size and cost of potholes. J. Wildl. Manage. 36: 459–468.

KAMINSKI, R. M., AND H. H. PRINCE. 1981. Dabbling duck and aquatic macroinvertebrate responses to manipulated wetland habitat. J. Wildl. Manage. 45:1–15.

KRULL, J. N. 1970. Aquatic plant macroinvertebrate associations and waterfowl. J. Wildl. Manage. 34: 707–718.

LEITCH, W. G. 1964. Water. Pages 273–281 *in* J. P. Linduska, ed. Waterfowl tomorrow. U.S. Dep. Inter., Washington, D.C.

MCNICOL, D. K., B. E. BENDELL, AND R. K. ROSS. 1987. Studies of the effects of acidification on aquatic wildlife in Ontario. Can. Wildl. Serv. Occas. Pap. 62. 76pp.

MOYLE, J. B. 1945. Some chemical factors influencing the distribution of aquatic plants in Minnesota. Am. Midl. Nat. 34:402–420.

———. 1956. Relationships between the chemistry of Minnesota surface waters and wildlife management. J. Wildl. Manage. 20:303–320.

PATTERSON, J. H. 1976. The role of environmental heterogeneity in the regulation of duck populations. J. Wildl. Manage. 40:22–32.

REID, G. K. 1961. Ecology of inland waters and estuaries. Van Nostrand Reinhold Co., New York, N.Y. 375pp.

SAS INSTITUTE INC. 1985. SAS user's guide: statistics. SAS Inst. Inc., Cary, N.C. 956pp.

SWANSON, G. A., T. C. WINTER, V. A. ADOMAITIS, AND J. W. LABAUGH. 1988. Chemical characteristics of prairie lakes in south-central North Dakota—their potential for influencing use by fish and wildlife. U.S. Fish and Wildl. Serv. Tech. Rep. 18. 44pp.

Received 26 March 1991.
Accepted 13 November 1991.
Associate Editor: Kaminski.

***Wildl. Soc. Bull.* 20:175–182, 1992**

THE ROLE OF AVIAN CARCASSES IN BOTULISM EPIZOOTICS

THOMAS M. REED, ***Wisconsin Cooperative Fishery Unit, College of Natural Resources, University of Wisconsin, Stevens Point, WI 54481***

TONIE E. ROCKE, ***U.S. Fish and Wildlife Service, National Wildlife Health Research Center, 6006 Schroeder Road, Madison, WI 53711***

Avian botulism can kill thousands of waterfowl in a single outbreak. The disease is caused by ingestion of a neurotoxin produced by the bacterium *Clostridium botulinum*. Type-C botulism is the serotype most commonly detected in waterfowl; type E also occurs in birds but less frequently and predominantly in fish-eating species.

Spores of *C. botulinum* type C are widely distributed in marsh sediments (Mitchell and Rosendal 1987, Wobeser et al. 1987) and also commonly occur in the tissues of marsh-associated vertebrates and invertebrates (Duncan and Jensen 1976). Production of type-C neurotoxin by this bacterium is dependent on the presence of specific bacteriophages (Eklund et al. 1987). A suitable substrate and environmental conditions (e.g., temperature and pH) that are conducive to spore germination, bacterial growth, and toxin production also are required. Waterfowl acquire botulism through ingestion of toxin-laden food items, presumably invertebrates.

Decaying avian carcasses that contain botulism cells or spores provide a suitable substrate for the production of toxin (Hunter 1970, Duncan and Jensen 1976, Malcolm 1982, Smith and Turner 1987). Fly larvae and other invertebrates that feed on decaying carcasses ingest and carry botulism toxin (Bell et al. 1955, Lee et al. 1962, Duncan and Jensen 1976). Feeding waterfowl then consume these toxic maggots

and invertebrates, contract botulism, and die. These waterfowl carcasses provide new substrate for toxin formation, and the cycle is perpetuated (Hunter 1970). Preformed toxin in the carcasses of birds that ingested lethal levels of botulism toxin also may contribute to carcass–maggot cycles of botulism (Brand et al. 1988).

