Avian Morbidity and Mortality from Botulism, Aspergillosis, and Salmonellosis at Jamaica Bay Wildlife Refuge, New York, USA

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Abstract.—During the summers of 1981 and 1982, studies were conducted at Jamaica Bay Wildlife Refuge, Long Island, New York, to determine whether annual water-level drawdowns used to create shorebird habitat also led to the occurrence of avian botulism (*Clostridium botulinum* type C). Low levels of morbidity and mortality from avian botulism occurred on the two ponds throughout both summers, but there was no apparent relationship between the occurrence or rates of botulism losses and drawdowns of the ponds. Botulism also occurred throughout both summers on other areas of the refuge. Botulinal toxin was found in fly larvae associated with avian carcasses, including birds that did not die from botulism. Toxin was not found in other samples of aquatic biota in the ponds, although it was demonstrated in a single sample of decomposing sea lettuce (*Ulva lactuca*) in Jamaica Bay. Aspergillosis (*Aspergillus fumigatus*) and salmonellosis (*Salmonella* spp.) were also frequently-diagnosed causes of morbidity and mortality. We believe that botulinal toxin present in carcasses of birds dying from botulism, or produced postmortem in birds dying from other causes, on the two ponds and other areas in Jamaica Bay were a major source of botulinal toxin. Toxin could be ingested by birds through direct scavenging on carcasses, or by consumption of toxic fly larvae associated with carcasses. Diligent carcass pickup at the two ponds is recommended to reduce mortality from avian botulism .

Key words.—Avian botulism, *Clostridium botulinum* type C. aspergillosis, *Aspergillus fumigatus*, salmonellosis, *Salmonella* spp., avian mortality.

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Most dramatic outbreaks of avian botulism (*Clostridium botulinum* type C) occur in western North America, where losses of tens of thousands of waterfowl, shorebirds, and other aquatic birds in a single outbreak are not unusual (Parrish and Hunter 1969, Hunter 1970a, Enright 1971, Malcolm 1982, National Wildlife Health Center (NWHC) unpublished data). Outbreaks of avian botulism in the coastal region of New York and New Jersey have been reported since 1950 (Toth 1974), with losses ranging from several to 1,000 birds (Reilly and Boroff 1967, Toth 1974, Figley and VanDruff 1982). At Jamaica Bay Wildlife Refuge (JBWR) on Long Island, New York, annual outbreaks attributed to avian botulism involving up to several hundred birds have occurred since 1975 (National Park Service unpublished reports).

Jamaica Bay Wildlife Refuge, part of the National Park Service (NPS) Gateway National Recreation Area (GNRA), is an important nesting and staging area for migratory birds along the Atlantic coast. Each fall, tens of thousands of migrating shorebirds and other aquatic birds stage on two ponds (East and West Ponds) on Ruler's Bar Hassock, the major island in JBWR (Fig. 1). The occurrence of botulism on the ponds preceding and during staging has raised concern about its threat to migratory birds, and whether drawdowns of the ponds for shorebird habitat contributes to the occurrence of the disease by the creation of mudflats and gradual sloping edges, as suggested in the western United States by Hunter (1970).

During 1981 and 1982, we investigated avian morbidity and mortality at JBWR, with special reference to factors contributing to botulism on East and West Ponds. We present here results of these investigations.

STUDY AREA

Jamaica Bay is a tidal lagoon on the south shore of western Long Island, New York, bounded by residential areas of Brooklyn, Queens and Rockaway Beach; and John F. Kennedy (JFK) International Airport (Fig. 1). The bay is shallow (<3 m at low tide) with numerous salt marsh islands and tidal mudflats that vary in extent with the lunar cycle (Black 1981, Burger 1982).

