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CONTROL OF AVIAN BOTULISM AT
JAMAICA BAY WILDLIFE REFUGE

FINAL REPORT

National Wildlife Health Laboratory
Madison, Wisconsin

GATEWAY INSTITUTE
FOR
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January 7, 1983

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BACKGROUND

Avian (type C) botulism is a paralytic disease of birds caused by the ingestion of toxin produced by the bacterium, Clostridium botulinum, (Rosen 1971). It has been recognized as an important disease of waterfowl and other waterbirds since the early 1900's (Giltner and Couch 1930, Hobmaier 1930, Kalmbach 1930), although massive waterfowl epizootics during the 1800's in the western United States reported as "western duck sickness" are now attributed to avian botulism. Most dramatic outbreaks of avian botulism still occur in the western states, where losses exceeding 100,000 birds in a single epizootic are not unusual (Friend and Brand 1980); however, the disease is now known to occur throughout North America and essentially world-wide (Rosen 1971, Allen and Wilson 1977, Wilson and Locke 1982).

Outbreaks of avian botulism in the New York Bight Region have occurred frequently since first reported in 1950 (Toth 1974), but losses are not as extensive as in the West. Outbreaks ranging from few birds to several hundred have been reported in New Jersey park ponds and marshes (Toth 1974, Figley and VanDruff 1982). Reilly and Boroff (1967) estimated that 1,000 waterbirds died from avian botulism during 1959 in a New Jersey tidal marsh. On Long Island, an outbreak of avian botulism at Breezy Point in 1978 killed about 100 terns, and annual outbreaks attributed to avian botulism involving up to several hundred waterbirds have occurred at Jamaica Bay Wildlife Refuge (JBWR) since 1975 (National Park Service [NPS], unpublished records). Although magnitudes of mortality from avian botulism in the Bight Region are relatively small, the close proximity of the outbreaks to millions of urban dwellers has resulted in considerable public concern over the losses and a need to address solutions to these losses.

During 1980, a two-year cooperative agreement between the NPS and the U. S. Fish and Wildlife Service (FWS) was established to develop an understanding of avian botulism at JBWR, and to develop control and management strategies for this disease. Specifically, the primary objectives of the agreement involved:

- A. Preparing a comprehensive bibliography on avian botulism.
- B. Providing an on-site workshop for training NPS personnel in the identification and control of avian botulism.
- C. Conducting limnological studies of West Pond.
- D. Conducting field studies of sites of botulinal toxin production and modes of toxin ingestion.
- E. Developing guidelines for the management of avian botulism outbreaks at JBWR.
- F. Developing recommendations for the prevention and control of avian botulism outbreaks at JBWR.
- G. Developing interpretive information on avian botulism suitable for public information displays.

This report summarizes accomplishments under the above seven objectives.

ACCOMPLISHMENTS

A. Preparation of a comprehensive bibliography on avian botulism.

Progress: The bibliography was completed and published during 1982 (Appendix A):

Wilson, S.S., and L.N. Locke, 1982. Bibliography of references to avian botulism: update. U.S. Dept. of Interior, Fish Wildl. Serv., Spec. Sci. Rep. 243. 10pp.

B. Provide an on-site workshop for training NPS personnel in the identification and control of avian botulism.

Progress: The National Wildlife Health Laboratory (NWHL) is prepared to conduct the workshop, and will do so following establishment of mutually suitable dates

C. Limnology of West Pond

Progress: Limnological studies of both East and West Ponds were conducted during 1981 by the New York Cooperative Fishery Research Unit (CFRU), Cornell University; and during 1982 by JBWR personnel and by Eco-Test Laboratories, Farmingdale, NY.

Water quality was assessed weekly during June 11 - August 25, 1981 at 3 sites on East Pond and 1 on West Pond; water samples were collected over the depths at each site. Water quality parameters measured included temperature, pH, dissolved oxygen, hardness, total alkalinity, and conductivity (Standard Methods 1976). A Secchi disc was used to index turbidity. In addition to weekly assessments, diel changes in water quality were measured at each site for one 24-hour period in June, July, and August. Aquatic invertebrates were collected at sampling sites during July and August; preserved samples are currently held at the CFRU. Duplicate invertebrate samples were also collected, frozen immediately, and later tested for the presence of botulinal toxin (discussed later). Results of water quality studies during 1981 are included in Appendix B.

During June 15 - September 14, 1982, water quality was assessed at West Pond and at 2 sites on East Pond (Appendix C). Invertebrate sampling was not conducted during 1982, except for collections of dead and living invertebrates observed along shorelines, which were tested for botulinal toxin. During August and September, 1982, water samples were also analyzed for chemical parameters (Appendix D), including total phosphate, nitrate, ammonia, total organic carbon, calcium, Kjeldahl nitrogen, and chloride. Algae samples were collected weekly and preserved in 5% formalin for later identification by NPS; duplicate algae samples were frozen and will be tested for algal toxin at NWHL in the event toxigenic species of algae are identified.

D. Sites of toxin production and mode of toxin ingestion.

Progress: Field studies were conducted during 1981 and 1982 to describe epizootiological patterns of avian botulism and sites of botulinal toxin production at JBWR to provide a basis for management and control strategies.

METHODS OF STUDY

Retrospective Studies of Mortality. NPS records of avian mortality during 1974-1980 were examined to provide perspectives of historical disease problems at JBWR.

Distribution and Causes of Mortality. Bird carcasses and carcass remains (scientific names listed in Appendix E) were collected during searches on East and West Ponds and on surrounding islands in Jamaica Bay. Searches of ponds were conducted 2 to 5 times per week, while islands were searched an average of once per week. Moribund birds were also captured; blood samples were taken from these birds and they were humanely killed. Although we do not know what percentages of total morbidity and mortality on the ponds were found during these searches, the numbers found can be used to reflect temporal trends in mortality, and serve as minimum mortality figures over the period of study. Undoubtedly, some carcasses were not found because predators or scavengers removed them, or because they died in dense stands of phragmites and were not detected. The magnitude and trends of mortality on islands could not be assessed from carcasses found because of infrequency of visits and rapid scavenging, decomposition, and disappearance of carcasses; however, subjective assessments were made during each visit of the magnitude of mortality occurring since the previous visit.

Dead birds were either necropsied by NWHL personnel at JBWR, submitted to NWHL for necropsy, or disposed of by burial; fresher specimens and euthanized birds were preferentially retained for necropsy. In this report, morbidity and mortality data are combined, unless otherwise specified.

Determination of causes of mortality were based on a wide variety of diagnostic studies, as follows:

Bacteriology - Determination of avian botulism was based on standard mouse protection tests (Quortrup and Sudheimer 1943). Blood serum from moribund birds and heart blood from dead birds were injected (intraperitoneally) into mice protected by type C botulinal anti-toxin and into unprotected mice; antibiotics were administered to prevent bacterial infections. Survival of protected mice and death of unprotected mice with signs characteristic of botulism provided a positive diagnosis. Other bacterial and fungal studies were conducted on a routine basis and on selected pathologic tissues using standard culture methods. Isolates of Salmonella spp. were identified by serotype at the Wisconsin State Laboratory of Hygiene.

Virology - Selected pathologic tissues and routinely collected cloacal and tracheal swabs were studied for evidence of viral infection using standard methods of preparation. Materials were inoculated into duck embryo cells, incubated at 40°C in 2% CO₂, and observed for cytopathic effect for 7 days; if no cell death occurred, media was blind-passaged and observed for an additional 7 days. Prepared samples were also inoculated into chicken eggs and observed for embryo mortality.

Parasitology - Parasites were prepared for identification using methods standard for each parasite group. Wet smears of intestinal contents were examined at 1000x using a light microscope. Trematode and cestode parasites were stained in Semichon's carmine, dehydrated, and mounted for identification. Nematodes were cleared in glycerine. Standard histologic studies also provided evidence of parasite infestation and pathologic responses to parasitism. In this report, parasitism is included as a diagnosis only when observed numbers of parasites were believed to be excessive, or when pathology was associated with the infestation.

Histology - Routine and pathologic tissues were collected and preserved at necropsy, and sectioned and stained with hematoxylin and eosin for histologic studies. Selected tissues were also treated with Ziehl-Nielson acid fast, Gomori's methenamine silver, Brown and Brenn, Mallory's trichrome, and Benhold's Congo red stains for further characterization of lesions or pathogens present.

Toxicology - Selected carcasses were prepared for toxicologic studies at Patuxent Wildlife Research Center (FWS). Organochlorine residues in carcasses will be determined by gas chromatography. Liver tissues will be analyzed by atomic absorption spectrophotometry for levels of 6 heavy metals: mercury, lead, nickel, cadmium, vanadium, and chromium. These studies are not yet completed, and will be reported when finished.