Natural resource agencies spend time and money collecting and disposing of avian carcasses during botulism outbreaks, but the effectiveness of carcass removal in controlling avian botulism has not been evaluated in a systematic manner (Duncan and Jensen 1976, Wobeser 1987). Managers continue to question whether carcass removal reduces deaths from botulism. We investigated botulism toxin production in carcasses of birds that died from botulism and from other causes and evaluated the role of both types of carcasses in the initiation of botulism outbreaks. We also compared the occurrence of botulism intoxication among age and sex cohorts.

STUDY AREA AND METHODS

Study Sites and Animals

Our study was conducted at the Sacramento National Wildlife Refuge (SNWR), 11 km south of Willows, California. The refuge is an intensively managed wetland complex consisting of 4,050 ha of seasonally and permanently flooded pools. We selected 2 pools that had previous histories of botulism outbreaks as study sites. Water depth in each pool was 40–60 cm. Dominant plant species were hardstem bulrush (*Scirpus acutus*), cattail (*Typha* spp.), swamp timothy (*Heleochloa schoenoides*), smartweed (*Polygonum* spp.), and cocklebur (*Xanthium strumarium*).

Sets of 3 adjacent 0.8-ha enclosures were built in both pools. Each enclosure was constructed with plastic poultry netting (Centoflex M, Internet, Inc., 2730 Nevada Avenue North, Minneapolis, MN 55427) secured to 9-gauge wire stretched between wooden brace posts at each corner. Metal posts were used to support the wire and netting.

Thirty pinioned or wing-clipped mallards (*Anas platyrhynchos*) obtained from a game farm (Wild Wings of Oneka, 9421 152nd Street North, Hugo, MN 55038) were placed within each enclosure and used as sentinels to monitor the occurrence of botulism. Fifteen hatch-year (HY) and 15 after-hatch-year (AHY) birds of mixed sex were maintained within each enclosure throughout the study. Each sentinel was marked with a numbered patagial tag and leg band.

Experimental Trials

Three experimental trials (20–29 days duration each) were conducted in each of 2 pools between 19 July and 15 December 1988. At the beginning of each trial, 10 mallard carcasses were placed in 2 of 3 adjacent enclosures; random numbers were used to determine carcass locations. In 1 enclosure, we placed carcasses of mallards known to have died from botulism (botulism-intoxicated) either by ingestion of toxin in previous outbreaks or by injection of toxin in experimental studies. Mouse neutralization tests (Quortrup and Sudheimer 1943) were used to demonstrate type-C botulism toxin in the bloodstream of these birds. Carcasses of healthy mallards that had been euthanized in a carbon dioxide chamber were placed in the second enclosure. Carcasses of both types were from birds that had been previously held in marsh enclosures at SNWR, and all were frozen immediately after death. Carcasses were kept frozen for up to 1 year prior to use in this study. For placement in enclosures, each carcass was thawed for 24 hours, wrapped securely with monofilament line, and tied to a wooden stake with a 40- to 60-cm line. The stake was pushed into the marsh substrate so that the carcass floated on the water surface. No carcasses were placed in the third (control) enclosure. The control treatment was assigned to the same enclosure for all 3 trials in each pool. The 2 carcass treatments (botulism-intoxicated and euthanized) were assigned to alternate enclosures for each trial to ensure that enclosure effects on maggot infestations and botulism outbreaks were minimized.

Enclosures were searched for sick and dead birds on foot and by canoe each day during each trial. A dog also was used to retrieve ducks. Sick and dead sentinels were removed from enclosures and replaced on the same day with healthy sentinels of the same age. Blood samples from sick birds and blood collected from the hearts of dead birds were centrifuged to collect serum, which was tested for type-C botulism toxin with mouse neutralization tests (Quortrup and Sudheimer 1943). Only those birds whose sera tested positive for type-C botulism toxin were considered to have contracted botulism.