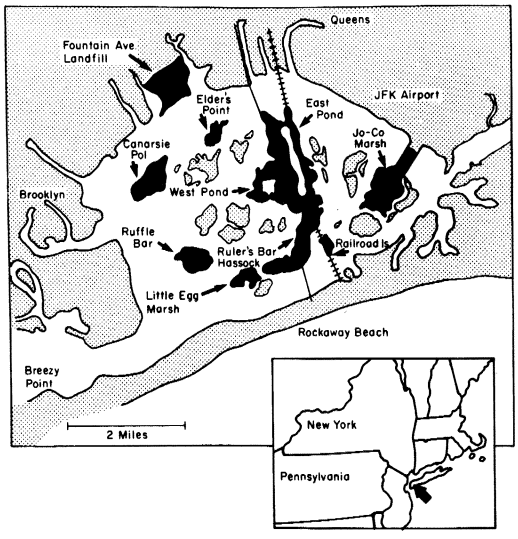

Figure 1. Sites of carcass searches (in black) in Jamaica Bay Wildlife Refuge during 1981 and 1982.

Jamaica Bay Wildlife Refuge was established within the bay in 1953 by the New York City Deparment of Parks, and was incorporated as part of GNRA in 1972. Two ponds, East Pond (39 ha) and West Pond (17 ha), created by spoil deposition are located on Ruler's Bar Hassock. Both ponds contain dense stands of reed grass (*Phragmites communis*) along shorelines. The water level in both ponds is generally shallow (<1 m), although East Pond has a channel bed up to 5.5 m deep. Each year water levels on both ponds are lowered to create habitat for shorebirds and wading birds. West Pond is drawn down in mid-April, exposing largely sandy beaches; while East Pond is lowered in early July, resulting in extensive mudflats. Water levels are restored through natural precipitation and drainage. While both ponds are unaffected by tidal fluctuations, West Pond was considerably more brackish than East Pond during the period of study (average salinity = 7.1 ± 0.4 ppt versus 4.9 ± 0.20 ppt) because faulty outlet valves used to drain the ponds periodically allowed inflow of water from Jamaica Bay.

METHODS

Magnitudes and trends in avian morbidity and mortality were determined by searching for sick and dead birds at East and West Ponds and other selected areas in Jamaica Bay (Fig. 1). These included islands with known nesting colonies of aquatic birds. Intensive searches of ponds were conducted on foot along shorelines and mudflats, and by canoe in open water and areas of emergent vegetation, two to five times weekly during June through September. Other areas of Jamaica Bay were searched an average of once per week, although not all areas were searched each week. We do not know what percentage of the total number of sick and dead birds was found during searches of the two ponds. Undoubtedly, some carcasses were not found either because scavengers or predators removed them, or they died in dense emergent vegetation. However, because of the intensive search effort, we believe that the numbers of birds found reflect temporal trends in morbidity and mortality on the ponds, and serve as minimum estimates for the period of study. Magnitudes and trends of mortality on other areas in Jamaica Bay could not be accurately determined because of the infrequency and irregularity of visits and the rapid disappearance and decomposition of carcasses. Nevertheless, the number of carcasses was recorded during each search as a subjective assessment of mortality occurring since the previous visit.

Moribund birds were captured by hand and humanely killed. These and fresh carcasses were necropsied. Autolyzed carcasses were buried away from the wetland. Causes of morbidity and mortality were determined on the basis of gross and histopathologic lesions and laboratory studies conducted at the NWHC involving appropriate standard virologic and bacteriologic procedures (Hitchner et al. 1980, Lennette et al. 1985). Serum from moribund birds and blood recovered from hearts of dead birds in suitable postmortem condition were routinely tested for type C botulinal toxin by the standard mouse protection test (Quortrup and Sudheimer 1943). The number of diagnosed cases of botulism is likely a minimum number among those examined, since mouse protection tests were not done when there was insufficient or unsuitable blood for testing. Aspergillosis (*Aspergillus fumigatus*) was diagnosed by culture and identification (Beneke 1980) in suspected cases of mycotic infections. *Salmonella* spp. were cultured (Kelly et al. 1985) from intestinal contents and from lesions observed at necropsy; serotypes were identified at the Wisconsin State Laboratory of Hygiene (Madison, Wisconsin 53706 USA). Because isolation of salmonellae was usually attempted for differential diagnosis in birds having gross lesions consistent with salmonellosis, these data do not reflect the prevalence of infection with *Salmonella* spp.

