

LEAD EXPOSURE AND RECOVERY RATES OF BLACK DUCKS BANDED IN TENNESSEE

Authors: Samuel, Michael D., Bowers, E. Frank, and Franson, J. Christian

Source: Journal of Wildlife Diseases, 28(4) : 555-561

Published By: Wildlife Disease Association

URL: <https://doi.org/10.7589/0090-3558-28.4.555>

BioOne Complete (complete.BioOne.org) is a full-text database of 200 subscribed and open-access titles in the biological, ecological, and environmental sciences published by nonprofit societies, associations, museums, institutions, and presses.

Your use of this PDF, the BioOne Complete website, and all posted and associated content indicates your acceptance of BioOne's Terms of Use, available at www.bioone.org/terms-of-use.

Usage of BioOne Complete content is strictly limited to personal, educational, and non - commercial use. Commercial inquiries or rights and permissions requests should be directed to the individual publisher as copyright holder.

BioOne sees sustainable scholarly publishing as an inherently collaborative enterprise connecting authors, nonprofit publishers, academic institutions, research libraries, and research funders in the common goal of maximizing access to critical research.

LEAD EXPOSURE AND RECOVERY RATES OF BLACK DUCKS BANDED IN TENNESSEE

Michael D. Samuel,¹ E. Frank Bowers,² and J. Christian Franson¹

¹U.S. Fish and Wildlife Service, National Wildlife Health Research Center, 6006 Schroeder Road, Madison, Wisconsin 53711, USA

²U.S. Fish and Wildlife Service, Refuges and Wildlife, 75 Spring Street S.W., Atlanta, Georgia 30303, USA

ABSTRACT: American black ducks (*Anas rubripes*) wintering in Tennessee during 1986 to 1988 were tested for exposure to lead. Twelve percent of the birds had blood lead concentrations exceeding 0.2 ppm. Significant differences in the prevalence of lead exposure were found for adults (14.4%) and juveniles (8.2%). Exposed birds had higher blood lead concentrations at one study site, corresponding with a lower survival index.

Key words: *Anas rubripes*, American black ducks, band recovery, lead poisoning, Tennessee.

INTRODUCTION

Lead poisoning long has been recognized as an important disease of waterfowl (Bellrose, 1959). Feierabend (1983) estimated that lead poisoning killed 1.6 to 3.8 million North American waterfowl when fall flights ranged between 80 and 130 million birds. Beginning in 1976, nontoxic shot zones were established, and in 1991 mandatory use of nontoxic shot for hunting waterfowl went into effect throughout the United States in an effort to reduce the availability of lead shot. However, decades of hunting have resulted in high densities of lead shot on some areas (Sanderson and Bellrose, 1986; U.S. Fish and Wildlife Service, 1986) which will continue to be ingested by waterfowl (Mauser et al., 1990; DeStefano et al., 1991). In addition, non-compliance with nontoxic shot regulations (Simpson, 1989) and continued use of lead shot in Canada (Schwab and Daury, 1989) will provide new sources of lead shot for migratory waterfowl. Consequently, eliminating or reducing the effects of lead shot on birds and monitoring changes in lead exposure are still important aspects of waterfowl management.

Ingestion of lead shot by waterfowl has been well documented (Sanderson and Bellrose, 1986). When investigating the significance of lead exposure in waterfowl, most investigators have relied on the prevalence of ingested lead shot in gizzards of hunter-harvested birds. However, the use-

fulness of ingested shot to estimate lead exposure in wild waterfowl has been questioned because birds with ingested lead shot may be more vulnerable to hunting (Jordan and Bellrose, 1951; Bellrose, 1959; Anderson and Havera, 1985; Sanderson and Bellrose, 1986; DeStefano, 1989). Blood lead is a more sensitive method for measuring lead exposure in live waterfowl (Anderson and Havera, 1985), is a better indicator of the amount of lead absorbed by birds, and has been correlated with physiological consequences (Dieter and Finley, 1978; Dieter and Finley, 1979; Anderson and Havera, 1985). In addition, the adsorption and toxicity of ingested lead depends strongly on the type of food consumed (Sanderson and Bellrose, 1986). We report the prevalence of lead exposure, based on blood lead analysis, of black ducks wintering in Tennessee (USA) prior to implementation of steel shot regulations and we also evaluate the possible effect of lead exposure on survival.

