TYPE C BOTULISM AMONG WILD BIRDS
A Historical Sketch

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By

E. R. KALMBACH

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On the cover: A flock of mallards on a western wildlife refuge area.
(Photo by M. C. Hammond)

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TYPE C BOTULISM AMONG WILD BIRDS--A HISTORICAL SKETCH

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Abstract.--About 1910, notice began to be taken of a disease among waterfowl in the western U.S.; in subsequent years great numbers of waterfowl were lost to "western duck sickness". It was suggested that the disease might be a form of "alkaline poisoning," but eventually the cause was recognized as toxin of the bacterium Clostridium botulinum type C. This sketch includes a number of references.

In the summer of 1910 a mysterious malady, almost unnoticed before, took a disastrous toll among waterfowl in the western United States. D. H. Madsen, the Game and Fish Commissioner of Utah at that time, described the conditions he and his associates found at the mouths of the Jordan and Bear Rivers in the Salt Lake Valley that year (Madsen, 1929):

> Our efforts were confined to gathering all the dead ones, loading them in flat-bottom boats with pitchforks, and hauling them to the nearest land to be piled up and buried or burned . . . . I have seen many acres of water where these men could, within throwing distance, put in more than two hundred birds without moving the boat . . . . We spent days at this work, until the utter uselessness of it all became apparent. The margins of the ponds and lakes soon became dotted with mounds of dead birds resembling rat dens in a marsh.

More definite figures of losses came to light in the years immediately following that early outbreak. Alexander Wetmore, in his study of what was then referred to as "western duck sickness," reported that about 30,000 dead birds were picked up on the Weber River flats west of Ogden, Utah, in 1912, and that on the delta of the Bear River at the northern end of Great Salt Lake more than 44,000 were gathered and burned between August 22 and September 21 that year. In the following summer more than 46,000 were buried between September 7 and 26, and Wetmore added that "attempt was made to clean up only those birds that lay in the open. Consequently the figure given represented a small part, probably less than a fifth, of the total loss" (Wetmore, 1918).

It was this great mortality that first attracted attention to a disease that is today recognized as type C botulism (caused by toxin of the bacterium Clostridium botulinum type C), one of the most destructive natural agencies affecting wild waterfowl and shorebirds that dwell in, or migrate through, our Western States and the Prairie Provinces of Canada. Since its victims are primarily game birds, its history and characteristics are perhaps better known to wildlife biologists than to bacteriologists, and this is a primary reason for compiling this brief outline of its incidence and for giving some highlights of research related to it.

Note.--This paper, in slightly different form, was presented at a symposium on botulism in wildlife conducted during a meeting of the American Institute of Biological Sciences, University of Maryland, August 18, 1966. It is a sequel to a similar compilation published 14 years ago (Sciple, 1953) and makes reference to the incidence of the disease and research associated with it in recent years.

HISTORY OF INCIDENCE

In attempting to trace back through the years the occurrence of some phenomenon in wild creatures, one soon reaches a point of diminishing returns. In this case, most of the people who witnessed early outbreaks of avian botulism are no longer with us, and few made any records of what they saw. In the days of waterfowl abundance, even substantial losses may have been obscured by the sheer numbers of healthy birds that crowded the environment, or were ignored by the hunters, who were not forced to go to centers where outbreaks occurred in order to fill their bags. With this situation, outbreaks among wild birds can be traced back with reasonable validity only to about the turn of the century. Beyond that, the reports are increasingly vague, either because of the factors just mentioned, or perhaps because of the former absence or rarity of the disease in areas where it now may be expected. Of course, the early records are classified as avian botulism largely on the basis of the symptoms reported and the presence of environmental conditions characteristic of the disease. The diagnoses made at the time were varied, some based on highly suggestive evidence, others on pure surmise.