Weekly morbidity and mortality from botulism and aspergillosis was estimated by multiplying the proportion of cases for each disease (number of diagnosed cases/total number examined) by the total number of sick and dead birds found during each weekly interval. This was done to account for the variation between weeks in the percentage of carcasses that were suitable for necropsy and laboratory studies.

Samples of aquatic biota and fly larvae (mostly Calliphoridae) were collected periodically on East and West Ponds when mortality from botulism was occurring, and tested for the presence of botulinal toxin using the extraction method described by Foster et al. (1974). Benthic invertebrates were collected using an Ekman grab (Wildlife Supply Co., Saginaw, Michigan 48602 USA). Fly larvae from avian and fish carcasses and other biotic samples were collected in sterile jars or plastic bags and frozen until assayed.

Avian carcasses can serve as sources of botulinal toxin. Birds dying from botulism have preformed toxin in circulation. In addition, however, birds dying from any cause can contain botulinal toxin. In these cases, toxin is produced postmortem by germination of botulinal spores normally present in the gut as the carcass decomposes (Hunter 1970b). To further investigate the role of avian carcasses as sources of botulinal toxin, we placed eight herring gull (*Larus*

Table 1. Numbers of birds found moribund or dead on East and West Ponds at Jamaica Bay Wildlife Refuge during 1981 and 1982.

Species	1981		1982	
	East Pond	West Pond	East Pond	West Pond
Herring gull (*Larus argentatus*)	173	11	104	31
Laughing gull (*L. atricilla*)	15	1	19	1
Great black-backed gull (*L. marinus*)	16	1	5	2
Ring-billed gull (*L. delawarensis*)	13	0	8	1
Total gulls	217	13	136	35
Mallard (*Anas platyrhynchos*)	4	4	8	15
American black duck (*A. rubripes*)	4	1	8	4
Green-winged teal (*A. crecca*)	1	2	0	2
Blue-winged teal (*A. discors*)	0	0	3	0
Gadwall (*A. strepera*)	1	0	4	3
White pekin (*A. platyrhynchos*)	1	0	1	0
Unidentified duck	0	0	3	0
Canada goose (*Branta canadensis*)	3	4	1	12
Brant (*Branta bernicla*)	0	3	1	0
Total waterfowl	14	14	29	36
Double-crested cormorant (*Phalacrocorax auritus*)	2	1	4	2
Great blue heron (*Ardea herodias*)	1	0	0	0
Snowy egret (*Egretta thula*)	0	0	1	0
Black-crowned night-heron (*Nycticorax nycticorax*)	1	0	1	1
Glossy ibis (*Plegadis falcinellus*)	2	0	0	1
Common moorhen (*Gallinula chloropus*)	0	0	1	0
Semipalmated plover (*Charadriis semipalmatus*)	2	0	1	0
Black-bellied plover (*Pluvialis squatarola*)	2	0	1	0
Semipalmated sandpiper (*Calidris pusilla*)	2	0	1	0
Willet (*Catoptrophorus semipalmatus*)	0	0	1	0
Lesser yellowlegs (*Tringa flavipes*)	0	0	1	0
Unidentified shorebird	0	0	4	0
Black skimmer (*Rhynchops niger*)	6	0	7	1
Common tern (*Sterna hirundo*)	1	0	2	0
Rock dove (*Columba livia*)	0	0	1	0
American crow (*Corvus brachyrhynchos*)	0	1	0	0
Northern mockingbird (*Mimus polyglottos*)	0	0	0	1
Total	250	29	191	77

argentatus) carcasses in shallow water at the shoreline of East and West Ponds. These birds had been found moribund on East Pond, and were bled and killed. Carcasses were covered with chicken wire. Invertebrates found on the carcasses during 1-49 h were collected and tested for toxin as described above.

RESULTS

A total of 279 birds (256 dead and 23 moribund) were found at East and West Ponds in 1981, and 268 (235 dead and 33 moribund) in 1982 (Table 1). Gulls (*Larus* spp.) were the major group: 230 (82%) in 1981 and 171 (64%) in 1982.