Demonstration of Botulinal Toxin in Environmental Samples. Environmental samples (including aquatic vegetation, aquatic sediments, fish carcasses, benthic and littoral invertebrates, and fly maggots and invertebrates collected from bird carcasses) were tested for the presence of botulinal toxin. Samples were collected in sterile plastic bags or jars and immediately frozen. Samples were processed using the extraction method described by Foster et al. (1974). The presence of botulinal toxin in the extract was determined by intraperitoneal injection into mice, as previously described.

RESULTS

Historical Perspectives of Avian Mortality at JBWR: 1974-1980. Prior to 1974, reports of avian mortality at JBWR are incomplete and anecdotal. Available NPS records of losses from 1974-1980 were examined.

During 1974, a total of 299 birds were found dead or moribund during August 1-14, including 166 (56%) mallards and 71 (24%) black

ducks. Avian botulism was diagnosed by Cornell University Duck Research Laboratory (DRL) among an unknown number of birds examined from West Pond. It was speculated that artificial lowering of the pond level, hot weather, and algae buildup contributed to the outbreak.

In 1975, about 200 dead birds were found during late July - mid-September on West Pond. Mortality was first noted in gulls; gulls accounted for most of the known mortality. Avian botulism was diagnosed by the DRL in 1 moribund mallard found on August 28.

During 1976-78 and 1980, only small numbers of birds were reported dead (24-46) during August and September. Avian botulism was not confirmed in any of these carcasses, although NWHL confirmed avian botulism in a die-off of about 100 common terns at nearby Breezy Point during early August of 1978.

Avian mortality in 1979 was reported from the week of July 7 through October 4, with a total of 392 dead and moribund birds collected. Gulls comprised 295 (75%) of the birds found, of which 246 were immature herring gulls (D. Riepe, unpublished report). Waterfowl and shorebirds accounted for 17% and 7% of the carcasses, respectively. The die-off occurred on both East and West Ponds; morbidity and mortality curves (weekly pickup) were similar between the 2 ponds (Fig. 1). Moribund birds exhibited clinical signs consistent with avian botulism; however botulinal toxin was not demonstrated in blood samples from 2 mallards examined at NWHL.

Magnitude and Distribution of Mortality. During June 4 - October 19, 1981, a total of 279 moribund and dead birds were found on East and West Ponds (Table 1). Although mortality occurred throughout the summer on both ponds (Figure 2), 250 (90%) of the losses occurred on East Pond. Weekly mortality figures on East Pond depict an apparent epizootic curve during early August to mid-October, reaching a peak in mid-September; increased mortality during the period occurred primarily in gulls. Mortality in waterfowl and other birds occurred at fairly constant levels on both ponds throughout the 1981 period of study.

Total mortality on East and West Ponds during 1982 (Table 2) was similar to 1981 (268 vs 279 birds); however, gulls comprised a smaller percentage during 1982 (64%) than during 1981 (82%), while waterfowl increased from 10% in 1981 to 24% in 1982. Temporal patterns of mortality in 1982 (Figure 3) were similar to 1981, where mortality in gulls increased on East Pond during late August to mid-October and mortality in waterfowl and other birds was constant.

Islands in Jamaica Bay which were searched included those on which gulls or other waterbirds nested, or where birds congregated in large numbers (Figure 4). Although we could not assess the magnitude of mortality on these islands, about 250 dead herring gulls were found on Ruffle Bar and 125 on Canarsie Pol on July 12, 1981. Almost all of the birds appeared to be about 2 weeks pre-fledgling, and had apparently died 7-10 days earlier. Postmortem autolysis of the carcasses precluded necropsies of these birds. No other evidence of large scale mortality was observed on the islands during 1981 and 1982, but low-level mortality

was noted throughout the summer at all sites. A total of 72 moribund and fresh dead birds, including 69 gulls, were collected each year from the islands (Tables 1 and 2) and submitted for necropsy.

Causes of Mortality. Tables 3 and 4 summarize diagnoses among 144 birds necropsied during 1981 and 135 during 1982, respectively.

Avian Botulism. Avian botulism was diagnosed in 33 (23%) of 144 birds examined during 1981 and 26 (19%) of 135 examined during 1982. Gulls comprised 20 (61%) of the 33 cases during 1981 and 11 (42%) of the 26 cases during 1982. Seventeen and 12% of the total gulls examined during 1981 and 1982, respectively, were diagnosed as avian botulism, as opposed to almost 50% of total waterfowl during each year. Avian botulism was found in only 2 (14%) of the other waterbirds examined. Proportional mortality from avian botulism among waterfowl was thus 3-to 4-times that in gulls and other waterbirds.

The temporal distribution of diagnosed cases of avian botulism during 1981 (Figure 5) and 1982 (Figure 6) shows that mortality occurred throughout the summer from late May to mid-September. During 1981, avian botulism was first diagnosed in a gadwall from East Pond on June 5; mortality on West Pond was not detected until late July; gull mortality from avian botulism on islands in Jamaica Bay occurred at least through July and August of 1981. During 1982, mortality from avian botulism on the ponds and islands likewise occurred throughout the summer, from late May to mid-September.

The temporal distribution of diagnosed cases of avian botulism does not accurately reflect patterns of total mortality from avian botulism, because the proportion of carcasses found which were suitable for necropsy varied. To estimate total mortality from avian botulism and its temporal patterns, we calculated the percentage of carcasses necropsied which were positive for avian botulism for 3 major bird groups (gulls, waterfowl, and other waterbirds) and applied these percentages to total numbers of carcasses found in each bird group for weekly intervals during 1981 and 1982. These values (Figures 7 and 8) are estimates of weekly mortality from avian botulism; since they are based on numbers of carcasses found, they certainly represent minimum estimates.

During 1981 (Figure 7), estimates of weekly mortality from avian botulism on East and West Ponds showed low levels (about 2/week) during early June to August, followed by a 4-fold increase to a peak of about 8/week in mid-September. This increase was accounted for primarily by gulls; average weekly mortality among gulls increased significantly (ANOVA; $P < 0.001$) during each month. Weekly mortality from avian botulism among waterfowl and other waterbirds remained constant during each month ($P > 0.05$). Total estimated mortality from avian botulism during 1981 at JBWR was 55 birds (30 gulls, 12 waterfowl, and 13 other waterbirds).

Estimated mortality from avian botulism during 1982 (Figure 8) showed a similar increase in weekly mortality among gulls during August-September ($0.025 < P < 0.05$), and constant mortality over the summer

among waterfowl and other waterbirds ($P > 0.05$). Total estimated mortality from avian botulism during 1982 at JBWR was 49 birds (15 gulls, 29 waterfowl, and 5 other waterbirds).

Although increases in estimated losses from avian botulism at JBWR occurred in August and September during both years, it does not appear that the actual mortality rates increased during this period. Systematic population estimates for East and West Ponds were not conducted regularly during the summer, and observational data are crude and variable. However, subjective evaluations of numbers of birds on ponds during carcass searches showed a great increase in shorebird populations beginning in late July and August, and at least a 2- to 3-fold increase in gulls during this same interval. Thus, while estimated numbers of gull mortalities from avian botulism increased during late summer, the rates of mortality apparently did not increase, since gull population levels increased similarly. Mortality rates in shorebirds, in fact, appeared to decrease during this period.

Other Causes. Mortality factors other than avian botulism represented a wide variety of infectious and parasitic agents (Tables 3 and 4). Aspergillosis, a mycotic infection caused by Aspergillus spp., was in fact the most frequent diagnosis during both 1981 and 1982 (31% and 22%, respectively). All isolates from fungal lesions were A. fumigatus. This disease was manifested in gulls and waterfowl by a wide variety of lesions, ranging from chronic airsacculitis and serositis to acute disseminated aspergillosis. Estimated total mortality from aspergillosis, calculated as described for avian botulism, was 125 gulls and waterfowl during 1981 and 51 during 1982. During both 1981 and 1982, mortality from aspergillosis occurred sporadically from May to mid August, followed by an increase in occurrence from mid-August through September (Figure 9). As with avian botulism, we do not know whether this represents an increase in mortality rates, since gull populations increased during this period; however, the mean magnitude of change in occurrence of aspergillosis between May-July and August-September was much greater (20-fold) than that of avian botulism (2- to 3-fold).

Bacterial enteritis due to Salmonella spp. was diagnosed in gulls, waterfowl, and 1 black skimmer. The latter case represents the first occurrence of the disease in this species to our knowledge. Although salmonellae were isolated from only 5 (3%) of the 144 birds examined during 1981, as opposed to 21 (16%) of 135 during 1982, not all cases of enteritis observed during 1981 were cultured. A wide variety of serotypes were identified among the 26 isolates, including S. typhimurium (12), S. heidelberg (5), S. blockley (3), and 1 isolation each of S. manhattan, S. thompson, S. agona, S. infantis, S. stanleyville, as well as Arizona hinshawii. Salmonellae were also isolated from intestines of several gulls in which an enteritis was not observed. These isolates are not included in Tables 3 and 4, as they were apparently not manifested by clinical disease.