The external condition of each carcass placed in the marsh was assessed and recorded daily throughout the duration of each trial using the following classifications: intact, first maggots visible, 50% maggot-infested, and 100% maggot-infested. Maggot samples of 1–10 g were collected daily, when available, from each staked carcass. They were stored in plastic tubes and kept cool until they could be frozen. After thawing, the samples were processed, tested, and titrated for type-C botulism toxin as described by Duncan and Jensen (1976). The

reciprocal of the highest dilution to kill both inoculated mice was considered the toxin titer, expressed as the minimum mouse lethal dose (MLD)/g of maggots.

At the end of each trial, all carcass remains were removed. Ten days passed before a new trial was begun in the same pool (except 3 days between trials 5 and 6). Sentinel birds were maintained in enclosures between trials and monitored daily for morbidity or mortality. No botulism occurred in sentinel birds between any of the trials.

To prevent botulism in wild birds during ongoing trials when carcasses were present, we attempted to scare them away from the experimental enclosures. Monofilament with streamers of flagging tape was strung above enclosures with carcasses. Scarecrows were placed in the marsh, and firecracker shot-shells were fired periodically. Our research activities also discouraged use of enclosures by wild birds.

Data Analysis

Sentinel mallards were regarded as separate observations, and we assumed the probability of contracting botulism was independent among sentinels. Survival rates for sentinel mallards in each enclosure were determined (STAGKAM program, Missouri Dep. Conserv., Columbia) with the Kaplan–Meier survival estimator (Kaplan and Meier 1958) using the staggered-entry design, which allows for new animals to be added after the study was begun (Pollock et al. 1989). Survival rates represent the cumulative probability of birds surviving (i.e., not contracting) botulism during the trial. We report the cumulative probability of birds contracting botulism (1.0 − survival rate) during each trial. A chi-square log-rank test (Pollock et al. 1989) was used to compare survival rates between sentinels in enclosures with carcasses and sentinels in enclosures without carcasses in the same trial. The chi-squared statistic was converted to a z-statistic (Crowley and Breslow 1984), which was compared to a table of critical values for a 1-sided z-distribution. In addition, the average daily probability of intoxication in each enclosure was calculated using the number of botulism deaths or illnesses ÷ the number of bird-exposure days (Mayfield 1961, 1975). This statistic was used to calculate relative risk by dividing the average daily probability of intoxication in enclosures with carcasses by the probability in enclosures without carcasses (Kahn 1983:38–39).

Comparisons also were made between survival rates of AHY and HY and between male and female sentinels within each enclosure in trials with botulism outbreaks. The chi-square log-rank statistic for each enclosure was converted to a z-statistic with the appropriate sign (Crowley and Breslow 1984), and significance was determined by comparing the z-statistic to tables of critical values for a 2-sided distribution. Overall trends in the survival rates between these cohorts were analyzed by pooling the z-statistic from each enclosure to produce a composite z-statistic (Anderson and Burnham 1976:23). This procedure increased the statistical power to detect consistent differences in the probability of intoxication by combining results of each individual trial.

RESULTS

Sentinel mallards contracted botulism in 3 of 6 trials. Morbidity and mortality of sentinels in all 3 trials began 1–4 days after maggots were observed on carcasses and continued for as long as 17 days. The largest outbreaks occurred in trials 1 and 5 (Table 1). In both of these trials, the cumulative probability of botulism intoxication was significantly higher ($P \leq 0.05$) in enclosures containing carcasses of either birds dead from botulism or euthanized birds than in enclosures without carcasses (Table 1). Average daily probabilities of intoxication were 0.021 (trial 1) and 0.036 (trial 5) in enclosures with carcasses of euthanized birds and 0.016 (for each of trials 1 and 5) in enclosures with carcasses of birds dead from botulism. Botulism also occurred in the control enclosures in both trials 1 and 5, with an average daily probability of 0.005, but the estimated relative risk of intoxication was an average of 4.5× higher in enclosures with carcasses.