Morbidity and mortality occurred throughout the summer on both East and West Ponds (Fig. 2). East Pond sustained the most losses during both years: 250 (90%) during 1981 and 191 (71%) during 1982. Major increases in morbidity and mortality began in early-August (1981) and mid-August (1982), primarily in gulls. A total of 161 birds from East and West Ponds were necropsied, including 77 during 1981 and 84 during 1982 (Table 2). There was one or more specific diagnosis for 129 (80%) of the birds examined; no specific diagnosis could be reached for the other 32 birds.

Moribund and dead birds were found throughout the summer on seven other areas in Jamaica Bay (Fig. 1). Ninety-six birds were necropsied (Table 3). The number of dead birds observed on each

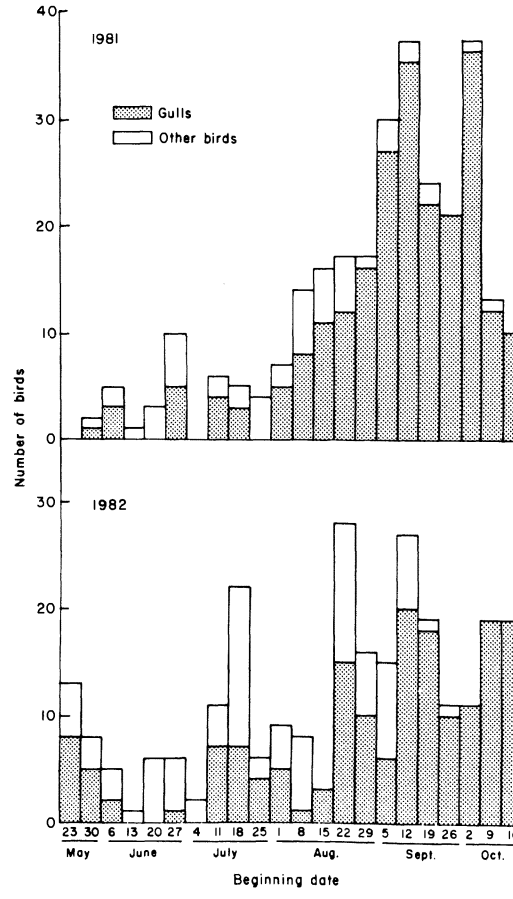

Figure 2. Numbers of moribund and dead birds found during weekly intervals at East and West Ponds, Jamaica Bay Wildlife Refuge, during 1981 and 1982. Beginning date refers to first day of weekly interval.

Table 2. Summary of diagnoses for 161 moribund and dead birds necropsied from East and West Ponds, Jamaica Bay Wildlife Refuge, during 1981 and 1982. Number of birds necropsied in parentheses.

Species/group	Avian botulism	Aspergillosis	Salmonellosis[1]	Other[2]	Unknown
Gulls (99)	12	46[3]	10	20	18
Ducks (28)	17[4]	2	1	5	4
Geese (10)	2	2	0	9	2
Shorebirds (8)	4	0	0	1	3
Black skimmer (8)	3	0	0	3	2
Glossy ibis (1)	1	0	0	0	0
Double-crested cormorant (2)	1	0	0	1	0
Other birds[5] (5)	0	0	0	2	3
Total (161)	40	50	11	36	32

[1]Includes cases where isolation of *Salmonella* spp. was associated with pathologic change, and was believed to have contributed to morbidity or mortality.
[2]Includes diagnoses of parasitism (n = 22), trauma (n = 10), unidentified bacterial septicemia (n = 2), predation (n = 1), and ruptured yolk peritonitis (n = 1); these factors were believed to have contributed to morbidity or mortality.
[3]Two cases of aspergillosis occurred concurrently with botulism, four cases occurred concurrent with enteritis due to *Salmonella* spp., and one case occurred concurrent with enteritis due to coccidiosis (unidentified species).
[4]One white pekin that died from botulism also had an enteritis due to *Salmonella typhimurium*.
[5]Includes two common terns, one common crow, and one rock dove.