Parasite infestations were common in all species examined. Tables 3 and 4, however, include only those cases where parasite burdens were unusually high or where pathology associated with the infestation was observed. Trematodiasis was the most frequent parasitic disease observed,

manifested by enteritis, renal tubular adenitis, and hepatitis. Trematodes identified included Renicola sp., Stephanospora sp., Austroilharzia sp., and unidentified schistosomes. Significant gastro-intestinal (GI) nematodes and cestodes included Cosmocephalus sp., Capillaria sp., Tetrameres sp., Microtetrameres sp., Pectinospirura sp., and Diplostoma sp. The pentastome parasite, Reighardia sterna, was observed to cause airsacculitis. Protozoan infections included enteric and renal coccidiosis; and sarcocystis, which caused myopathy and cardiomyopathy.

We were unable to determine causes of mortality in 52(36%) of the 144 birds examined in 1981 and 35 (26%) of 135 in 1982; however, toxicological studies have not been completed for some birds examined during 1982.

Multiple Causes of Morbidity and Mortality. Many of the birds examined had multiple disease processes occurring simultaneously, as indicated in Tables 3 and 4. We examined the occurrence of multiple diagnoses in birds during 1982 to determine if associations existed between the various disease factors (Figure 10). Chi-square analyses were performed on all individual combinations of disease categories. Although no significant associations were found between individual categories of diagnoses in Figure 10, there was a significant association between the occurrence of aspergillosis and combined occurrence of parasitism ($\chi^2 = 10.61$; $P < 0.005$). Although these results do not imply cause and effect, they suggest that aspergillosis is more likely to be found in birds with parasitic diseases, and vice versa. The occurrence of avian botulism was not related to any of the disease categories examined. There were no cases of trauma or predation associated with any of the diagnosed infectious, parasitic, or toxic diseases.

Sources of Botulinal Toxin. We investigated the presence of botulinal toxin in environmental samples collected on East and West Ponds and in Jamaica Bay to identify possible sources of toxin to birds (Table 5). All samples were collected during weeks when mortality from avian botulism was occurring (Figures 5 and 6).

Botulinal toxin was not found in any of the samples of living or dead invertebrates, algae and vegetation, bottom sediments, or fish carcasses collected on East and West Pond. A pooled sample of sea lettuce (Ulva lactuca) collected from 3 sites at high-tide line on Jamaica Bay, adjacent to JBWR, was weakly positive for botulinal toxin on July 22, 1982; subsequent samples of sea lettuce were negative. Although toxin was present in the first sample of sea lettuce, we do not know where this toxin was produced.

During the week of July 18, 1982, large numbers of ostracods were observed along shorelines and stagnant pools of both East and West Ponds; their appearance occurred during a period when dissolved oxygen reached seasonal lows (0 to 1.8 and 4.0 to 4.2 mg O_2 /l in water columns, respectively; Appendix C), and water temperatures on West Pond were at seasonal highs (28.2 to 28.5° C). Samples of living ostracods and of sediments in drying pools where ostracods were dying were negative for botulinal toxin (Table 5). Although the estimated number of birds

dying from avian botulism increased about 3 - fold during this week (Figure 8), we could not attribute this mortality to botulinal toxin produced by either changes in water quality or appearance of ostracods along shorelines; likewise, linear correlation of water temperature and dissolved oxygen with estimated mortality from avian botulism over the summers of 1981 and 1982 did not disclose any significant relationships (range of r values was from 0.07 to 0.43; all $P > 0.1$).

Botulinal toxin was demonstrated in fly maggots collected from 2 carcasses (mallard and laughing gull) found dead on East Pond, and from a herring gull on Canarsie Pol. To further investigate avian carcasses as sources of botulinal toxin, we placed 8 fresh herring gull carcasses in wire cages on East and West Ponds, and tested invertebrates collected on these carcasses for the presence of botulinal toxin. Botulinal toxin was demonstrated in fly maggots from 3 of the 8 gull carcasses as early as 42.5 hours after the carcass was placed on the pond shoreline (Table 6). Two of these 3 gulls producing toxic maggots died from avian botulism; the other died from aspergillosis. Toxin was not found in other invertebrates (Coleoptera-Silphidae and Carabidae, and snails) found on the carcasses. We do not know whether toxin would have been found in invertebrates from other carcasses if they had been left in the field for longer periods of time; however, by 49 hours, most carcass flesh had been consumed by invertebrates. These results demonstrate that fly maggots from avian carcasses serve as a potential source of botulinal toxin, and that toxin-laden maggots can occur on carcasses of birds dying from causes other than avian botulism.

DISCUSSION

Effective management and control of avian botulism requires an understanding of the epizootiology of this disease to determine links in the disease cycle which can be broken. Despite years of research and observations on the epizootiology of avian botulism, its ecology is still not clearly defined (Moulton et al. 1976). Epizootiological studies of avian botulism have largely been done in the western U.S. (Kalmbach and Gunderson 1934, Bell et al. 1955, Hunter 1970, Duncan and Jensen 1976, Moulton et al. 1976), yet the world-wide occurrence of avian botulism in a wide variety of habitat, environmental, and climatic situations (viz., Reilly and Boroff 1967, Crisley et al. 1968, Smith 1979, Brand et al., in press) indicates that each outbreak may represent a unique set of conditions, which may require unique control and prevention activities. The purpose of field studies at JBWR was to better understand the epizootiology of avian botulism in order to recommend feasible control and management strategies.

During field investigations in 1981 and 1982, we have shown that avian botulism occurred throughout the summer months at low levels, both on JBWR and surrounding islands in Jamaica Bay. Although numbers of cases on JBWR increased during late summer, principally among gulls, this probably reflected an increase in population levels on the ponds rather than increased mortality rates from avian botulism. The increase in gull populations on JBWR likely represents post-nesting movements

of adults and recent fledglings. Mortality from avian botulism among waterfowl and shorebirds appeared constant over both summers. These observations indicate that botulinal toxin was continuously available to birds over a widespread area of Jamaica Bay.

The major source of toxin at JBWR during 1981 and 1982 appeared to be fly maggots associated with avian carcasses. Birds dying from avian botulism as well as other causes (aspergillosis) were shown to produce toxin-laden maggots. This relationship between toxic maggots and avian carcasses in initiating and perpetuating avian botulism outbreaks has been well established (Kalmbach and Gunderson 1934, Hunter 1970, Duncan and Jensen 1976, Malcolm 1982). Although the botulinal toxin found in sea lettuce offered a potential source for waterfowl, means of transmission to gulls and shorebirds is unknown. In addition, it is unlikely that the toxin was produced in the sea lettuce alone, without the presence of a proteinaceous microenvironment, such as invertebrate carcasses, to provide suitable conditions for toxigenesis (Bell et al. 1955). In contrast, consumption of maggots associated with avian carcasses by gulls, waterfowl, and shorebirds is a common occurrence (Hunter 1970, Duncan and Jensen 1976).

Since mortality from avian botulism was present throughout the summer on both ponds during 1981 and 1982, its occurrence could not be related to changes in any measured water quality parameters, and there were no apparent relationships between the magnitude of mortality and any of these parameters. In fact, the occurrence of avian botulism throughout Jamaica Bay suggests that the major sources of botulinal toxin occurred independent of any characteristics unique to either or both ponds. The appearance of large numbers of ostracods along shorelines and pools of the ponds during late July 1982 was the only evidence of changes in invertebrate population levels; such changes have been associated with avian botulism outbreaks in other studies (Jensen and Allen 1960); however, toxin was not found in any samples of living or dead ostracods at JBWR. In experimental studies in which environmental factors were manipulated in an attempt to precipitate an avian botulism outbreak, Moulton et al. (1976) likewise found that the presence of dead invertebrates was not always sufficient to explain how avian botulism is initiated in nature.

The apparent absence of epizootic mortality from avian botulism during 1981 and 1982 was in contrast to previous reports of outbreaks at JBWR. However, mortality reported during previous outbreaks (viz, 299 during 1974 and 392 during 1979) was not much greater than the total number of carcasses found at JBWR during 1981 (279) and 1982 (268). Diagnostic examinations during previous outbreaks were limited to several birds collected at single points in time; avian botulism was not diagnosed consistently in those birds examined. We suspect that during previous years, a large proportion of the mortality was actually due to a wide variety of causes, as we found during 1981 and 1982. This is supported by our observation of large increases in numbers of cases of aspergillosis during August - October, which corresponded to periods of peak mortality reported during previous years. It is also possible that mortality from avian botulism during 1981-1982 was low relative to other outbreak years because environmental

conditions were not conducive for initiation and perpetuation of epizootic mortality. Intensive carcass pickup at JBWR throughout summers of 1981 and 1982 associated with field studies removed large proportions of the food source for fly maggots, and may have been responsible for successfully breaking the maggot cycle of botulinal toxin production during these two years.