Sentinels from all age and sex cohorts succumbed to botulism during the 3 trials in which outbreaks occurred. AHY sentinels had a greater ($P \leq 0.01$) probability of intoxication from botulism than HY sentinels in 2 of the 4 enclosures with carcasses in trials 1 and 5 (Table 2). Combining results from all enclosures and trials, AHY sentinels had a higher overall probability of intoxication than HY sentinels (composite $z = 2.871$, $P \leq 0.01$). Intoxication of male sentinels was not significantly different ($P > 0.10$) than females in any of the individual enclosures (Table 2). However, the trend in all enclosures was similar with males becoming sick or dying at a higher rate than females. The composite z-statistic indicated that males had a significantly higher overall prob-

Table 1. Cumulative probability of botulism intoxication[a] in sentinel mallards placed in enclosures with carcasses of ducks that died of botulism, carcasses of ducks that were euthanized as healthy birds, and no carcasses (control) at the Sacramento National Wildlife Refuge, Willows, California, in 1988.

			Cumulative probability of intoxication		
Trial	Date	Pool	Intoxicated carcasses	Euthanized carcasses	No carcasses
1	19 Jul–10 Aug	10	0.39[b]	0.55[b]	0.15
2	22 Aug–12 Sep	10	0.10[b]	0.07	0.00
3	16 Sep–10 Oct	1	0.00	0.00	0.00
4	21 Sep–10 Oct	10	0.00	0.00	0.00
5	20 Oct–18 Nov	1	0.40[b]	0.70[b]	0.13
6	21 Nov–12 Dec	1	0.00	0.00	0.00

[a] Probability of intoxication determined with the Kaplan–Meier survival estimator (Kaplan and Meier 1958).
[b] Indicates intoxication significantly ($P \leq 0.05$) different from corresponding control by 1-tailed z-statistic.

ability of intoxication than females (composite $z = -1.971$, $P \leq 0.025$). Botulism intoxication was lowest in HY females and progressively increased in AHY females, HY males, and AHY males.

Maggots were observed on an average of 83% (SE = 4.9%) of carcasses in the first 5 trials; no maggots were observed on any carcass in trial 6 (Table 3). Maggots were first observed on the exterior of some carcasses within 2–4 days after placement in enclosures, and most had visible maggots within 5–6 days. The average length of time maggots were observed on carcasses was 6.5 days (SD = 2.8). The period of maggot availability during each trial varied from 9–15 days (Table 3).

An average of 41% (SE = 9%) of maggot-infested carcasses had maggots that contained detectable type-C botulism toxin (Table 3). Maggots with high levels of toxin (≥100 MLD/g) were found on an average of 16% (SE = 6%) of the maggot-infested carcasses of euthanized birds and 10% (SE = 4%) of the maggot-infested carcasses of botulism-intoxicated birds. These proportions were not different by chi-square analysis in any of the trials. Toxic maggots were found as early as 3 days after carcass placement in the enclosures (Table 3) and persisted as long as 10 days on some carcasses. Peak toxin titers of 50,000 MLD/g of maggots were detected 5–10 days after carcass placement in enclosures, coinciding with the period of peak maggot infestation. The highest toxin titer recorded in this study was nearly

Table 2. Cumulative probability of botulism intoxication[a] in after-hatch-year (AHY), hatch-year (HY), female (F), and male (M) sentinel mallards in wetland enclosures with carcasses of birds dead from botulism (A) and carcasses of euthanized birds (B) at the Sacramento National Wildlife Refuge, Willows, California, in 1988.