Table 3. Summary of diagnoses for 96 moribund and dead gulls from areas other than East and West Ponds in Jamaica Bay Wildlife Refuge during 1981 and 1982. Number of birds necropsied in parentheses.

Species	Avian botulism	Aspergillosis	Salmonellosis	Other	Unknown
Herring gull (86)	20	9[1]	4[2]	22[3]	34
Black-backed gull (8)	0	3	1	0	4
Laughing gull (2)	0	1	0	0	1
Total (96)	20	13	5	22	39

[1]One case of asperillosis occurred concurrently with botulism, and one case occurred concurrently with enteritis due to *Salmonella* spp.
[2]Includes cases where isolation of *Salmonella* spp. was associated with pathologic change, and was believed to have contributed to morbidity or mortality. One gull had an enteritis due to *Salmonella thompson* but was also moribund from botulism.
[3]Includes diagnoses of parasitism (n = 12), nephritis and nephrosis of unknown etiology (n = 7), trauma (n = 2), and amyloidosis (n = 1); these factors were believed to have contributed to morbidity or mortality.

search of other areas was relatively constant, with the exception of 12 July 1981, when about 250 and 125 dead herring gulls were found at Ruffle Bar and Canarsie Pol, respectively. Most were about 2 wk prefledging, and death had apparently occurred 7 to 10 days earlier. Postmortem autolysis and scavenging of carcasses precluded necropsies of these birds.

Avian Botulism

Avian botulism was diagnosed in 40 (25%) of the 161 birds necropsied from East and West Ponds (Table 2). This is a minimum number, since tests for botulism could not be done on 42 (26%) of the 161 birds examined. However, 37 (88%) of the 42 birds not tested for botulism were diagnosed as moribund or dead from other confirmed causes. Seventeen (42%) of the 40 cases of botulism were moribund birds. Of the 23 cases of botulism among dead birds, it is possible that postmortem toxin formation in carcasses resulted in positive botulism tests. However, if postmortem toxin formation in carcasses was an important source of error in the diagnosis of botulism, then we might expect the proportion of dead birds testing positive for toxin (23/63 tested) to be greater than for moribund birds (17/56), presuming the true proportion of birds that died from botulism was similar to the proportion of moribund birds with botulism. These proportions were not significantly different (χ^2 = 0.26; P>0.5), suggesting that false positive diagnoses of botulism due to postmortem toxin formation were not an important source of error in our study. In addition, botulism was diagnosed in 20 (21%) of the 96 gulls examined from other areas in Jamaica Bay (Table 3).

Botulism occurred from late May to mid-September, both on the ponds and other areas in Jamaica Bay. The estimated total loss from botulism on East and West Ponds during 1981 was 70 (35 gulls, 17 waterfowl, and 18 other aquatic birds); during 1982 it was 69 (21 gulls, 35 waterfowl, and 13 other aquatic birds). During 1981, the number of cases per week was low and constant during June and July, then increased about four-fold to a peak in mid-September (Fig. 3). This increase was accounted for primarily by gulls. In contrast, waterfowl accounted for a significantly higher percentage of the botulism cases in 1982 (χ^2 = 9.28; P<0.005). The number of cases per week in 1982 was constant until a 3-fold increase among gulls in mid-September, but was relatively irregular among other bird groups.

Despite the increase in botulism cases among gulls during August and September, it does not appear that actual morbidity and mortality rates from this disease increased during this period. Populations were not estimated at the two ponds regularly. However, subjective evaluations of bird numbers indicated an approximate three-fold increase in gulls beginning in late July and early August, compared to the three- and four-fold increases in number of botulism cases.