E. Guidelines for management of avian botulism outbreaks at JBWR.

Wobeser (1981) discussed 4 basic management strategies to control avian botulism outbreaks by preventing access by birds to sources of toxin. These measures included the following:

1. Drainage of area to discourage bird use. This control option is not feasible at JBWR because of inability to remove water rapidly and completely from East and West Ponds. Incomplete drawdown of ponds may serve to attract more waterbirds rather than deter them, and extensive drawdowns could possibly create suitable conditions for aquatic invertebrate die-offs with subsequent development of botulinal toxin under proper environmental conditions.
2. Dispersal of birds by herding or scaring techniques, or other active methods. This option is also not generally recommended for avian botulism control at JBWR. We have shown that mortality from avian botulism occurs widespread in Jamaica Bay. Dispersal of birds from East and West Ponds would not eliminate the availability of toxin sources to birds; the result would be that they would continue to obtain the toxin and die in areas which are not intensively monitored for mortality, and the maggot cycle of toxin production would be perpetuated. Individual situations may arise, however, where bird dispersal may be warranted to prevent massive disease losses. We recommend that contingencies be made by the NPS to have rapid access to such equipment as zonguns, shellcrackers, etc., in the event such a situation occurs.
3. Flooding area to render sources of toxin unavailable. The feasibility of this option is questionable for JBWR because of the current inability to pump large quantities of water rapidly to East and West Ponds; rapid influx of salt water may also cause invertebrate mortality, thus possibly creating new sources of toxin. This option also implies that sources of toxin can be made unavailable by flooding, but we have shown that sources are available throughout Jamaica Bay; and the availability of toxic maggots on East and West Ponds may not be related to water levels in the ponds.

We recommend that during avian botulism outbreaks at JBWR, water levels in both East and West Ponds be stabilized as much as possible through existing water control structures. In the event of drought or other water loss in the ponds, the capability of supplying fresh water to maintain levels should be developed and maintained.

4. Carcass removal and disposal to prevent buildup of toxic maggots. During field studies in 1981 and 1982, maggots from avian carcasses appeared to be the major source of toxin to birds at JBWR. We

recommend that rapid removal and disposal of all avian carcasses from East and West Ponds be done during May-October to remove food sources for fly maggots, thus breaking a major link in the cycle of avian botulism. Frequency of carcass searches should be every day, since toxic maggots can develop within 42 hours on avian carcasses. All carcasses, including skeletal remains and decomposed tissues, should be double-bagged in sturdy plastic bags and disposed. Since burning of carcasses is infeasible at JBWR, we recommend deep burial at a fenced site as far removed from the ponds and water table as possible. Burial should be deep enough to prevent avian and mammalian scavenging.

Mortality occurring throughout Jamaica Bay can contribute to avian botulism at JBWR. While it is infeasible to conduct thorough carcass searches throughout the Bay, we recommend frequent searches on the islands to monitor mortality. If significant mortality is found, intensive carcass removal can be implemented on a site-by-site basis.

Removal of carcasses is a disease control method which is applicable to many other diseases; its practice will help reduce mortality from other causes as well as from avian botulism, and provide for more aesthetic recreational use of JBWR and the Bay.

F. Developing recommendations for the prevention and control of avian botulism outbreaks at JBWR.

Preventative measures for avian botulism developed in the western U.S. have primarily involved water control and management, aimed at preventing conditions conducive for production and ingestion of botulinum toxin. Such measures have traditionally included: 1) land leveling to eliminate hummocks, 2) creating steep-sloped water edges to avoid feather edging and to create depths of at least 18 inches, 3) constructing contour levees and dikes to control water flow and flooding, and 4) removing carcasses and other organic debris supplying nutrients for fly larvae and other invertebrates (Rosen 1971). The first 3 measures apply to outbreaks due to toxin production in shallow, warm, anerobic environments caused by changes in water levels. This mode of toxin production was not demonstrated at JBWR during 1981 and 1982, although it cannot be ruled out as important during other years. The application of these measures at JBWR cannot then be guaranteed to prevent avian botulism outbreaks, although they would reduce the potential for toxin production. Their application would, however, reduce habitat for many of the resident and migratory shorebirds and other waterbirds which utilize JBWR. Large-scale alteration of habitat and of pond management at JBWR is therefore not recommended. The following preventative measures are recommended to prevent and control losses from avian botulism:

1. Maintain diligent carcass removal activities during May-October on the ponds, and as feasible on the islands, to reduce sources of nutrients for fly maggots and other invertebrates.
2. During drawdown and subsequent refilling of ponds for creation of shorebird habitat, maintain a constant and gradual change in water

levels to prevent rapid exposure or inundation of areas which could cause explosive die-offs of aquatic or terrestrial invertebrates. This would require development of the capacity to rapidly drain water in the event of a sudden deluge, and to supply fresh water in the event of drought or other water loss from the ponds.

3. Shallow stagnant pools created along the shorelines of the ponds by wind and wave action should be eliminated by filling or temporary channeling.
 4. Vegetation and organic matter, such as algal mats, deposited on shorelines of ponds should be removed during June-September to reduce the potential for botulinal toxin formation in dying aquatic invertebrates entrapped in such material.
 5. Contingency plans should be made to adequately deal with epizootics from any cause which may occur. Such plans should include strategies for communications, manpower, and equipment mobilization, and manipulation of populations and habitats. Examples of existing disease contingency plans and evaluation of written plans can be provided by the NWHL.
- G. Developing interpretive information on avian botulism suitable for public information displays.

Progress: A leaflet on avian botulism for public information and dissemination has been prepared and is currently undergoing editorial review within the FWS. This leaflet will be printed during FY-83.

LITERATURE CITED

- Allen, J.P., and S.S. Wilson. 1977. A bibliography of references to avian botulism. U.S. Fish and Wildlife Service Spec. Sci. Rep. - Wildl. 204. 6pp.
- Bell, J.F., G.W. Sciple, and A.A. Hubert. 1955. A microenvironment concept of the epizootiology of avian botulism. J. Wildl. Manage. 19(3): 352-357.
- Brand, C.J., R.M. Duncan, S.P. Garrow, D. Olson, and L.E. Schumann. In press. Waterbird mortality from botulism type E in Lake Michigan: an update.
- Crisley, F.D., V.R. Dowell, and R. Angelotti. 1968. Avian botulism in a mixed population of resident ducks in an urban river setting. Bull. Wildl. Dis. Assoc. 4(3): 70-77.
- Duncan, R.M., and W.I. Jensen. 1976. A relationship between avian carcasses and living invertebrates in the epizootiology of avian botulism. J. Wildl. Dis. 12: 116-126.
- Figley, W.K., and L.W. VanDruff. 1982. The ecology of urban mallards. Wildl. Manage. 81: 1-40.
- Foster, E.M., R.H. Diebel, C.L. Duncan, J.M. Goepfert, and H. Sugiyama. 1974. Bacterial food poisoning. Pp. 858-861 in Manual of clinical microbiology, second edition (E.H. Lennette, E.H. Spaulding, and J.P. Truant, eds.). Am. Soc. for Microbio. Washington, D.C.
- Friend, M., and C.J. Brand. 1980. Waterfowl diseases - historic and current perspectives. 42nd Midwest Wildlife Conference, St. Paul.
- Giltner, L.T., and J.F. Couch. 1930. Western duck sickness and botulism. Science 72 (1878): 660.
- Hobmaier, M. 1930. Duck disease caused by the poison of the Bacillus botulinus. Calif. Fish Game 16(4):285-286.
- Hunter, B.F. 1970. Ecology of waterfowl botulism toxin production. Trans. N. Am. Wildl. Nat. Resour. Conf. 35:64-72.
- Jensen, W.I., and J.P. Allen. 1960. A possible relationship between aquatic invertebrates and avian botulism. Trans. N. Am. Wildl. Nat. Resour. Conf. 25:171-180.
- Kalmbach, E.R. 1930. Western duck sickness produced experimentally. Science 72(1878):658-660.
- _____, and M.F. Gunderson. 1934. Western duck sickness, a form of botulism. U.S. Dep. Agric. Tech. Bull. 411. 81pp.

- Malcolm, J.M. 1982. Bird collisions with a power transmission line and their relation to botulism at a Montana wetland. *Wildl. Soc. Bull.* 10(4):297-304.
- Moulton, D.W., W.I. Jensen, and J.B. Low. 1976. Avian botulism epizootiology on sewage oxidation ponds in Utah. *J. Wildl. Manage.* 40(4): 735-742.
- Quortrup, E.R., and R.L. Sudheimer. 1943. Detection of botulinus toxin in the blood stream of wild ducks. *J. Am. Vet. Med. Assoc.* 102 (793): 264-266.
- Reilly, J.R., and D.A. Boroff. 1967. Botulism in a tidal estuary in New Jersey. *Bull. Wildl. Dis. Assoc.* 3(1): 26-29.
- Rosen, M.N. 1971. Botulism. Pp. 100-117 in *Infectious and parasitic diseases of wild birds* (J.W. Davis, R. Anderson, L. Karstad, and D.O. Trainer, eds). Iowa State Univ. Press, Ames, IA.
- Smith, L.P. 1979. The effect of weather on the incidence of botulism (*Clostridium botulinum*) in water fowl. *Agric. Meteorol.* 20(6):483-488.
- Standard Methods. 1975. Standard methods for the examination of water and wastewater, 14th edition. Amer. Public Health Assoc. Washington, D.C. 1193pp.
- Toth, S. 1974. Botulism (western duck sickness) and its effects on waterfowl in New Jersey. *N.J. Outdoors* 1(4): 12-14.
- Wilson, S.S., and L.N. Locke. 1982. Bibliography of references to avian botulism: update. U.S. Fish Wildl. Serv. Spec. Sci. Rep. - Wildl. 243. 10pp.
- Wobeser, G.A. 1981. Diseases of wild waterfowl. Plenum Press, N.Y. 300pp.