Trial	Carcass type	AHY	HY	z[b]	F	M	z[b]
1	A	0.64	0.07	3.444[c]	0.36	0.40	−0.074
	B	0.59	0.51	0.486	0.53	0.55	−0.609
2	A	0.07	0.13	−0.577	0.00	0.17	−1.585
	B	0.00	0.13	−1.439	0.00	0.11	−1.095
5	A	0.54	0.31	1.171	0.38	0.42	−0.112
	B	0.90	0.35	3.950[c]	0.56	0.77	−1.498
Composite z				2.871[c]			−2.030[d]

[a] Probability of intoxication determined with the Kaplan–Meier survival estimator (Kaplan and Meier 1958).
[b] z-statistics were calculated by comparing AHY with HY sentinels and male with female sentinels in each enclosure.
[c] Indicates z-statistic significant at $P \leq 0.025$.
[d] Indicates z-statistic significant at $P \leq 0.01$.

Table 3. Maggot infestations of 20 mallard carcasses placed in marsh enclosures in 6 experimental trials at the Sacramento National Wildlife Refuge, Willows, California, in 1988.

Trial dates (trial no.)	Average daily maximum temperature[a] (C)	Carcasses with maggots	Percent maggot-infested carcasses with toxic maggots[b] (*n*)	Days after carcasses placed in marsh		
				Maggots visible	Peak infestation[c]	Toxic maggots[b] present
19 Jul–10 Aug (1)	35.5	16	69 (11)	2–13	4–8	3–13
22 Aug–12 Sep (2)	36.7	20	45 (9)	3–16	5–9	3–12
16 Sep–10 Oct (3)	30.6	16	19 (3)	4–19	9–17	6, 7, 11–16
21 Sep–10 Oct (4)	30.6	14	43 (6)	4–15	6–12	4–15
20 Oct–18 Nov (5)	22.5	17	29 (5)	3–16	5–12	3–12
21 Nov–12 Dec (6)	16.8	0				

[a] Data obtained from the National Weather Service, Tehama–Colusa Station, Willows, California.
[b] Maggots containing type-C botulism toxin detectable by mouse-neutralization tests.
[c] More than half of carcasses >50% maggot-infested.

100,000 MLD/g and was detected in maggots that developed on the carcass of a euthanized bird.

DISCUSSION

In 3 of 6 trials conducted at the SNWR, botulism killed sentinel mallards enclosed with 2 types of carcasses, mallards that died from avian botulism and mallards that were euthanized as healthy birds. Maggots containing high levels of type-C botulism toxin were found on both types of carcasses within 3 days, and mortality in sentinels occurred only after maggots were available. The risk of botulism intoxication for sentinels was significantly higher in enclosures with carcasses than without, confirming an earlier report (Hunter 1970) of carcass-associated botulism in mallards enclosed with carcasses.

The occurrence of botulism in sentinel mallards was not evenly distributed among cohorts. When we used composite-z analysis to evaluate trends that occurred over all trials, we found that AHY sentinels contracted botulism at a greater rate than did HY sentinels, and males contracted botulism at a greater rate than did females. Hammond (1950) also indicated that males died in greater numbers than females during botulism outbreaks in North Dakota but assumed this resulted from a higher proportion of males at risk; however, no data were collected to verify this conclusion. In limited experimental studies of mallards inoculated with botulism toxin intraperitoneally, no differential age or sex susceptibility to botulism intoxication could be detected (Natl. Wildl. Health Res. Cent., Madison, Wis., unpubl. data, 1988). However, the oral route of inoculation was not tested, and there may be some differences among waterfowl age and sex cohorts in the absorption of botulism toxin through the gut lining. Another explanation may be age- or sex-specific preferences in food habits among sentinels that led to a greater rate of botulism intoxication in AHY males.

We made several assumptions in the analysis of sentinel survival data that potentially limit the scope of inference from these experiments. First, we regarded individual sentinel mallards as separate observations and assumed that the probability of contracting botulism was independent among sentinels. Alternatively, if sentinel mallards were highly dependent, we would expect most of the botulism cases among sentinels within an enclosure to occur over a short period of time. In fact, sentinels became sick or died from botulism over a period of many days (up to 17 days in 1 trial), supporting our contention that sentinel birds were not acting as a single group but were, to a large extent, independent. In addition, each time an indi-

vidual died or was removed, it was replaced by a new sentinel that was unfamiliar with the enclosure and other birds.