MATERIALS AND METHODS

Cross Creeks National Wildlife Refuge (NWR) (36°20'N, 87°40'W) is located along the Cumberland River in northwestern Tennessee (USA). The refuge extends for 16 km on either side of Barkley Lake near the town of Dover. The refuge consists of hardwood forest, cropland and brush, and wetlands in about equal proportions. Since the refuge was established in 1964, only 4 yr of waterfowl hunting (1984 to 1987) have been allowed (steel shot required) on approximately 120 ha of the 3,650-ha refuge. Major

wintering duck species include mallards (*Anas platyrhynchos*), black ducks (*Anas rubripes*), and American wigeon (*Anas americana*). Waterfowl populations typically peak in December and January at an estimated 60,000 ducks, including about 7,000 black ducks.

Tennessee NWR (35°50'N, 87°5'W) contains three separate management units along the Tennessee River in western Tennessee. The Duck River Unit includes >10,500 ha of moist soil units, open water, upland hardwoods, and croplands. A limited goose hunt was allowed on the unit in 1967 and 1968. Otherwise, no waterfowl hunting has been allowed since the refuge opened in 1945. Major wintering duck species include mallard, black duck, and American wigeon. Approximately 300,000 ducks use the refuge during fall and winter migration, with numbers peaking during December and January. Black duck counts usually peak at 10,000 to 12,000 birds annually. Combined surveys at Cross Creeks and Tennessee NWRs have accounted for approximately one-third of the mid-winter black duck count in the Mississippi Flyway (Rusch et al., 1989).

Extensive hunting with lead shot has occurred near the boundaries of both Cross Creeks and Tennessee NWR's. Steel shot was required in the vicinity of Cross Creeks NWR beginning in 1989. Beginning in 1990, steel shot also was required for waterfowl hunting in areas surrounding the Tennessee NWR.

Black ducks at Tennessee (Duck River Unit) and Cross Creeks NWR's were trapped using cannon nets and swim-in traps baited with corn during January, February, or March, 1986 to 1988. Age and sex of each bird were determined according to plumage and cloacal characteristics. One to 2 ml of blood was obtained from the jugular vein with a 5 cc heparinized syringe. Blood was frozen and stored for subsequent analysis at the National Wildlife Health Research Center, Madison, Wisconsin (USA), to determine blood lead concentrations. The whole heparinized blood was diluted ten-fold in a mixture of 0.5% alkylaryl polyether alcohol (Triton X-100, J. T. Baker Chemical Company, Phillipsburg, New Jersey USA) and 0.2% ammonium dihydrogen phosphate. The diluted samples were stirred immediately prior to the assay. Lead concentrations were determined using a Perkin-Elmer HGA-400 graphite furnace with an AS-40 autosampler coupled to a Model 2380 atomic absorption spectrophotometer (Perkin Elmer Corporation, Norwalk, Connecticut USA). The method was essentially that of Fernandez and Hilligoss (1982), except for the difference in blood dilution.

Blood lead concentrations ≥ 0.2 ppm were considered to represent exposure to lead above

normal background levels (Friend, 1985). Birds were classified as either exposed (≥ 0.2 ppm) or unexposed (< 0.2 ppm) to lead. A stepwise logistic regression (Dixon et al., 1985) was used to evaluate factors influencing variations in proportions of exposed birds. The median blood lead concentrations of exposed birds were compared using a nonparametric Kruskal-Wallis test (Daniel, 1978) by analysis of variance on ranks (SAS Institute, Inc., 1987). Because sample sizes were generally small, statistical tests were considered significant at the 0.10 level.

Black ducks tested for lead exposure were banded with U.S. Fish and Wildlife Service (USFWS) aluminum legbands. Band recovery and recapture records were obtained from the USFWS Bird Banding Laboratory, Laurel, Maryland (USA). The proportion of birds recovered and recaptured was used as an index of birds surviving ≥ 1 yr after banding. Recaptures of birds at either Tennessee NWR or Cross Creeks NWR were not considered because of the potential bias caused by differential retrapping of local birds at each refuge. Stepwise logistic regression was used to identify factors that resulted in significant differences in this survival index.