Table 1. Number of birds found moribund or dead at East and West Ponds in Jamaica Bay Wildlife Refuge and on surrounding islands during June-October 1981.

Species	Number of birds		
	East Pond	West Pond	Islands ^a
Herring gull	173	11	63
Laughing gull	15	1	0
Gr. black-backed gull	16	1	6
Ring-billed gull	13	0	0
Total gulls	217	13	69
Mallard	4	4	1
Black duck	4	1	1
Green-winged teal	1	2	0
Gadwall	1	0	0
White Pekin	1	0	0
Canada goose	3	4	0
Brant	0	3	0
Total waterfowl	14	14	2
Double-crested cormorant	2	1	1
Great blue heron	1	0	0
Black-crowned night heron	1	0	0
Glossy ibis	2	0	0
Semi-palmated plover	2	0	0
Black-bellied plover	2	0	0
Semi-palmated sandpiper	2	0	0
Black skimmer	6	0	0
Common tern	1	0	0
Common crow	0	1	0
Total	250	29	72

^a Includes only birds which were collected and submitted for necropsy.

Table 2. Number of birds found moribund or dead at East and West Ponds in Jamaica Bay Wildlife Refuge and on surrounding islands during May-October 1982.

Species	Number of birds		
	East Pond	West Pond	Islands ^a
Herring gull	104	31	59
Laughing gull	19	1	2
Gr. black-backed gull	5	2	7
Ring-billed gull	8	1	1

Total gulls	136	35	69
Mallard	8	15	0
Black duck	8	4	0
Green-winged teal	0	2	0
Blue-winged teal	3	0	0
Gadwall	4	3	0
White Pekin	1	0	0
Unid. duck	3	0	0
Canada goose	1	12	0
Brant	1	0	1

Total waterfowl	29	36	1
Double-crested cormorant	4	2	0
Snowy egret	1	0	0
Black-crowned night heron	1	1	0
Glossy ibis	0	1	0
Common gallinule	1	0	0
Semi-palmated plover	1	0	0
Black-bellied plover	1	0	0
Semi-palmated sandpiper	1	0	0
Least sandpiper	0	0	1
Willet	1	0	0
Lesser yellowlegs	1	0	0
Unid. shorebird	4	0	0
Common tern	2	0	0
Black skimmer	7	1	1
Rock dove	1	0	0
Mockingbird	0	1	0

Total collected	191	77	72

^a Includes only birds which were collected and submitted for necropsy.

Table 3. Summary of diagnoses among 144 moribund and dead birds found at Jamaica Bay Wildlife Refuge and surrounding islands during May-October 1981.

Species	Avian botulism	Asper- gillosis	Salmo- nellosis	Trema- todiasis	Enteric nematodiasis and cestodiasis	Trauma	Other	Unknown	No. birds examined
Herring gull	16	38	3	4	0	5	0	38	99 ^a
Gr. black-backed gull	0	4	1	0	0	0	0	3	8
Laughing gull	2	1	0	0	0	2	0	1	6
Ring-billed gull	2	1	0	0	0	0	0	2	5
Total gulls	20	44	4	4	0	7	0	44	118
Mallard	0	0	0	0	0	1	0	0	1
Black duck	2	0	0	0	0	1	1 ^b	0	4
Cadwall	1	0	0	0	0	0	0	0	1
Green-winged teal	1	0	0	0	0	0	0	0	1
Canada goose	1	0	0	0	1	0	0	2	4
Brant	1	0	0	0	0	0	1 ^c	1	2 ^a
Total waterfowl	6	0	0	0	1	2	2	3	13
Double-crested cormorant	1	0	0	0	0	0	0	0	1
Black-crowned night heron	0	0	0	0	0	0	0	1	1
Semi-palmated plover	1	0	0	0	0	0	0	1	2
Black-bellied plover	1	0	0	0	0	0	0	1	2
Semi-palmated sandpiper	2	0	0	0	0	0	0	0	2
Common tern	0	0	0	0	0	0	0	1	1
Black skimmer	2	0	1	0	0	0	0	0	3
Common crow	0	0	0	0	0	0	0	1	1
Total (Σ)	33(23)	44(31)	5(3)	4(3)	1(1)	9(6)	2(1)	52(36)	144

^a The row-total number of diagnoses exceeds the number of birds examined because multiple diagnoses were made for one or more individual birds.

^b Ruptured oviduct resulting in peritonitis.

^c Pericarditis of unknown bacterial etiology.

Table 4. Summary of diagnoses among 133 moribund and dead birds found at Jamaica Bay Wildlife Refuge and surrounding islands during May-October 1982.

Species	Enteric					Vermineous/ pneumonia/ airsacculitis	Parasitism of upper GI tract		Parasitic hepatitis	Other parasitism	Trauma and Predation	Unknown	No. birds examined	
	Avian botulism	Aspergillus	Salmonellosis	Trans-todiosis	Enteric nematodiasis and cestodiasis		Coccidiosis	Sarcocystis						
Herring gull	9	23	17	10	7	4	3	2	4	3	1 ^a	1	18	75
Gr. black-backed gull	0	2	2	1	0	1	0	0	1	2	1 ^b	0	1	6
Laughing gull	2	1	1	0	0	0	0	0	1	0	1 ^c	2	5	10
Ring-billed gull	0	0	0	1	0	1	0	0	0	0	1 ^d	0	1	3
Total gulls	11	26	20	12	7	6	3	2	6	7	4	3	25	94
Mallard	8	1	0	0	1	0	0	2	0	0	0	0	3	13
Black duck	2	0	0	1	0	0	0	0	0	0	0	0	1	3
White pelican	1	0	1	0	0	0	0	0	0	0	0	0	0	1
Gadwall	1	0	0	1	1	0	0	0	0	0	0	0	0	2
Green-winged teal	1	0	0	0	0	0	0	1	0	0	0	0	0	2
Blue-winged teal	0	1	0	1	0	0	0	0	0	0	0	0	0	2
Canada goose	0	1	0	0	0	0	1	0	0	0	0	1	0	3
Brant	0	1	0	0	0	0	0	0	0	0	0	0	1	2
Total waterfowl	13	4	1	3	2	0	1	3	0	0	0	1	5	27
Double-crested cormorant	0	0	0	0	0	0	0	0	1	0	0	0	0	1
Glossy ibis	1	0	0	0	0	0	0	0	1	0	0	0	0	1
Semi-palmated sandpiper	0	0	0	0	0	0	0	0	0	0	0	0	1	1
Willet	0	0	0	0	1	0	0	0	0	0	0	0	0	1
Common tern	0	0	0	0	0	0	0	0	0	0	0	1	1	2
Black skimmer	1	0	0	0	0	0	1	0	1	0	1 ^e	1	3	7
Rock dove	0	0	0	0	0	0	0	0	0	0	0	1	0	1
Total (Σ)	25(19)	30(22)	21(16)	15(11)	10(7)	6(4)	5(4)	5(4)	9(7)	7(5)	5(4)	7(5)	35(26)	135

^a Parasitic adenitis.

^b Acanthocephala infestation.

^c Vermineous enteritis with larval migrans.

^d Parasitic larval migrans.

^e Oral nematodiasis.

Table 5. Environmental samples collected to determine sources of botulinal toxin at Jamaica Bay Wildlife Refuge during 1981 and 1982.

Year	Sample description	Collection site	No. samples	Date collected	Botulinal toxin test results
1981	Living benthic invertebrates ^a	East Pond	2	28 July	negative
	Living benthic invertebrates ^a	West Pond	4	20-27 July	negative
	Algae mat	East Pond	1	20 July	negative
	Bottom sediment from stagnant pool	West Pond	1	14 August	negative
	Fly maggots from mallard carcass	East Pond	1	9 June	positive
	Fly maggots from laughing gull carcass	East Pond	1	14 July	positive
1982	Fish carcass	East Pond	1	28 June	negative
	Fly maggots from fish carcasses	East Pond	1	28 June	negative
			1	1 July	negative
	Bottom sediment from stagnant pool ^b	East Pond	3	29 June	negative
			3	22 July	negative
			3	24 August	negative
		West Pond	3	9 September	negative
			3	29 June	negative
			3	22 July	negative
			3	26 August	negative
			3	8 September	negative
	Fly maggots from herring gull carcass	Canarsie Pol	1	1 July	positive
	Dead annelid on shoreline	East Pond	1	22 July	negative
	Living ostracods from pond shoreline and stagnant pools ^b	East Pond	3	22 July	negative
		West Pond	3	22 July	negative
	Bottom sediment from pool at high tide ^b	Jamaica Bay	3	22 July	negative
	Sea lettuce at high tide line during low tide ^b	Jamaica Bay	3	22 July	positive
			21	24-26 August	negative
			3	8 September	negative

^aInvertebrate samples consisted primarily of chironomid larvae.