Secondly, we assumed treatments within trials were independent. However, several sentinels contracted botulism in enclosures without carcasses in the 2 trials with the greatest mortality. Because there were no ongoing outbreaks in wild waterfowl in other parts of the wetland and no other carcasses were found after thorough searches, the source of botulism toxin in these control enclosures was probably toxic maggots or other invertebrates that floated or dispersed into these areas from adjacent enclosures with carcasses. In both trials, the nearest carcass with toxic maggots was at least 20 m from the control enclosure. Maggot-infested carcasses attract other invertebrates, which can ingest maggots and also become toxic (Duncan and Jensen 1976). These predatory invertebrates may play an important role in the spread of carcass-associated botulism. In retrospect, our control enclosures should have been placed a greater distance from enclosures with carcasses. Even so, the lack of independence between enclosures within a trial simply makes the statistical comparisons, and thus our conclusions, more conservative. The risk of botulism intoxication to sentinel mallards at SNWR was much higher in the presence of carcasses, substantiating the use of carcass pickup to prevent botulism. However, our study was restricted to 1 wetland complex and 1 season of data collection, and more research may be needed to confirm our results.

We also found that maggot-infested carcasses of euthanized ducks were as likely to contain toxic maggots as carcasses of ducks that died from botulism. Duncan and Jensen (1976) suggested that maggots on birds that died of botulism developed higher levels of toxin than those dead from other causes, but we did not detect a difference in our study. These findings, along with those of Bell et al. (1955), Hunter (1970), Graham and Smith (1977), Smith and Turner (1987), and Brand et al. (1988), support the hypothesis that postmortem toxin formation drives the carcass–maggot cycle of botulism. Type-C botulism spores are known to be abundant in marsh sediments at SNWR (Sandler 1990) and appear to be common in tissues of live, healthy waterfowl. We recently determined that 21 of 40 healthy sentinel mallards maintained on a wetland at SNWR for 2 months had detectable type-C botulism spores in their livers or ceca (Natl. Wildl. Health Res. Cent., Madison, Wis., unpubl. data, 1990). All carcasses, regardless of cause of death, should be considered as potential sources of botulism toxin.

The occurrence of carcass–maggot cycles of botulism is dependent on a number of factors in addition to the presence of carcasses with toxigenic botulism spores, including fly density and environmental conditions that facilitate fly egg-laying, maggot development, and maggot dispersal from carcasses. In our study, maggots were detected on carcasses within 3 days of placement in the marsh in 3 trials in which botulism occurred in sentinels, and >50% were infested with maggots within 4–5 days. In 2 trials conducted in late September and early October, in which no botulism occurred, maggots were not observed on carcasses until days 6 and 4, and <50% were infested with maggots until days 8 or 9. Wind speeds were higher (>24 km/hr) in late September and early October during these 2 trials than during the other trials (≤14 km/hr; Natl. Weather Serv., Tehama–Colusa Station, Willows, Calif., unpubl. data), especially during the first 6 days. High wind speeds may have hindered flies from landing and laying eggs on the carcasses, thus delaying the onset of maggot infestations and the availability of toxic maggots. Temperature also influences fly activity and maggot development on carcasses. Average maximum daily temperatures were lower during the last trial (16.8 C), when no maggots were observed on any carcass, than during previous trials (22.5–35.5 C). In wetlands in Great Britain, a daily

maximum temperature of ≥21 C was required for botulism outbreaks to occur (Smith 1979).

MANAGEMENT IMPLICATIONS

The results of this study demonstrate the importance of carcass pickup as a management tool to prevent and control avian botulism. At SNWR, the average daily relative risk of contracting botulism in enclosures with carcasses in the trials with the greatest mortality was 4.5× higher than in enclosures without carcasses. Thus, intensive carcass pickup (the absence of carcasses) improved the daily survival of sentinels in our study. This index of relative risk approximates the benefits of carcass pickup during an avian botulism outbreak. Although it is a difficult and time-consuming task, waterfowl managers should monitor wetlands and remove avian carcasses, regardless of the cause of death.