Although mortality had been observed in earlier years, it was in the period 1909 to 1913 that major attention was first drawn to duck sickness. It was then that heavy losses of waterfowl occurred in three widely separated localities in North America--on the deltas of rivers flowing into Great Salt Lake in Utah, at lakes in the southern part of the San Joaquin Valley in California, and in the Elfros region of Saskatchewan, 180 miles north of the U.S. border. The year 1910 was one of particularly severe losses. Those in Utah have been alluded to (Madsen, 1929). In California, mortality on several lakes in the San Joaquin Valley from 1908 to 1913 was reported by F. C. Clarke (1913). In Canada, C. C. Plummer recorded that "the first year I was there [at Elfros in 1910] the ducks died off in untold thousands." He added, "I am well within the truth and this, remember, was when we first noticed the disease" (Plummer, 1913).

Plummer's comment may be significant in view of the absence of any clear reports of the disease in earlier years at places where it later became a recurring event. Observant naturalists accompanied many of the early exploring, military, and geological expeditions into the western regions, some crossing the very areas where severe outbreaks of duck sickness were recorded later. Expeditions of Williamson in the Warner Valley and Klamath regions of Oregon, Stansbury in the Salt Lake Valley, Lewis and Clark in the upper Missouri River, and Fremont in southern Oregon and the Salt Lake Valley are cases in point. In the U.S. Army Medical Corps at the time were a number of keenly observant ornithologists, some stationed at points where duck sickness might have occurred; and there were the early venturesome naturalists who explored regions in the West--Coues, Bendire, Elliot, Ridgway, Merriam, and Henshaw, among others. So far I have been unable to discover in their reports any evidence of avian mortality that could be construed as botulism. A record of the death of western grebes at Owen's Lake, Calif., in June 1891 (Fisher, 1893) remains diagnostically doubtful.

No attempt will be made in this text to note all the points in North America where "duck sickness" (now diagnosed as botulism) has been reported. Comments will be confined to those that give a general indication of the increasing area where the malady has occurred, at times with marked severity. Wetmore (1918) reported losses in 1915 at Lake Bowdoin in Montana, in 1916 and 1917 in southeastern Oregon, and in 1918 in the Warner Valley in the same state. Kalmbach and Gunderson (1934) have mentioned the following noteworthy outbreaks of later years. In the 1920's several localities were added to the list in California and Oregon. There were reasonably valid reports from Nevada and from the Dakotas at points where no outbreaks had previously occurred within the memory of local residents. Minnesota, Nebraska, and Kansas contributed highly suggestive records, and one isolated case was demonstrated in Virginia. From Canada came reports of outbreaks at Lake Johnstone in Saskatchewan and Lake Newell in Alberta, and later at Whitewater Lake in Manitoba. In 1934 a malady suggestive of avian botulism was noted near Welstead Lake, north of the 58th parallel, the most northerly point at which it has been reported among wild birds in North America (Soper, 1934). Although reports

of outbreaks came from an increasing number of localities, none appeared as severe as those in the Salt Lake Valley in 1910-13. In general, outbreaks were most prevalent in areas of alkaline waters and were associated largely with the hot, dry days of late summer.

There is no intention in this sketch to convey the impression that there has been a progressive dispersal of the disease organism over the areas listed, even though something of the kind may have been possible through bird movements. More likely, environmental conditions favorable to toxin production became more common through the years. Whatever the reason, it is evident that the disease was being reported from places where it had not been noted before. Severity has varied greatly, and as recently as 1965, a year of abundant water in Utah, an estimated 20,000 waterfowl died on flooded terrain in Bear River Bay, an area outside the Federal Migratory Bird Refuge where there was no opportunity to manipulate the water flow to lessen the danger.