RESULTS

Four hundred thirty-five black ducks were tested for lead exposure during 1986 to 1988 (Table 1). A large percentage of the black ducks bled at Cross Creeks NWR were adults; 12 unexposed ducks were of unknown age and sex and were excluded from the statistical analyses related to lead exposure and survival. Fifty-one black ducks had blood lead concentrations ≥ 0.2 ppm, an 11.7% prevalence of birds exposed to lead. Based on stepwise logistic regression, age was the only variable significantly ($P = 0.05$) associated with exposure to lead. Adult black ducks (14.4%) were almost twice as likely to have elevated blood lead concentrations as juveniles (8.2%). No differences were found for refuge ($P = 0.47$), year ($P = 0.72$), or sex ($P = 0.64$), after the influence of age was removed.

Median blood lead concentrations of exposed birds were evaluated by nonparametric one-way analysis of variance for differences in age, sex, year, and refuge. No significant differences in median blood

TABLE 1. Number tested and percentage of adult/juvenile and male/female black ducks exposed to lead (≥ 0.2 ppm blood lead) at Cross Creeks and Tennessee National Wildlife Refuges 1986–88.

Year	Age	Sex	Cross Creeks NWR		Tennessee NWR	
			Number tested	% positive	Number tested	% positive
1986	Ad	Female			40	20.0
		Male			28	0.0
	Juv	Female			39	7.7
		Male			27	7.4
	Total				134	9.7
1987	Ad	Female	14	28.6	15	6.7
		Male	22	22.7	16	12.5
	Juv	Female			7	0.0
		Male	2	0.0	12	8.3
	Total		38	23.7	50	8.0
1988	Ad	Female	23	17.4	26	15.4
		Male	60	11.7	20	15.0
	Juv	Female	11	0.0	10	0.0
		Male	7	14.3	44	13.6
	Total		101	11.9	100	13.0
All	Ad	Female	37	21.6	81	16.1
		Male	82	14.6	64	7.8
	Total		119	17.6	145	12.4
	Juv	Female	11	0.0	56	5.4
		Male	9	11.1	83	10.8
Total		20	5.0	139	8.6	

lead concentrations were found among age classes ($P = 0.44$), sex classes ($P = 0.63$), or year ($P = 0.42$). However, blood lead concentrations were higher ($P = 0.09$) at Cross Creeks NWR than at Tennessee NWR (Fig. 1).

Twenty-three birds tested for blood lead concentration were either recovered by hunters ($n = 22$) or recaptured ($n = 1$) during banding operations at least one hunting season after banding. Six birds were shot in Ontario (Canada), four in Tennessee, two each in Michigan (USA) and North Carolina (USA), and single recoveries were reported in Alabama (USA), Arkansas (USA), Ohio (USA), Kentucky (USA), Mississippi (USA), South Carolina (USA), Wisconsin, and Quebec (Canada). One bird was recaptured during banding operations in Ontario. Two of the banded ducks with elevated blood concentrations (0.20 and 0.21 ppm) were recovered by

hunters (one in Ontario and one in Alabama) and one was recaptured in Ontario (0.37 ppm). Based on logistic regression, the proportion of black ducks surviving at least 1 yr was higher ($P = 0.087$) at Tennessee NWR (19 of 284) than at Cross Creeks NWR (4 of 139). No differences in

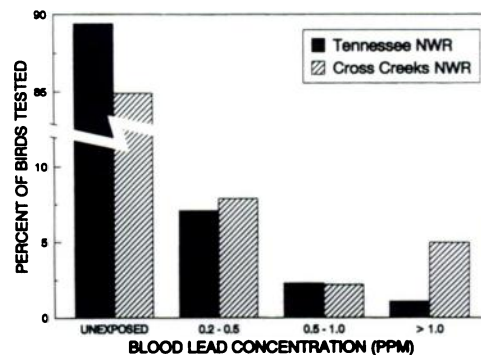


FIGURE 1. Blood lead concentration (ppm) for black ducks at Cross Creeks and Tennessee NWRs 1986–88.

survival were found, based on sex ($P = 0.26$), age ($P = 0.69$), or blood lead exposures ($P = 0.37$) after the effect of refuge differences was removed.