Sources of Botulinal Toxin

Fly larvae collected from a Mallard (*Anas platyrhynchos*) and a Laughing Gull (*Larus atricilla*) found dead on East Pond

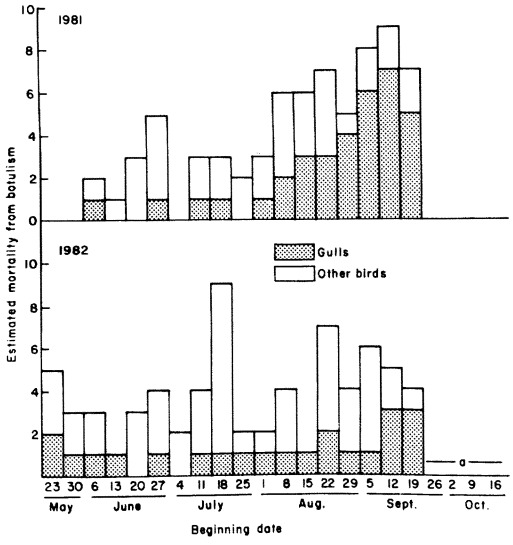

Figure 3. Estimated weekly morbidity and mortality from avian botulism at East and West Ponds, Jamaica Bay Wildlife Refuge, during 1981 and 1982. During weeks of 26 September-16 October 1982, carcasses were not examined, precluding estimates of mortality during this period (denoted by a). Beginning date refers to first day of weekly interval.

and a herring gull found on Canarsie Pol were positive for botulinal toxin. These carcasses were too autolyzed to determine the cause of death. In addition, a pooled sample of decomposing sea lettuce (*Ulva lactuca*) collected at three sites on Jamaica Bay at the high tide line on 22 July 1982 contained type C toxin. The amount of toxin in these samples was not quantified. However, mouse protection tests on samples of fly larvae resulted in death of non-protected mice; while those conducted on sea lettuce produced only sickness with characteristic signs of botulism in non-protected mice, suggesting toxin levels were lower. Twenty-four samples of sea lettuce subsequently collected on 24 August and 8 September were negative for toxin.

Botulinal toxin was not detected in samples of living benthic (n = 6) or littoral (n = 6) invertebrates, bottom detrital samples (n = 25), algae mats (n = 1 pooled sample), a fish (undetermined species) carcass (n = 1), or unidentified fly larvae from two fish carcasses (n = 2). All samples were collected when mortality from botulism was occurring.

Invertebrates associated with the Herring Gull carcasses placed along the shoreline were collected at 18, 22, 42, and 49 hr. By 49 hr, flesh and soft tissues of carcasses mostly had been consumed, and further collecting was discontinued. Botulinal toxin was demonstrated in fly larvae from three of the eight carcasses at 42 and 49 hr. Toxin was not found in coleopterans (Silphidae and Carabidae) or snails (undetermined species) found on the carcasses. Two of the three gulls producing toxic fly larvae were moribund from botulism when captured; the other had aspergillosis. Causes of morbidity in the five gulls that did not produce toxic fly larvae or other invertebrates included botulism (n = 3), aspergillosis (n = 1), and unknown (n = 1).

Aspergillosis

Aspergillosis was the most frequently diagnosed disease during both years in birds from East and West Ponds, affecting 50 (31%) of the 161 birds examined (Table 2). Eleven (22%) of the cases were diagnosed in moribund birds. Aspergillosis was manifested as a mycotic pneumonia, air sacculitis, and pericarditis in 28 cases; and as an acute disseminated mycosis in 22 cases. In all cases, we believe lesions observed were sufficient to cause debilitation or death. Two dead herring gulls included above were likely debilitated from mycotic pneumonia due to aspergillosis; however, botulinal toxin was demonstrated in blood from these birds and we believe they died from botulism. Forty-six (92%) of the cases of aspergillosis occurred in gulls; 40 of these were Herring Gulls. Estimated total morbidity and mortality from aspergillosis on the ponds was 125 during 1981 and 51 during 1982.

Ninety-six percent of the herring gulls diagnosed as having aspergillosis were subadult birds. While we do not have the corresponding age ratio for healthy birds, this percentage is different than that (72%) for herring gulls dying from other causes ($\chi^2 = 12.89$; $P<0.005$), suggesting that subadults were at greater risk for this disease than adults.