^bSamples were pooled into groups of 3 for testing; initial sampling site was selected randomly within a given habitat, and subsequent samples were taken at regular intervals along a transect.

Table 6. Botulinal toxin in invertebrates collected from fresh avian carcasses placed on East and West Ponds in relation to cause of death of bird and elapsed time from placement of carcass in field.

Carcass species	Cause of death	Elapsed time (hrs)	Invertebrate sample	Botulinal toxin test results
Herring gull	Aspergillosis ^a	21.5	Coleoptera	negative
		42.5	Fly maggots	positive
		48.5	Fly maggots	negative
Herring gull	Avian botulism	18.0	Coleoptera	negative
		42.5	Fly maggots	negative
		48.0	Fly maggots	negative
Herring gull	Avian botulism	18.0	Coleoptera	negative
		22.0	Coleoptera	negative
		43.5	Fly maggots	positive
		49.0	Fly maggots	positive
Herring gull	Avian botulism	42.5	Fly maggots	positive
		46.5	Fly maggots	positive
Herring gull	Aspergillosis ^a	17.5	Adult fly	negative
		42.5	Fly maggots	negative
		48.5	Fly maggots	negative
Herring gull	Unknown ^a	42.5	Adult fly	negative
		48.5	Fly maggots	inconclusive ^b
Herring gull	Avian botulism	18.0	Snails	negative
		43.5	Fly maggots	negative
		49.0	Fly maggots	negative
Herring gull	Avian botulism	18.0	Coleoptera	negative
		43.5	Snails	negative
		49.0	Fly maggots	negative

^a Botulinal toxin was not demonstrated in blood.

^b Both unprotected and protected mice died.

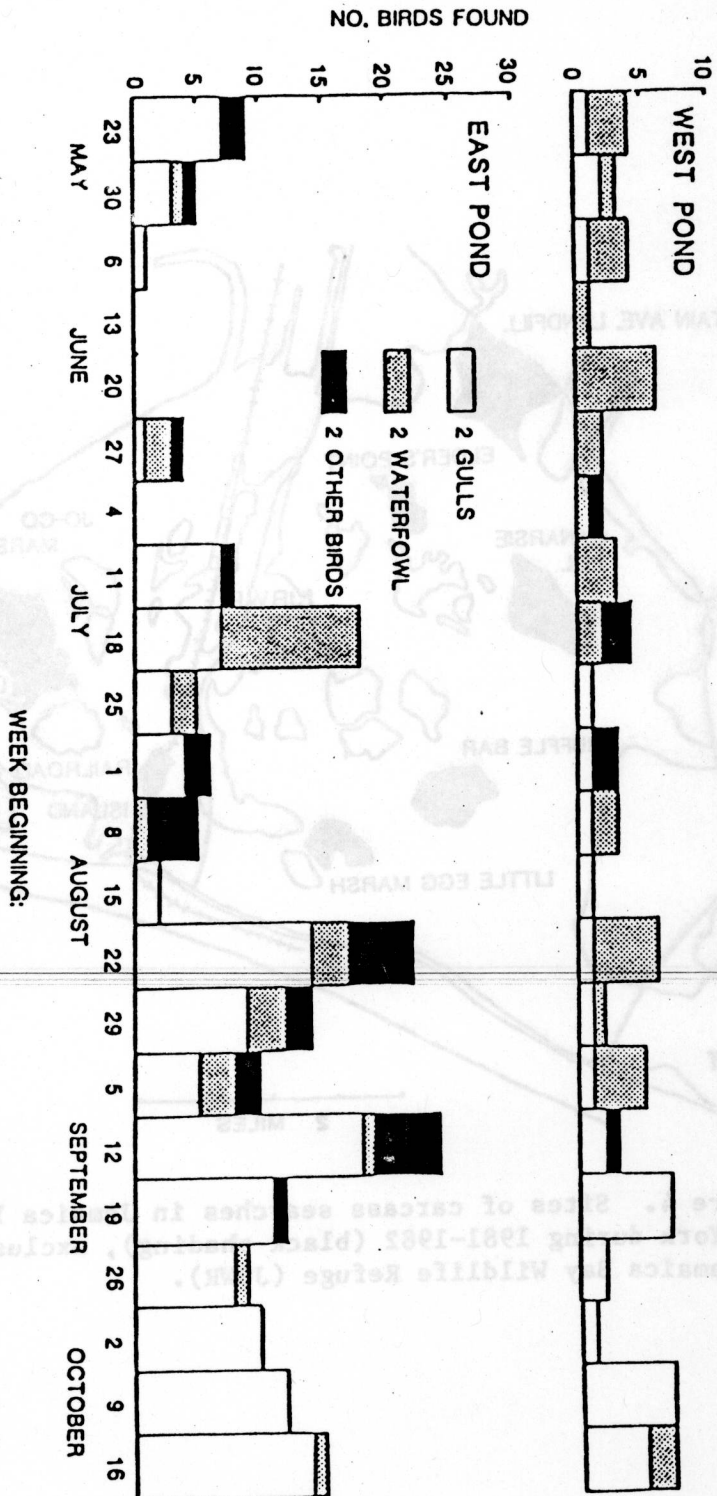


Figure 3. Weekly distribution of 268 moribund and dead birds found at Jamaica Bay Wildlife Refuge during May-October 1982.

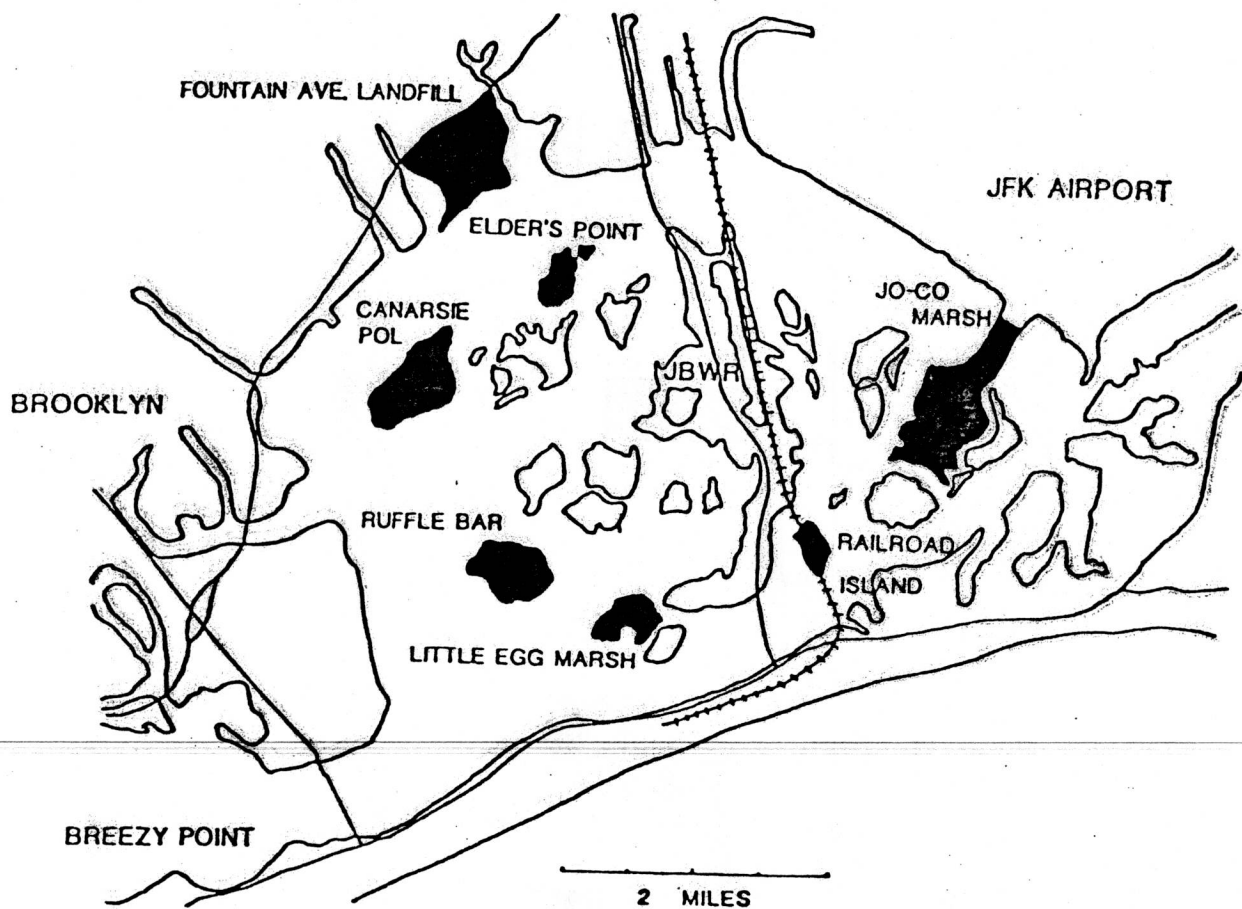


Figure 4. Sites of carcass searches in Jamaica Bay, New York during 1981-1982 (black shading), exclusive of Jamaica Bay Wildlife Refuge (JBWR).