Mention may be made at this point of the records of botulism elsewhere in the world. In 1921, near San Vincente de Castillos, Uruguay, Wetmore (1921) encountered waterfowl suffering from symptoms similar to those he had studied in Utah. E. M. Pullar (1934) described botulism among waterfowl on the backwater of the Hume Reservoir on the Murray River in Victoria, Australia, and other investigators reported it in New South Wales, North Queensland, and Tasmania (Rose, 1935). Quite recently, W. B. Grubb (1964) identified the cause of avian mortality in Perth in western Australia as type C alpha botulism, the same form responsible for outbreaks in this country. Further review of the literature on avian diseases will doubtless disclose other foreign records. So we cannot claim that type C botulism in wild birds is strictly a North American phenomenon, although more of the pioneering research on it was done here than elsewhere.

HISTORY OF RESEARCH

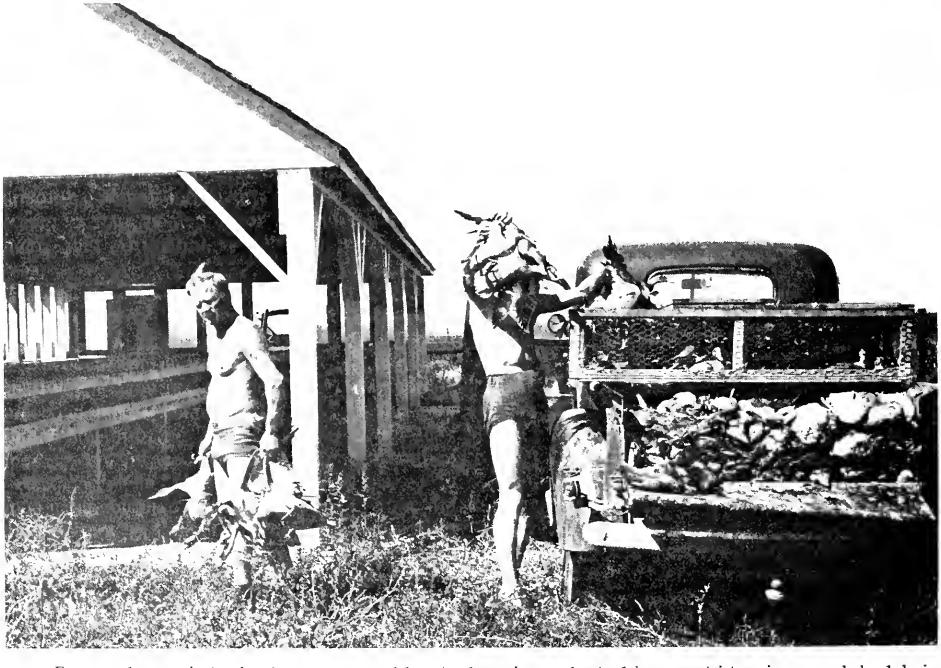
In reviewing the history of research in the diagnosis and control of type C botulism, comments will be restricted to that directly connected with wild birds. No attempt will be made to review the literature of type C botulism associated with domestic birds.

The first attempt to determine the cause of duck sickness was in 1911, when dead specimens from Utah were sent to the Bureau of Animal Industry in Washington, D.C. An early, tentative diagnosis of intestinal coccidiosis was later not substantiated (Wetmore, 1915), and in following years other theories were suggested, drawing for explanation on the whole gamut of avian diseases then known.

Aside from cursory inspections at areas where outbreaks occurred, the earliest field investigation of duck sickness among wild birds in this country was that carried out for the California Fish and Game Commission by F. C. Clarke and associates in the southern part of the San Joaquin Valley. They concluded that "the cause of the trouble lay in the lake water, either as a mineral or as an organic constituent" (Clarke, 1913). In July 1914 Wetmore began his studies for the Biological Survey at the mouth of Bear River at the northern end of Great Salt Lake in Utah. In a preliminary report (Wetmore, 1915), he concluded that "mortality results from alkaline poisoning, the exact nature of which is still to be determined." In the final report of the 3-year study (Wetmore, 1918), this idea was restated in expanded form. Wetmore was fully aware of the involvement of wind action in the flooding of mud flats and the increased mortality that resulted. In the light of knowledge then available, he considered this a method by which dense concentrations of soluble salts became available to puddling ducks, resulting in poisoning. It is also significant that he called attention to the paralysis of the nictitating membrane of the eye in affected birds--a symptom often appearing in cases of avian botulism. At that time only two types of botulism, A and B, were known, and neither was considered a threat to wild birds. Type C was not isolated and described until 5 years after Wetmore's final report.