DISCUSSION

Lead poisoning has caused mortality of 2 to 3% of the fall population of waterfowl in North America for more than 30 years (Bellrose, 1959). For mallards, annual losses have been estimated at 4% in the Mississippi Flyway and 3 to 4% in the other flyways (Bellrose, 1959). Black ducks have similar or higher prevalence of lead ingestion than mallards in the Mississippi Flyway. Both species also have similar susceptibility to lead toxicity (Rattner et al., 1989); however, the effect of lead may be reduced in black ducks if their diet contains more animal matter than mallards (Chasko et al., 1984). In addition to direct mortality from lead poisoning, lead can cause weight loss (Sanderson and Bellrose, 1986; Finley and Dieter, 1978), neurological dysfunction (Dieter and Finley, 1978, 1979), and reduced immunological function and disease resistance (Trust et al., 1990; Rocke and Samuel, 1991). Dieter (1979) demonstrated that delta-aminolevulinic acid dehydratase (ALAD) enzyme activity was inhibited in canvasbacks (*Aythya valisineria*) at blood lead concentrations of 0.2 ppm. At lead concentrations >0.5 ppm, canvasbacks exhibited abnormal levels of blood ALAD activity (<50 units) which closely reflect liver and brain ALAD enzyme activity (Dieter and Finley, 1979). Reduced ALAD activity in the brain has been associated with biochemical lesions and cerebellar damage caused by lead poisoning (Dieter and Finley, 1979). These direct and indirect factors probably contribute to an increased mortality rate of black ducks and other species exposed to lead.

Bellrose (1959) reported that 8.3% of black ducks obtained from hunters between 1938 and 1953 had at least one lead shot in their gizzards, with a higher exposure rate in the Mississippi Flyway

(21.0%) compared to the Atlantic Flyway (6.1%). Black ducks collected in Tennessee had a prevalence of 6% (Bellrose, 1959), but sample size was relatively small ($n = 17$). Stendell et al. (1979) found an increase in lead concentrations in wing-bones of black ducks from north to south, and suggested an increased exposure as birds migrated south. The prevalence of ingested shot in black duck gizzards for 1976 to 1984 also was greater in the Mississippi Flyway than in the Atlantic Flyway (U.S. Fish and Wildlife Service, 1986), but no results were reported for Tennessee during that time period. Longcore et al. (1982) found a 6.9% prevalence of ingested lead shot in black ducks from Maine (USA) for 1976–80. Di Giulio and Scanlon (1984) found a prevalence of ingested lead shot of 18% for black ducks in the Chesapeake Bay region. In recent surveys (U.S. Fish and Wildlife Service, 1986), the prevalence of lead pellets in black duck gizzards (8.8%) has been documented for the states of Maine, New York (USA), Virginia (USA), Michigan, Indiana (USA), and Ohio (Rusch et al., 1989). Schwab and Daury (1989) found that 4 (9.8%) of 41 gizzards from black ducks collected by hunters in Nova Scotia (Canada), during the 1987 hunting season contained lead shot.

Previous studies of ingested lead in black ducks are not directly comparable to our results (Scheuhammer, 1989). Birds with ingested lead can be more vulnerable (1.6 to 2 times) to hunting and are more likely to be included in gizzard surveys than ducks without ingested shot (Bellrose, 1959). Gizzard examination can underestimate exposure because shot may not be detected in 20 to 30% of gizzards containing lead pellets (Anderson and Havera, 1985). Also, lead shot will be completely eroded or pass through the digestive system within 3 wk in >90% of the affected birds (Dieter and Finley, 1978). In contrast, blood lead concentrations can remain elevated for >45 days after lead ingestion (Franson et al., 1986). Therefore, the prevalence of lead we found for black ducks

in Tennessee (11.7%) was difficult to compare with values reported in earlier studies. Despite this limitation, blood lead concentrations represent a more realistic measure of the potential physiological impacts of lead exposure on black ducks (Anderson and Havera, 1985).