Temporal patterns of morbidity and mortality from aspergillosis were similar to those described for botulism, with increases during mid-August through September (Fig. 4), primarily in subadult herring gulls. However, the magnitude of change in occurrence of aspergillosis was

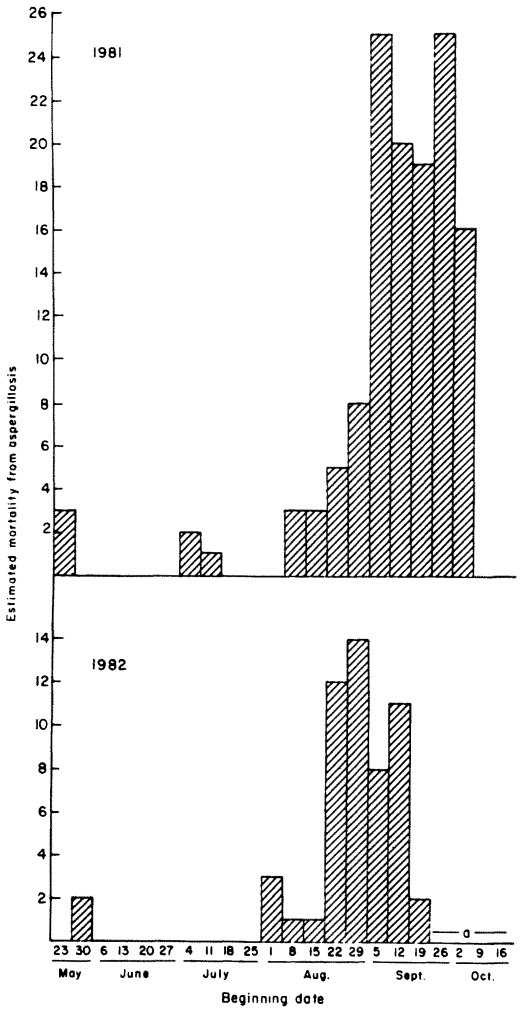

Figure 4. Estimated weekly morbidity and mortality from aspergillosis among gulls at East and West Ponds, Jamaica Bay Wildlife Refuge, during 1981 and 1982. During weeks of 26 September-16 October 1982, carcasses were not examined, precluding estimates of mortality during this period (denoted by a). Beginning date refers to first day of weekly interval.

much greater (about 20-fold) than that for botulism, and appeared greater than the increase in gull populations at that time. Thus, it is likely that mortality rates from aspergillosis increased during this period.

Salmonellosis

Salmonella spp. were isolated from large or small intestines or livers of 26 moribund or dead birds from JBWR, including 24 gulls, a white pekin (*Anas platyrhynchos*), and a Black Skimmer (*Rynchops niger*). Gross and histologic lesions consistent with enteritis or hepatitis associated with salmonellosis were present in 17 of the 26 birds, including the white pekin and Black Skimmer. Eleven of the 17 cases of salmonellosis occurred in birds from East and West Ponds (Table 2); the other cases were from other islands in JBWR. In 6 cases (2 moribund gulls, 3 dead gulls, and 1 dead Black Skimmer), enteritis due to salmonella was the only significant finding at necropsy, and was likely the cause of morbidity or mortality. In the other cases, salmonellosis was probably incidental to other identified causes of morbidity and mortality, including botulism (4 cases) and aspergillosis (4 cases).

A wide variety of *Salmonella* serotypes were identified, including *S. typhimurium* (n = 12), *S. heidelberg* (n = 6), *S. blockley* (n = 3); and *S. manhattan, S. thompson, S. agona, S. infantis, S. stanley, S. arizona,* and an unidentfied serotype (n = 1 each). Concurrent infections with two serotypes occurred in two cases (*S. typhimurium* with *S. manhattan,* and *S. typhimurium* with *S. heidelberg*). There was no relationship between serotype and presence or degree of pathology associated with infection. The isolation of *S. typhimurium* from the intestine of the black skimmer apparently represents the first report of enteric salmonellosis in this species.