In the years immediately following, no further serious research was undertaken on duck sickness in wild birds. In South Africa, however, much attention was given to study of "lamziekte" in domestic livestock, a closely related form of botulism (Theiler, 1920). In the early 1920's K. F. Meyer and associates at the Hooper Foundation for Medical Research contributed much to the literature of types A and B, and in 1923 Ida A. Bengtson (1923)

Bear River Refuge in the Utah marshes where western duck sickness was identified as botulism. Top right is a botulism area. Photo by Rex Gary Schmidt.



Formerly, sick ducks were collected, given botulism antitoxin, and held in pens until they recovered. Photo of Bureau biologists by W. F. Kubichek.

Typical scene of waterfowl dead and dying in the Bear River marshes. The duck at center, head in water, is characteristic. Photo by W. F. Kubichek.

Now, sick ducks are given antitoxin in the field and left to recover under the vegetation. More can be treated this way. Photo by Richard Gritman.

described from fly larvae the toxin-producing anaerobe that causes type C botulism. Its association with the "limberneck" of domestic poultry soon followed (Graham and Boughton, 1923), but its connection with duck sickness among wild birds was not established until nearly a decade later.

In the meantime, duck sickness was being encountered in environments less highly saline than those around Great Salt Lake. This led to doubts about the adequacy of the alkaline-poisoning theory, and demands gradually arose for reconsideration of the problem. This point was brought out by the field studies of J. A. Munro of the Canadian Wildlife Service in 1925 and 1926 at Lake Newell, Alberta, where the conditions necessary to support the idea of toxic salts were not in evidence (Munro, 1927).

Because of such misgivings, the Biological Survey resumed consideration of the problem, in a modest way, in the summer of 1927. C. C. Sperry was assigned to the work, which was conducted near Klamath Falls, Oreg. This was well within the range of western alkaline waters, but was less saline than the Salt Lake Valley. That summer's field work used several approaches, and one of them, the feeding of body tissues of affected birds to healthy ones, produced a highly significant result. A mallard duck given such treatment developed typical symptoms of duck sickness, probably the first experimental production of type C botulism in a captive wild bird. Despite continued experiments of this kind, no similar result was obtained, and its basic cause remained a mystery.

About this same time, studies on several aspects of saline toxicity were undertaken by P. A. Shaw under a cooperative project between the California Fish and Game Commission, the Hooper Foundation for Medical Research, and the Department of Pharmacology of the University of California Medical School. Although some symptoms suggestive of botulism were experimentally produced, the typical syndrome of duck sickness was not (Shaw, 1929).

The work begun by the Biological Survey at Klamath Falls in 1927 was discontinued in 1928 because of low incidence of the disease, but was resumed in 1929 and continued for 3 years, with additional technical assistance from the Bureau of Animal Industry, U.S. Department of Agriculture. The earlier concepts were reviewed and tested, including those of saline toxicity and the possibility of transmission through the feeding of body tissues. The possible role of parasites was also appraised by E. B. Cram, but no suggestive evidence was revealed. In 1930 J. F. Couch was assigned to assist in the continued search for possible leads in the field of toxic salts. At one point, as a result of inadequate refrigeration, a supply of body tissues (liver) suddenly developed a source of toxin which, when fed to gulls, produced the typical, long-sought-for syndrome (Kalmbach, 1930). The disease produced was identified as type C botulism by Giltner and Couch (1930), and from that point the direction of further research was clear. The presence of the bacterium and its preformed toxin was demonstrated under field conditions in a variety of organisms, mainly invertebrates. Many of these were acceptable food items to birds that customarily feed in shallow water or on mud flats (Kalmbach, 1932).