Prevalence of exposure to lead was significantly different between adults (14.4%) and juveniles (8.2%). The reasons for these differences are unknown, but possible explanations include: (1) differences in food habits, (2) differences in habitat preference, (3) a higher mortality of juvenile black ducks exposed to lead, (4) higher deposition of blood lead in the bones of juvenile black ducks. We found no published studies of food habits of black ducks in the lower Mississippi Flyway to evaluate age-related feeding differences. Reinecke et al. (1982) found that adult female black ducks had a higher nutrient reserve than juvenile females. Thus, juvenile black ducks may be more physically stressed than adults during winter and possibly more susceptible to lead poisoning mortality. Conroy et al. (1987) found that juvenile black ducks in poor body condition spent more time foraging in some habitats than did adult or juvenile birds in good body condition. Conroy et al. (1989) also found that juvenile birds were more vulnerable to non-hunting mortality than adults during the winter. Stendell et al. (1979) reported that only juvenile black ducks had slightly higher levels of lead in their wing bones than adults. Sanderson and Bellrose (1986) suggested that higher wing bone lead means that juvenile black ducks either ingest more shot than adults or that the ossification of bone in juveniles takes up a greater share of blood lead than occurs in other species. Further research will be required to explain the higher lead exposure rate for adult black ducks in Tennessee.

We found indications of lower survival and significantly lower recovery of black ducks banded at Cross Creeks NWR than those banded at Tennessee NWR. Lead poisoning is one explanation for the ap-

parent lower survival of black ducks banded at Cross Creeks NWR; however, alternative explanations are also possible. Ducks banded at Cross Creeks had higher median blood lead concentrations than birds banded at Tennessee NWR. Blood concentrations in ducks at Cross Creeks also had much higher values (≤ 9.8 ppm) than birds at Tennessee, and 33% of the lead-exposed birds at Cross Creeks NWR had > 1.0 ppm compared to 10% of the Tennessee NWR birds. We suspect that management differences related to the amount and types of food available (particularly invertebrates vs. grains) at the two refuges could have influenced the absorption and toxicity of ingested lead. Also, the proximity of public waterfowl hunting areas at Cross Creeks NWR could have resulted in higher ingestion of lead pellets.

We failed to find a statistically significant difference in survival of ducks with ≥ 0.2 ppm blood lead concentrations compared to birds with < 0.2 ppm. A positive relationship would have provided additional evidence to support the correlation of reduced survival with increased blood lead concentrations at our two study sites. However, the lack of a positive relationship may have resulted from the small sample sizes in our study. We estimated (Zar, 1984) that a sample size of $> 2,300$ ducks would have been needed to detect a hypothetical difference of 0.03 in the recovery rates of lead-exposed and unexposed birds (e.g., 0.02 for lead-exposed and 0.05 for unexposed birds) using a Type I error rate (α) of 0.1 and a Type II error rate (β) of 0.2.

In previous studies of lead poisoning, workers have used wild ducks experimentally dosed with lead shot (Bellrose, 1959; Deuel, 1985) or have assessed the prevalence of lead exposure by examining hunter-killed birds (e.g., Sanderson and Bellrose, 1986; Schwab and Daury, 1989). However, few authors (Anderson and Havera, 1985; Mauser et al., 1990) have estimated the exposure prevalence of ducks based on blood lead concentrations. Use of

this method is more likely to represent the natural prevalence of lead exposure in ducks without the bias associated with vulnerability to harvest. In addition, this method provides a more sensitive measure of the physiological effects of lead poisoning. We believe we are the first to report differences in exposure of adult and juvenile black ducks and to report lower survival of birds at a site with greater exposure to lead. A significant reduction in survival also has been found for Canada geese (*Branta canadensis*) exposed to lead shot (DeStefano et al., 1991; National Wildlife Health Research Center, unpubl. data).

MANAGEMENT IMPLICATIONS

Because American black duck populations have been declining throughout eastern North America (Rusch et al., 1989), lead exposure of black ducks wintering in Tennessee should be of concern for several reasons. First, a substantial proportion of the Mississippi Flyway black duck population spends at least part of the winter in Tennessee. Factors affecting the survival and productivity of these birds could have an important impact on black duck population levels within the Mississippi Flyway. Second, exposed birds probably represent a minimum estimate of the lead exposure rate and the effect of lead poisoning on black ducks wintering in Tennessee. Blood lead concentrations have been demonstrated to measure lead exposure for approximately 45 days (Franson et al., 1986). Some birds were undoubtedly exposed to lead, but were able to recover prior to banding; others were unexposed at trapping, but became exposed to lead during the remainder of the winter period. In addition, some birds may have died from lead poisoning before banding operations. Finally, lead exposure continued to occur after the hunting season and represents a potentially additive source of mortality. Periodic monitoring of black duck populations should be conducted to evaluate the

trends in lead exposure following conversion to nontoxic shot.