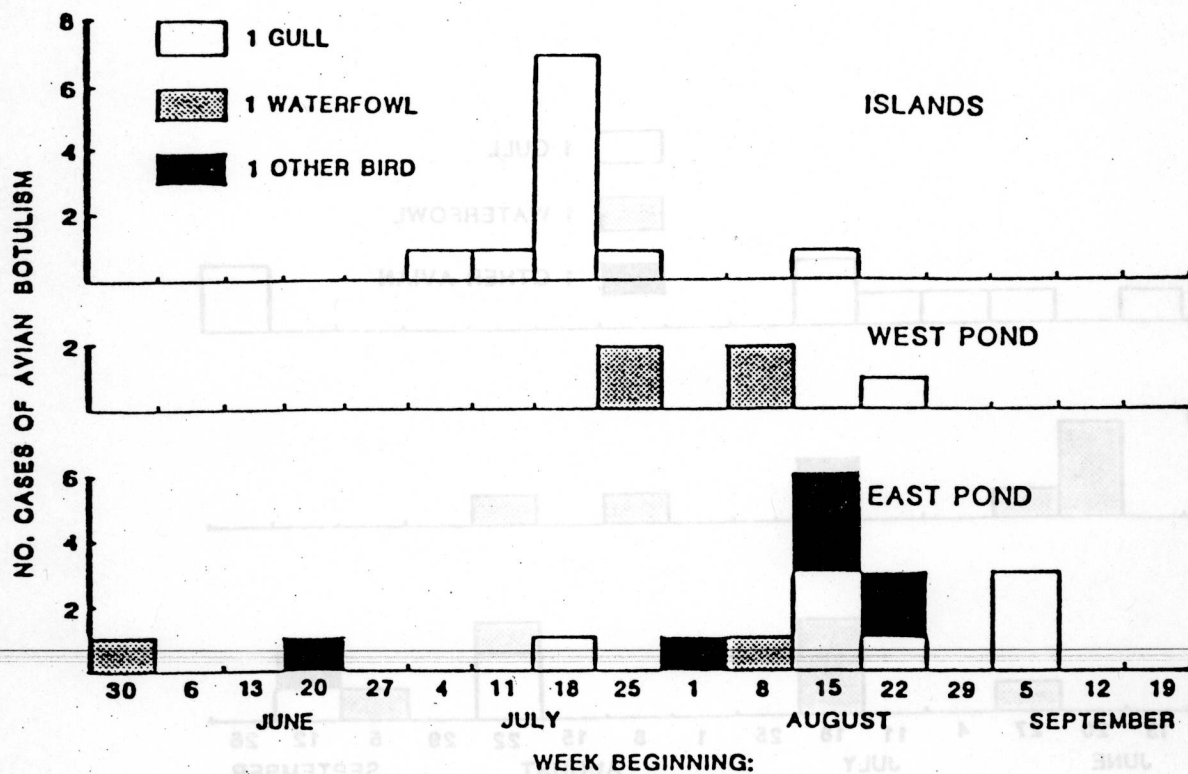


Figure 5. Weekly distribution of diagnosed cases of avian botulism at Jamaica Bay Wildlife Refuge and surrounding islands during May-October 1981.

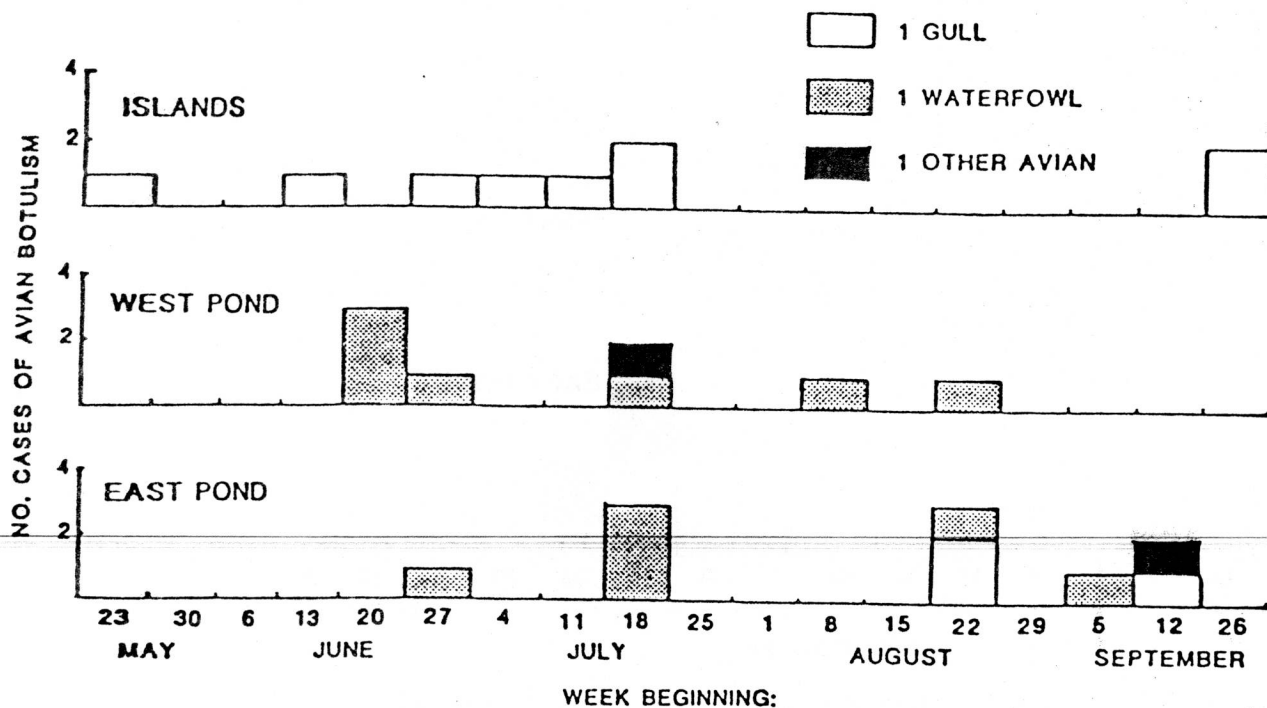


Figure 6. Weekly distribution of diagnosed cases of avian botulism at Jamaica Bay Wildlife Refuge and surrounding islands during May-October 1982.

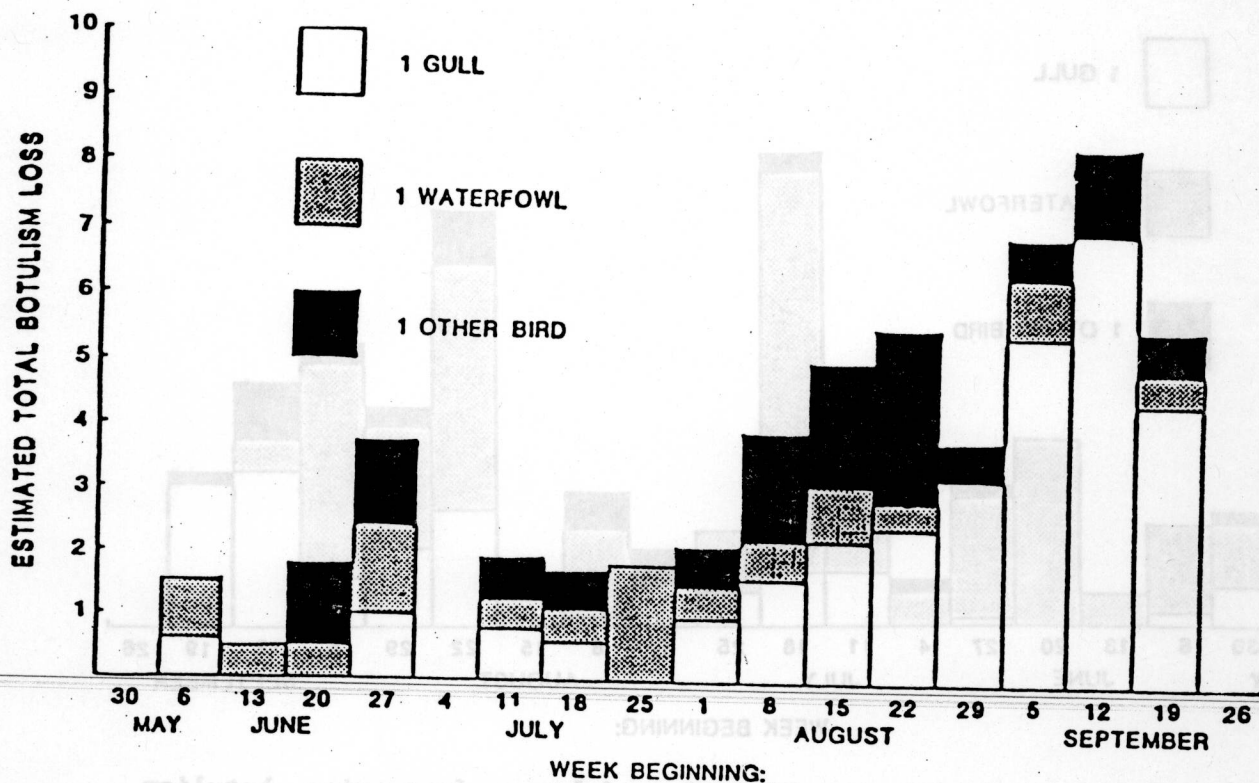


Figure 7. Weekly distribution of estimated losses from avian botulism at Jamaica Bay Wildlife Refuge during June-September 1981. East and West Pond data are combined.