Supporting evidence came from other sources, including the Hooper Foundation for Medical Research, where M. Hobmaier, following the lead disclosed at Klamath Falls, demonstrated the presence of type C botulism in the bloodstream of afflicted wild birds (Hobmaier, 1930). Working independently, Gunnison and Coleman (1932) also confirmed the presence of type C in wild birds. At the conlusion of further studies in 1931, in which M. F. Gunderson contributed to the bacteriological aspects, the Biological Survey published a bulletin, "Western Duck Sickness, a Form of Botulism" (Kalmbach and Gunderson, 1934), summarizing the information available at that time.

During the following decade, a number of other investigators produced further confirming evidence. Shaw and Simpson (1936) found type C botulism to be the cause of waterfowl mortality at Stobart and Nemaka Lakes in Alberta; Pullar (1934) in Australia and Rose (1935) in New South Wales incriminated a closely related organism.

At the same time, other ideas were still being advanced and tested. Twomey and Twomey (1936) and Twomey et al. (1939) felt that selenium poisoning produced symptoms characteristic of duck sickness, though this was

later contested (Lakin et al., 1944). The possibility of toxic algae and even the inroads of leeches were given consideration at scattered points.

During the 1940's a sustained program of research on avian botulism was inaugurated at the Bear River Refuge in Utah, where the Biological Survey established laboratory and field facilities, and where research is still pursued by the Bureau's successor, the Bureau of Sport Fisheries and Wildlife of the Department of the Interior. In the course of time, nearly a score of investigators have been stationed there, and the total of their work has been extensive and varied. One of the important series of experiments revealed the effect of water manipulation on the incidence of the disease under field conditions, a prelude to possible remedial measures. It was demonstrated that dispersal of a thin sheet of water over flat lake beds or mud flats during midsummer was highly conducive to outbreaks, and that a program of progressively lowering the water levels to prevent such overflow materially reduced their likelihood. C. S. Williams, C. C. Sperry, and G. H. Jensen were leaders in this program, the results of which were summarized by Sperry (1947). Associated with the foregoing circumstances was the demonstration of a close relationship between the abundance of aquatic invertebrates and the incidence of botulism among wild fowl feeding on mud flats or flooded areas (Jensen and Allen, 1960). Another line of research demonstrated the value of administering type C antitoxin to affected birds, even to those completely prostrated by the disease. Where field conditions permit convenient and safe handling of the victims, such procedure is now considered appropriate and economically feasible.

From 1950 to 1955 the Microbiological Laboratory of the U.S. Public Health Service at Hamilton, Mont. and the Denver laboratory of the Fish and Wildlife Service collaborated in a program that joined technical knowledge and facilities of the Hamilton laboratory with the field experience of the Denver staff. The program included studies of the physiology of the bacterium, development of methods for detecting it and its toxin in the field, experimental production of toxic areas, and assembling of bibliographic information. Results of that program are filed at the Bear River Research Station, Brigham City, Utah, mainly in the form of processed documents.

With this, the historical sketch of important research on type C botulism in wild birds may properly be terminated, but it is obvious that the subject is far from closed. Recent work has opened a number of new fields of inquiry. For example, Fay (1966) has reported work incriminating type E botulism as the cause of mortality among loons, mergansers, and gulls in the Great Lakes region, and this finding raises the question how species that are not customarily carrion feeders ingest the preformed toxin. Jensen and Gritman (1966) have reported a definite potentiation of type C toxin in mallards and California gulls by addition of a strain of type E that alone is not toxic for these birds. Finally, F. G. Cooch of the Canadian Wildlife Service has studied the inhibiting effect of sublethal doses of botulism toxin on the functioning of the salt gland in waterfowl (Cooch, 1964). This leads one to the intriguing surmise that, since outbreaks of avian botulism among wild birds are often associated with saline areas, Wetmore's original theory of "alkaline poisoning" may again play some role in elucidating duck sickness.

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