ACKNOWLEDGMENTS

V. C. Grafe, C. L. Ryan, and the refuge staff at Tennessee and Cross Creeks NWRs collected blood samples during banding operations. Blood lead analyses were conducted by M. R. Smith and D. L. Finley. The manuscript was reviewed by N. T. Weiss, L. N. Locke, T. E. Rocke, C. J. Brand, and M. J. Conroy.

LITERATURE CITED

- ANDERSON, W. L., AND S. P. HAVERA. 1985. Blood lead, protoporphyrin and ingested shot for detecting lead poisoning in waterfowl. *Wildlife Society Bulletin* 13: 26-31.
- BELLROSE, F. C. 1959. Lead poisoning as a mortality factor in waterfowl populations. *Illinois Natural History Survey Bulletin* 27: 235-288.
- CHASKO, G. G., T. R. HOEHN, AND P. HOWELL-HELLER. 1984. Toxicity of lead shot to wild black ducks and mallards fed natural foods. *Bulletin of Environmental Contamination and Toxicology* 32: 417-428.
- CONROY, M. J., G. R. COSTANZO, AND D. B. STOTTS. 1987. Winter movements of American black ducks in relation to natural and impounded wetlands in New Jersey. *In Waterfowl and wetlands symposium: Proceedings of a symposium in waterfowl and wetlands management in the coastal zone of the Atlantic flyway*, W. R. Whitman and W. H. Meredith (eds.). Delaware Coastal Management Program, Delaware Department of Natural Resources and Environmental Control, Dover, Delaware, pp. 31-45.
- , ———, AND ———. 1989. Winter survival of female American black ducks on the Atlantic coast. *The Journal of Wildlife Management* 53: 99-109.
- DANIEL, W. W. 1978. *Applied nonparametric statistics*. Houghton Mifflin Company, Boston, Massachusetts, 503 pp.
- DESTEFANO, S. 1989. Ecological relationships of lead exposure in Canada geese of the Eastern Prairie population. Ph.D. Thesis. University of Idaho, Moscow, Idaho, 97 pp.
- , C. J. BRAND, D. H. RUSCH, D. L. FINLEY, AND M. M. GILLESPIE. 1991. Lead exposure in Canada geese of the Eastern Prairie Population. *Wildlife Society Bulletin* 19: 23-31.
- DEUEL, B. 1985. Experimental lead dosing of northern pintails in California. *California Fish and Game* 71: 125-128.
- DIETER, M. P. 1979. Blood delta-aminolevulinic acid dehydratase (ALAD) to monitor lead contamination in canvasback ducks (*Aythya valisineria*). *In Animals as monitors of environmental pollut-*

