulinum* type E are widely distributed in sediments and in the alimentary tracts of fish from the Great Lakes (Bott et al., 1966, 1968; Graikoski et al., 1970; Sugiyama et al., 1970). Monheimer (1968) and Graikoski et al. (1970) found type E toxin in fish carcasses from the Great Lakes, as we did. Brand et al. (1983) demonstrated the actual consumption by loons and gulls of fish that contained preformed toxin; however, it was not known whether these fish were alive or dead when they were ingested. Although gulls commonly scavenge fish carcasses and thus would be at high risk of ingesting toxin-laden carcasses, loons appear to feed entirely on living fish (Olson, 1951). It is possible that fish carcasses in the water could have been mistaken for live fish and consumed. It is possible also that fish eaten by loons may have been moribund from botulism, since outbreaks of type E in fish have been reported (Huss and Eskildsen, 1974; Haagsma, 1975; Cann and Taylor, 1982; Eklund et al., 1984).

Without a better understanding of the epizootiology of type E botulism in loons, control and prevention methods are difficult to recommend. Type E botulism in loons is not likely to be perpetuated by the carcass-maggot cycle, as with type C botulism in waterfowl. Type E outbreaks generally occur in cool fall weather (Brand et al., 1983) when fly infestation of carcasses is less likely. Also, healthy loons usually raft offshore in the fall and are not likely to be exposed to carcasses along the shore or to consume maggots or other invertebrates associated with carcasses. However, removal of carcasses is still recommended to prevent mortality in scavengers, particularly gulls, and because of the potential hazard to human health of type E toxin (Dolman and Iida, 1963).

The sporadic reports of epizootics of type E botulism suggest that factors determining toxin production or its availability to birds vary from year to year. However, it is also possible that mortality may occur more frequently than is reported. Carcasses may not be observed or reported in remote areas. In addition, mortality may be mistakenly attributed to other factors such as large-scale drowning in gill nets (Klein, 1985), or post mortem condition of carcasses examined may preclude accurate tests for type E botulism. Increased surveillance during fall staging on the Great Lakes, and comprehensive examination of

carcasses in fresh condition and of sick birds is recommended. Further investigations are needed to understand the conditions under which the toxin is elaborated in the Great Lakes ecosystem, the means by which it is made available to loons, and the impact of losses from type E botulism on loon populations.

Treatment of sick birds during outbreaks of type C botulism with type C antitoxin has been used successfully, although it is labor consumptive and costly (Rosen, 1971), and birds remain susceptible to subsequent intoxication. Use of type E antitoxin to rehabilitate loons sick from type E botulism also may be feasible, and perhaps warranted because of the apparent decline in status of loon populations and the current interest and public appeal of loons (Klein, 1985).

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