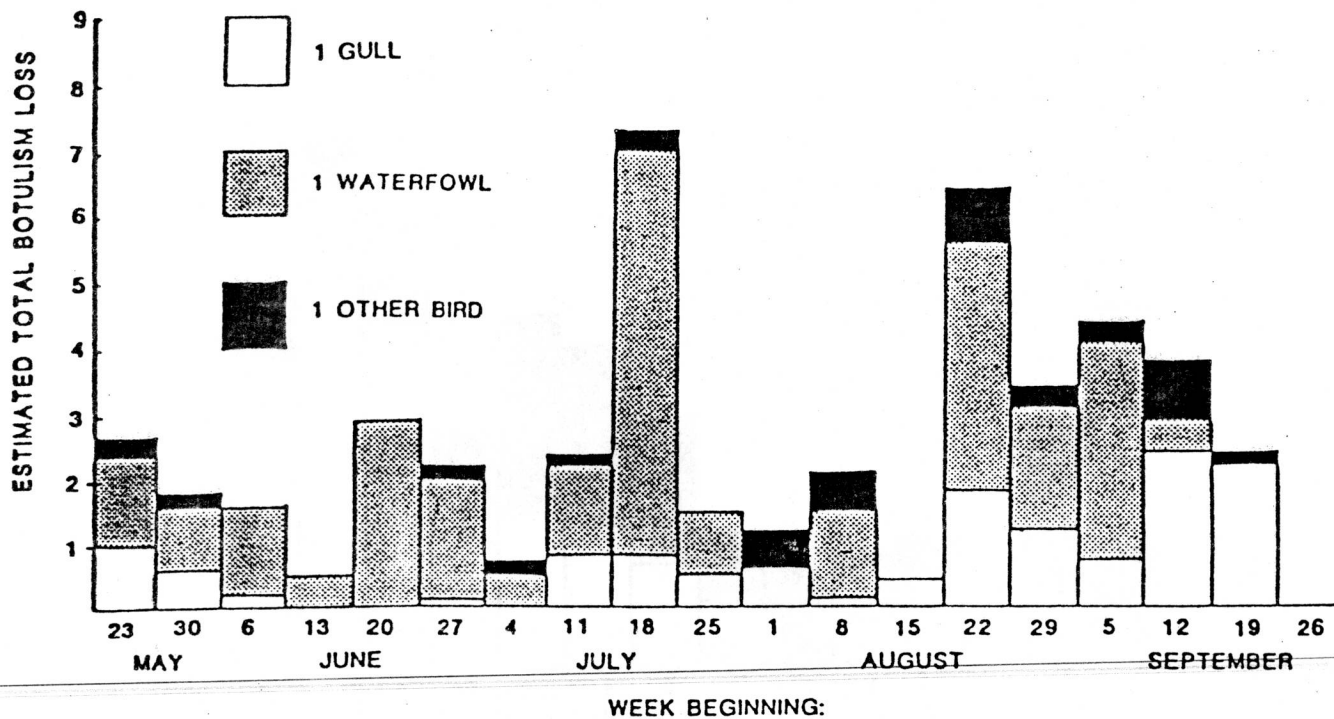


Figure 8. Weekly distribution of estimated losses from avian botulism at Jamaica Bay Wildlife Refuge during June-September 1982. East and West Pond data are combined.

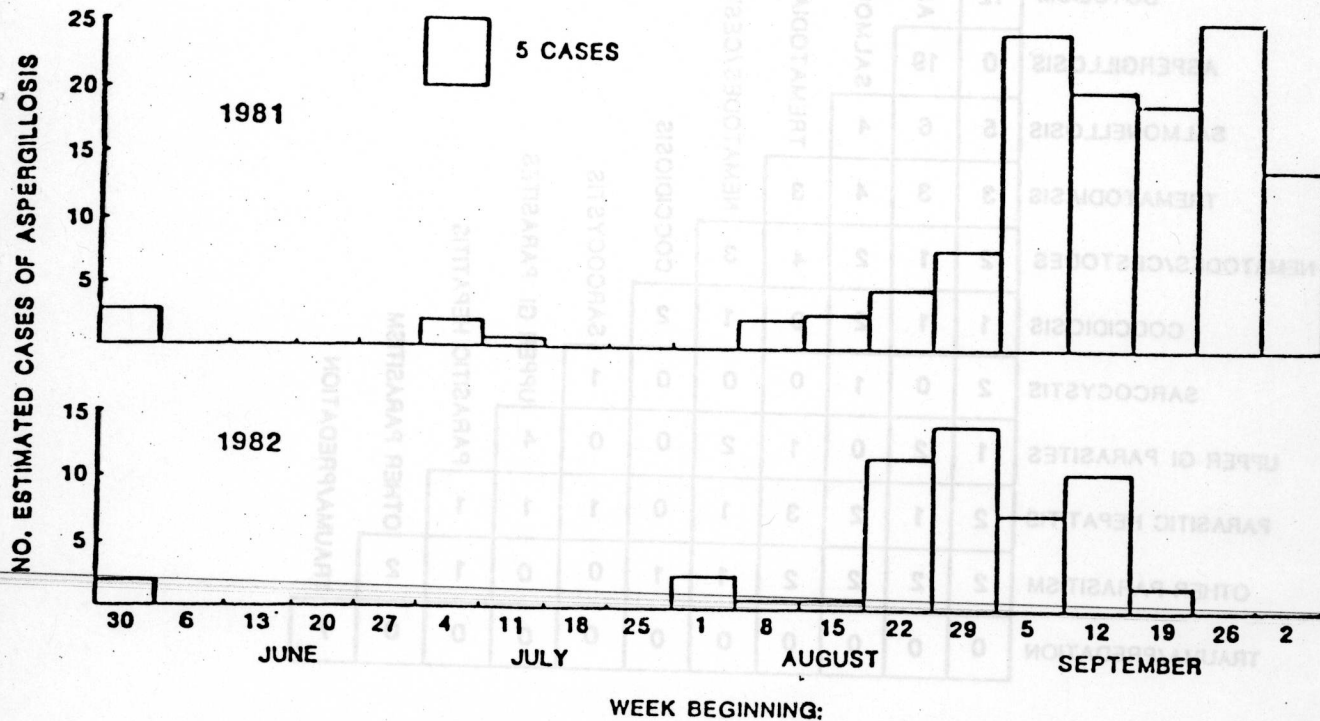


Figure 9. Weekly distribution of estimated losses from aspergillosis among gulls and waterfowl at Jamaica Bay Wildlife Refuge during 1981 and 1982. East and West Pond data are combined.

	BOTULISM	ASPERGILLOSIS	SALMONELLOSIS	TREMATODIASIS	NEMATODES/CESTODES	COCCIDIOSIS	SARCOCYSTIS	UPPER GI PARASITES	PARASITIC HEPATITIS	OTHER PARASITISM	TRAUMA/PREDATION
BOTULISM	12										
ASPERGILLOSIS	0	19									
SALMONELLOSIS	5	6	4								
TREMATODIASIS	3	3	4	3							
NEMATODES/CESTODES	2	1	2	4	3						
COCCIDIOSIS	1	1	2	0	1	2					
SARCOCYSTIS	2	0	1	0	0	0	1				
UPPER GI PARASITES	1	2	0	1	2	0	0	4			
PARASITIC HEPATITIS	2	1	2	3	1	0	1	1	1		
OTHER PARASITISM	2	2	2	2	1	1	0	0	1	2	
TRAUMA/PREDATION	0	0	0	0	0	0	0	0	0	0	7

Figure 10. Occurrences of multiple diagnoses in birds examined from Jamaica Bay Wildlife Refuge and surrounding islands during 1982. Number of cases for each paired diagnosis are in corresponding boxes; boxes along top diagonal refer to the number of cases with corresponding single diagnosis.