Our study also revealed differences in the probability of botulism intoxication between adult and juvenile sentinels and male and female sentinels. We do not know if these differences occur in wild birds. Differential deaths from botulism among age and sex cohorts could have important ramifications for population management and warrant further consideration and study.

Acknowledgments.—This research was supported by the U.S. Fish and Wildlife Service. The cooperation and assistance of personnel from the Sacramento National Wildlife Refuge was appreciated. Technical assistance was provided by R. J. Dusek, D. R. Goldberg, G. H. Golet, S. R. Smith, and B. B. Stone. Statistical advice was provided by M. D. Samuel, National Wildlife Health Research Center. We are grateful to C. J. Brand, R. M. Duncan, J. W. Hardin, and M. D. Samuel for critical review of this manuscript.

LITERATURE CITED

ANDERSON, D. R., AND K. P. BURNHAM. 1976. Population ecology of the mallard. VI. The effect of exploitation on survival. U.S. Fish and Wildl. Serv. Resour. Publ. 128. 66pp.

BELL, J. F., G. W. SCIPLE, AND A. A. HUBERT. 1955. A microenvironment concept of the epizootiology of avian botulism. J. Wildl. Manage. 19:352–357.

BRAND, C. J., R. M. WINDINGSTAD, L. M. SIEGFRIED, R. M. DUNCAN, AND R. M. COOK. 1988. Avian morbidity and mortality from botulism, aspergillosis and salmonellosis at Jamaica Bay Wildlife Refuge, New York, USA. Colonial Waterbirds 11:284–292.

CROWLEY, J., AND N. BRESLOW. 1984. Statistical analysis of survival data. Annu. Rev. Public Health 5: 385–411.

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.

EKLUND, M. W., F. T. POYSKY, K. OGUMA, H. IIDA, AND K. INOUE. 1987. Relationship of bacteriophages to toxin and hemagglutinin production by *Clostridium botulinum* types C and D and its significance in avian botulism outbreaks. Pages 191–222 *in* M. W. Eklund and V. R. Dowell, Jr., eds. Avian botulism: an international perspective. Charles C Thomas, Publ., Springfield, Ill.

Graham, J. M., and G. R. Smith. 1977. Observations on the possible invasiveness of *Clostridium botulinum* for waterfowl. Res. Vet. Sci. 22:343–346.

HAMMOND, M. C. 1950. Some observations on sex ratio of ducks contracting botulism in North Dakota. J. Wildl. Manage. 14:209–214.

HUNTER, B. F. 1970. Ecology of waterfowl botulism toxin production. Trans. N. Am. Wildl. Nat. Resour. Conf. 35:64–72.

KAHN, H. A. 1983. An introduction to epidemiologic methods. Oxford Univ. Press, New York, N.Y. 166pp.

KAPLAN, E. L., AND P. MEIER. 1958. Nonparametric estimation from incomplete observations. J. Am. Stat. Assoc. 53:457–481.

LEE, V. H., S. VADLAMUDI, AND R. P. HANSON. 1962. Blowfly larvae as a source of botulinum toxin for game farm pheasants. J. Wildl. Manage. 26:411–413.

MALCOLM, J. M. 1982. Bird collisions with a power transmission line and their relation to botulism at a Montana wetland. Wildl. Soc. Bull. 10:297–304.

MAYFIELD, H. 1961. Nesting success calculated from exposure. Wilson Bull. 73:255–261.

———. 1975. Suggestions for calculating nest success. Wilson Bull. 87:446–456.

