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AN OUTBREAK OF BOTULISM IN WILD WATERBIRDS IN SOUTHERN AUSTRALIA

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ABSTRACT: An outbreak of Type C botulism in waterbirds was investigated at Lake Lalbert in northwestern Victoria, Australia. Total losses were estimated at 1,500 birds, with 80% of affected birds being grey teal (*Anas gibberifrons*). The outbreak was associated with a falling water level, and extensive decomposition of plant material.

INTRODUCTION

Botulism is an important disease of wild waterbirds and is prevalent along the Murray River in some years (Pullar, 1934). It is recognized as a severe, frequently fatal intoxication caused by the ingestion of the preformed neurotoxin of *Clostridium botulinum*.

There are seven recognized types of *C. botulinum* labelled A to G. These are differentiated on the basis of the antigenicity of the neurotoxin produced (Smith, 1978; Wobeser, 1981). Botulism can occur in man (types A, B, E, F and G), farm animals (types A, B, C, and D) and wild animals and birds (type C and rarely type E) (Smith, 1977; Wobeser, 1981; Farrow et al., 1983).

This report describes an outbreak of avian botulism in northwestern Victoria during November and December 1983.

MATERIALS AND METHODS

Lake Lalbert is a shallow flat-bottomed lake located approximately 50 km south of Swan Hill in northwestern Victoria. During November–December 1983 it was approximately 750 ha in area with a depth of approximately 2 meters. Live trees extended from the edge of the lake for approximately 150 meters and there was a treeless central area. The lake had filled following heavy winter rainfall and flooding from local creeks. There was a floating mass of algae and rotting vegetation which extended 50–100 meters from the edge of the lake (Fig. 1).

In early December, Fisheries and Wildlife and Agriculture Department officers visited Lake Lalbert following a report from a local farmer of heavy losses of wild waterbirds. They found a number of carcasses and also birds affected with flaccid paralysis typical of botulism. On 6 December 1983 it was estimated that there were 700 dead and 300 clinically affected birds (a census was not possible because of the nature of the terrain and vegetation). From the degree of carcass decomposition it was estimated that the birds had been dead for up to 2 wk.

Approximately 80% of carcasses and affected birds were grey teal (*Anas gibberifrons*). Other species affected were Pacific black duck (*Anas superciliosa*), Australian little grebe (*Podiceps ruficollis*), swamphen (*Porphyrio porphyrio*) and black swan (*Cygnus atratus*).

Live and dead birds of all affected species were sent to the Regional Veterinary Laboratory, Bendigo, for post mortem examination. Sera from five clinically affected birds were collected (three grey teal, one grebe and one Pacific black duck) and 0.4 ml was inoculated intraperitoneally into pairs of mice, to test for the presence of *C. botulinum* toxin. Sera were mixed also with 5 IU of monospecific *C. botulinum* type C antitoxin prior to inoculation.

Daily maximum and minimum temperatures for the period 15 November 1983 to 22 December 1983 were recorded from the Swan Hill weather station (Fig. 2). Water temperature was measured on 22 December 1983.

RESULTS

Affected birds showed varying degrees of paralysis and paresis. Mildly affected birds were ataxic owing to involvement of legs and wings, but were able to move by propelling themselves with their wings.

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FIGURE 1. Lake Lalbert, a shallow flat bottomed lake with a floating mass of algae and other rotting vegetation in the foreground.

More severely affected birds had developed flaccid paralysis of the legs, wings, and neck. Terminally affected birds were totally paralyzed.

Gross post mortem examinations on 16 birds (5 live, 11 dead) revealed no lesions except for one Pacific black duck which had acute pneumonia. *Aeromonas hydrophila* and *Pasteurella* spp. were isolated from the lungs of this bird. All other bacteriologic tests were negative.

All pairs of mice inoculated with sera alone developed signs of botulism, including "hour glass" respiration, within 48 hr of inoculation. Mice inoculated with sera incubated with type C antitoxin remained normal, suggesting *C. botulinum* type C was the probable cause of the disease.

The average daily maximum and minimum temperatures for the period 15 November 1983 to 22 December 1983 were 26.7 C and 12.7 C, respectively. The water temperature was 27.5 C at the shallow edge of the lake and 26 C in the center, when measured at 11 A.M. on 22 December 1983.

The outbreak lasted until mid-December and estimated total losses were 1,500 waterbirds (80% grey teal).

DISCUSSION

Diagnosis of botulism is based on clinical and ecological evidence and confirmed by the demonstration of toxin in the serum of affected birds. The toxin causes blockade at the neuromuscular junction through interference with the release of acetylcholine (Wobeser, 1981). This blockade, when widespread, causes flaccid paralysis especially of the legs, wings, and neck.

Wobeser (1981) suggested two hypotheses regarding toxigenesis. The first, termed the "sludge-bed hypothesis," suggests that rotting organic material provides a suitable environment for toxigenesis providing there are suitable conditions of pH, salinity, and a depressed oxygen content. The second, termed the "microenvironment concept," stresses that toxigenesis is dependent upon the presence of animal tissue and that it occurs within carcasses such as those of small aquatic or terrestrial invertebrates. This hypothesis suggests that toxin is held within bacterial cells in these carcasses and that these carcasses act as the source of a botulism outbreak. Once an outbreak has begun the carcasses of affected birds become an important source of toxin through the intermediary of fly maggots and other invertebrate carrion feeders (Borland, 1976; Wobeser, 1981).