DISCUSSION AND CONCLUSIONS

Outbreaks of avian botulism have been reported with increasing frequency on the eastern coast of the United States (viz., Reilly and Boroff 1967, Forrester et al. 1980, Shayegani et al. 1984. NWHC unpublished data). Most epizootiological studies of this disease in North America have been done in the West, where botulism has been a common avian disease since before the turn of the century (Kalmbach and Gunderson 1934, Bell et al. 1955, Hunter 1970b, Duncan and Jensen 1976, Moulton et al. 1976). However, the worldwide occurrence of botulism (Jensen and Price 1987) in a wide variety of habitat, environmental, and climatic conditions suggests that its epizootiology varies considerably over its geographic range. The purpose of this study at JBWR was to better understand the disease as it occurs there, and its potential relationship to water drawdowns on East and West Ponds.

Epizootic mortality from avian botulism did not occur on either East or West Pond during the two summers of this study; although the number of botulism cases increased 3- to 4-fold during late summer, this reflected an increase of similar magnitude in gull populations at the ponds rather than an increase in morbidity and mortality rates from botulism. There was no apparent association between the occurrence of botulism and drawdowns of either pond. Mortality from botulism occurred at relatively low levels throughout the period of study. Botulism also occurred through the summer on other islands in Jamaica Bay, suggesting that sources of toxin were available over a widespread area. Thus, it does not appear that water management activities on the ponds had any major impact on botulism during the period of study.

The only consistent source of botulinal toxin was in fly larvae associated with avian carcasses. Larvae consuming carcasses of birds dying from botulism as well as from other causes became toxic by concentrating toxin present in the carcass. The relationship between toxic fly larvae and avian carcasses in initiating and perpetuating botulism outbreaks has been well established (Kalmbach and Gunderson 1934, Hunter 1970b, Duncan and Jensen 1976, Malcolm 1982). The ability of a carcass to produce toxic larvae may be related to the presence of preformed toxin in tissues of birds dying from botulism, or to toxin produced postmortem by germination and growth of cells or spores of *Clostridium botulinum* present in the gut or contaminating the carcass under suitable microenvironmental (Bell et al. 1955) or microclimatic (Wobeser and Galmut 1984) conditions. A potential additional source of toxin was identified in a sample of decomposing sea lettuce. However, previous studies (Bell et al. 1955) have suggested that vegetation is not an important substrate for toxin production. It is possible that toxin in this sample was concentrated within invertebrate carcasses entrapped in the sea lettuce, as suggested by the microenvironment concept of Bell et al. (1955).

A variety of morbidity and mortality factors in addition to botulism was found in our study, particularly aspergillosis and salmonellosis. Aspergillosis appeared to be the most important disease, occurring in epizootic form during late summer and early fall of both years, primarily in subadult herring gulls. This period of time corresponds to influxes of first-year gulls to the refuge after fledging (Burger 1981), possibly accounting for their apparent disproportionate morbidity and mortality from aspergillosis. Infection of gulls with salmonellae was also common, but not always associated with pathologic changes. Gulls are recognized as common hosts of *Salmonella* spp., particularly gulls associated with garbage dumps and landfills (Edel et al. 1978, Nielsen 1960, Müller 1965, Fricker et al. 1983). Likewise, a large landfill site near JBWR (Fig. 1) was utilized by gulls associated with East and West Ponds (Burger 1981).

The continual presence throughout Jamaica Bay of avian carcasses dying from a variety of causes provided an abundant substrate for production and maintenance of botulinal toxin and its concentration in fly larvae, as was demonstrated in our study. We suspect that botulism morbidity and mortality at JBWR was caused largely by the avian carcass-fly larvae cycle. Botulism in gulls may also have been due to direct scavenging of toxin-laden carcasses, as suggested by MacDonald and Standring (1978).

The magnitude of losses from botulism in this study was low relative to previous reports of botulism outbreaks on East and West ponds, where as many as 392 birds were previously reported to have died during late summer with symptoms consistent with botulism (NPS unpulished reports). Intensive searches for carcasses during our study may have contributed to this low level by removing a major portion of the substrate for toxin production. It is also possible that previous epizootics may have been caused by factors in addition to botulism, such as aspergillosis. Continued intensive searches for carcasses throughout the summer is recommended to minimize mortality from the carcass-fly larvae cycle of botulism, and periodic submission of carcasses for diagnostic studies is encouraged to verify causes of mortality.

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