MITCHELL, W. R., AND S. ROSENDAL. 1987. Type C botulism: the agent, host susceptibility, and predisposing factors. Pages 55–71 *in* M. W. Eklund and V. R. Dowell, Jr., eds. Avian botulism: an international perspective. Charles C Thomas, Publ., Springfield, Ill.

POLLOCK, K. H., S. R. WINTERSTEIN, C. M. BUNCK, AND P. D. CURTIS. 1989. Survival analysis in telemetry studies: the staggered entry design. J. Wildl. Manage. 53:7–15.

QUORTRUP, E. R., AND R. L. SUDHEIMER. 1943. Detection of botulinus toxin in the bloodstream of wild ducks. J. Am. Vet. Med. Assoc. 102:264–266.
SANDLER, R. 1990. Microbial interactions and population dynamics in relation to avian botulism. M.S. Thesis, Univ. Wisconsin, Madison. 79pp.
SMITH, L. P. 1979. The effect of weather on the incidence of botulism (*Clostridium botulinum*) in waterfowl. Agric. Meteorol. 20:483–488.
SMITH, G. R., AND A. TURNER. 1987. Factors affecting the toxicity of rotting carcasses containing *Clostridium botulinum* type C. Epidemiology and Infect. 98:345–354.
WOBESER, G. 1987. Control of botulism in wild birds. Pages 339–348 *in* M. W. Eklund and V. R. Dowell, Jr., eds. Avian botulism: an international perspective. Charles C Thomas, Publ., Springfield, Ill.
———, S. MARSDEN, AND R. J. MACFARLANE. 1987. Occurrence of toxigenic *Clostridium botulinum* type C in the soil of wetlands in Saskatchewan. J. Wildl. Dis. 23:67–76.

Received 8 March 1991.
Accepted 24 December 1991.
Associate Editor: Ringelman.

***Wildl. Soc. Bull.* 20:182–188, 1992**

SURVIVAL AND REPRODUCTION OF GAME-FARM FEMALE MALLARDS AT REMINGTON FARMS, MARYLAND

JOHN D. STANTON III,[1] ***Fisheries and Wildlife Program, Forestry Department, North Carolina State University, Raleigh, NC 27695***

EDWARD C. SOUTIERE,[2] ***Remington Farms, Chestertown, MD 21620***

RICHARD A. LANCIA, ***Fisheries and Wildlife Program, Forestry Department, North Carolina State University, Raleigh, NC 27695***

Population declines in mallards (*Anas platyrhynchos*) and other North American duck species have prompted some states, private groups, and individuals to release game-farm mallards. Maryland's Department of Natural Resources (DNR), private groups, and some individuals in Maryland began releasing game-farm mallards in the late 1940's and 1950's (V. D. Stotts, unpubl. rep., U.S. Fish and Wildl. Serv., Laurel, Md., 1987). These releases were intended to establish local breeding populations, supplement decreasing numbers of wild conspecifics, and provide increased hunting opportunities.

Recently, 120,000–140,000 game-farm mallards have been released annually along the eastern shore of Chesapeake Bay (Soutiere 1986). The Maryland DNR has released 20,000–40,000 8-week-old game-farm mallards each summer (W. F. Harvey, Maryland Dep. Nat. Resour., Wye Mills, pers. commun., 1989), whereas about 100,000 game-farm mallards were released by Maryland landowners (L. Johnson, unpubl. rep., Grand Natl. Waterfowl Assoc., Cambridge, Md., 1990). Soutiere (1986) described 3 such release programs in Maryland.

The percentages of released game-farm mallards that survive to breed and reproduce are unknown (Soutiere 1989). To our knowledge, only Schladweiler and Tester (1972) used radiotelemetry data to estimate survival of 6-week-old game-farm mallards. Other studies of survival of game-farm mallards have been

[1] Present address: Lake Ophelia National Wildlife Refuge, U.S. Fish and Wildlife Service, P.O. Box 256, Marksville, LA 71351.
[2] Present address: Tudor Farms, 3675 Decoursey Bridge Road, Cambridge, MD 21613.