The outbreak of botulism described here was seen at a time when Lake Lalbert was beginning to dry up. Conditions present fitted both hypotheses. There were large quantities of rotting organic material

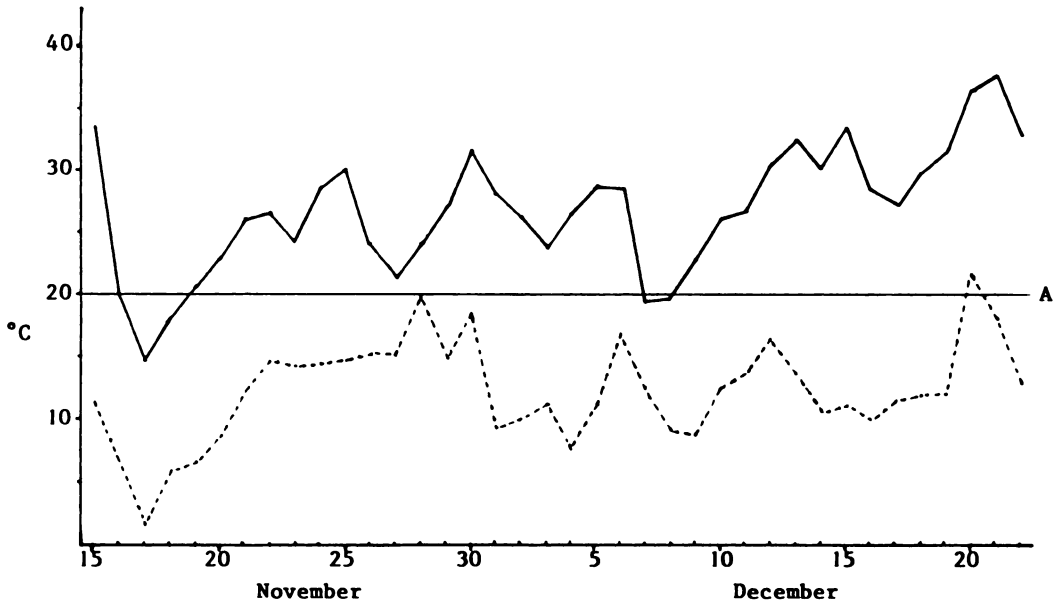

FIGURE 2. Daily maximum and minimum temperatures for the period 15 November 1983 to 22 December 1983 recorded at the Swan Hill weather station. (A is the temperature which Segner et al. (1971) suggested was necessary for botulism toxigenesis.)

around the edges of the lake (Fig. 1), and the falling water level and increasing water temperature meant that many small invertebrates would have died, thus providing a suitable environment for toxin production.

Segner et al. (1971) found that a temperature of 20–23 C was required for toxigenesis. Figure 2 shows that temperature in relation to the daily maximum and minimum temperatures recorded in nearby Swan Hill. Water temperatures measured on 22 December 1983 were above that needed for toxigenesis.

The high percentage of grey teal affected can be related to the fact that they were the predominant species on the lake and that they feed in the shallow (warmer) waters around the edges of the lake.

These findings are in agreement with Woodall (1982), who associated the apparent differing susceptibility of various avian species to botulism with their method of feeding. Grey teal strip seed from aquatic plants, filter surface water, and dredge mud in shallow water (Frith, 1977). Mud is recognized as a more favorable environment for *C. botulinum* than soil (Smith, 1978).

Wobeser (1981) outlined measures that can be used to minimize the probability of an outbreak of botulism. They include: 1) The construction of ponds with steep sides so that when the water level changes there is no flooding or exposure of large areas of land which can act as the focus of botulism toxigenesis. 2) Ponds should be permanent rather than seasonally flooded. 3) If flooding is unavoidable it should be done when the weather is cold and production of toxin less likely. 4) Regular surveillance to detect mortalities and remove carcasses (and therefore maggots) which could act as a further source of toxin.

These control measures are difficult, if not impossible, to implement in northwestern Victoria because: a) The lakes are shallow and any change in water level exposes or floods a large area of land which

is capable of acting as the focus of botulism toxigenesis. Normally the lakes fill during winter flooding and dry up during the hotter summer months (most botulism outbreaks are seen during the summer). b) Dense vegetation in the area means that carcass clean up and disposal is difficult. c) Water is a scarce commodity during the summer and often cannot be obtained to maintain a constant level in the shallow lakes. d) Such manipulations would drastically alter the existing ecosystem.

This outbreak, in common with many previously recorded outbreaks, featured a large expanse of shallow water, warm weather, fly activity, and large amounts of decaying vegetation. Though little can probably be done to alter the environment itself, in the future it may be possible, by closer monitoring of environmental conditions and bird densities, to take some control action such as the rapid removal of carcasses.

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