- ants, F. W. Nielson, G. Migaki, and D. G. Scarpelli (eds.). National Academy of Sciences, Washington, D.C., pp. 177–191.
- , AND M. T. FINLEY. 1978. Erythrocyte δ -aminolevulinic acid dehydratase activity in mallard ducks: Duration of inhibition after lead shot dosage. *The Journal of Wildlife Management* 42: 621–625.
- , AND ———. 1979. δ -aminolevulinic acid dehydratase enzyme activity in blood, brain, and liver of lead-dosed ducks. *Environmental Research* 19: 127–135.
- Di Giulio, R. T., and P. F. Scanlon. 1984. Heavy metals in tissues of waterfowl from the Chesapeake Bay, USA. *Environmental Pollution (Series A)* 35: 29–48.
- DIXON, W. J., M. B. BROWN, L. ENGELMAN, J. W. FRANE, M. A. HILL, R. I. JENNRICH, AND J. D. TOPOREK. 1985. BMDP statistical software. University of California Press, Berkeley, California, 734 pp.
- FEIERABEND, J. S. 1983. Steel shot and lead poisoning in waterfowl. National Wildlife Federation. Scientific and Technical Series 8, Washington, D.C., 62 pp.
- FERNANDEZ, F. J., AND D. HILLIGOSS. 1982. An improved graphite furnace method for the determination of lead in blood using matrix modification and the L'vov platform. *Atomic Spectroscopy* 3: 130–131.
- FINLEY, M. T., AND M. P. DIETER. 1978. Toxicity of experimental lead-iron shot versus commercial lead shot in mallards. *The Journal of Wildlife Management* 42: 32–39.
- FRANSON, J. C., G. M. HARAMIS, M. C. PERRY, AND J. F. MOORE. 1986. Blood protoporphyrin for detecting lead exposure in canvasbacks. *In* Lead poisoning in wild waterfowl—A workshop, J. S. Feierabend and A. B. Russell (eds.). National Wildlife Federation, Washington, D.C., pp. 32–37.
- FRIEND, M. 1985. Interpretation of criteria commonly used to determine lead poisoning problem areas. U.S. Fish and Wildlife Service Wildlife Leaflet 2, Washington, D.C., 4 pp.
- JORDAN, J. S., AND F. C. BELLROSE. 1951. Lead poisoning in wild waterfowl. *Illinois Natural History Survey Biological Notes* 26, Champaign, Illinois, 27 pp.
- LONGCORE, J. R., P. O. CORR, AND H. E. SPENCER. 1982. Lead shot incidence in sediments and waterfowl gizzards from Merrymetting Bay, Maine. *Wildlife Society Bulletin* 10: 3–10.
- MAUSER, D. M., T. E. ROCKE, J. G. MENSİK, AND C. J. BRAND. 1990. Blood lead concentrations in mallards from Delevan and Colusa National Wildlife Refuges. *California Fish and Game* 76: 132–136.
- RATTNER, B. A., W. J. FLEMING, AND C. M. BUNCK. 1989. Comparative toxicity of lead shot in black ducks (*Anas rubripes*) and mallards (*Anas platyrhynchos*). *Journal of Wildlife Diseases* 25: 175–183.
- REINECKE, K. J., T. L. STONE, AND R. B. OWEN. 1982. Seasonal carcass composition and energy balance of female black ducks in Maine. *The Condor* 84: 420–426.
- ROCKE, T. E., AND M. D. SAMUEL. 1991. Effects of lead shot ingestion on selected cells of the mallard immune system. *Journal of Wildlife Diseases* 27: 1–9.
- RUSCH, D. H., C. D. ANKNEY, H. BOYD, J. R. LONGCORE, F. MONTALBANO, III, J. RINGELMAN, AND V. D. STOTTS. 1989. Population ecology and harvest of the American black duck: A review. *Wildlife Society Bulletin* 17: 379–406.
- SANDERSON, G. C., AND F. C. BELLROSE. 1986. A review of the problem of lead poisoning in waterfowl. *Illinois Natural History Survey Special Publication* 4, Champaign, Illinois, 34 pp.
- SAS INSTITUTE, INC. 1987. SAS/STAT guide for personal computers. Version 6. SAS Institute, Inc., Cary, North Carolina, 1,028 pp.
- SCHEUHAMMER, A. M. 1989. Monitoring wild bird populations for lead exposure. *The Journal of Wildlife Management* 53: 750–765.
- SCHWAB, F. E., AND R. W. DAURY. 1989. Incidence of ingested lead shot in Nova Scotia waterfowl. *Wildlife Society Bulletin* 17: 237–240.
- SIMPSON, S. G. 1989. Compliance by waterfowl hunters with nontoxic shot regulations in central South Dakota. *Wildlife Society Bulletin* 17: 245–248.
- STENDELL, R. C., R. I. SMITH, K. P. BURNHAM, AND R. E. CHRISTENSEN. 1979. Exposure of waterfowl to lead: A nationwide survey of residues in wing bones of seven species, 1972–73. U.S. Fish and Wildlife Service Special Report Wildlife No. 223, Washington, D.C., 11 pp.
- TRUST, K. A., M. W. MILLER, J. K. RINGELMAN, AND I. M. ORME. 1990. Effects of ingested lead on antibody production in mallards (*Anas platyrhynchos*). *Journal of Wildlife Diseases* 26: 316–322.
- U.S. FISH AND WILDLIFE SERVICE. 1986. Final supplemental environmental impact statement—Use of lead shot for hunting migratory birds in the United States. (FES 86-16) Office of Migratory Bird Management, Washington, D.C., 549 pp.
- ZAR, J. H. 1984. Biostatistical analysis. Prentice-Hall, Inc., Englewood Cliffs, New Jersey, 718 pp.

Received for publication 15 July 1991.