



Lead Exposure in Canada Geese of the Eastern Prairie Population

Author(s): Stephen DeStefano, Christopher J. Brand, Donald H. Rusch, Daniel L. Finley,  
Murray M. Gillespie

Source: *Wildlife Society Bulletin*, Vol. 19, No. 1, (Spring, 1991), pp. 23-32

Published by: Allen Press

Stable URL: <http://www.jstor.org/stable/3782411>

Accessed: 01/07/2008 13:18

---

Your use of the JSTOR archive indicates your acceptance of JSTOR's Terms and Conditions of Use, available at <http://www.jstor.org/page/info/about/policies/terms.jsp>. JSTOR's Terms and Conditions of Use provides, in part, that unless you have obtained prior permission, you may not download an entire issue of a journal or multiple copies of articles, and you may use content in the JSTOR archive only for your personal, non-commercial use.

Please contact the publisher regarding any further use of this work. Publisher contact information may be obtained at <http://www.jstor.org/action/showPublisher?publisherCode=acg>.

Each copy of any part of a JSTOR transmission must contain the same copyright notice that appears on the screen or printed page of such transmission.

---

JSTOR is a not-for-profit organization founded in 1995 to build trusted digital archives for scholarship. We work with the scholarly community to preserve their work and the materials they rely upon, and to build a common research platform that promotes the discovery and use of these resources. For more information about JSTOR, please contact [support@jstor.org](mailto:support@jstor.org).

## LEAD EXPOSURE IN CANADA GEESE OF THE EASTERN PRAIRIE POPULATION

STEPHEN DeSTEFANO,<sup>1</sup> *Department of Fish and Wildlife Resources, University of Idaho, Moscow, ID 83843*

CHRISTOPHER J. BRAND,<sup>2</sup> *U.S. Fish and Wildlife Service, National Wildlife Health Research Center, 6006 Schroeder Road, Madison, WI 53711*

DONALD H. RUSCH, *U.S. Fish and Wildlife Service, Wisconsin Cooperative Wildlife Research Unit, 226 Russell Laboratories, Madison, WI 53706*

DANIEL L. FINLEY, *U.S. Fish and Wildlife Service, National Wildlife Health Research Center, 6006 Schroeder Road, Madison, WI 53711*

MURRAY M. GILLESPIE, *Manitoba Department of Natural Resources, 1495 St. James Street, Winnipeg, Manitoba R3H 0W9, Canada*

Lead poisoning in waterfowl has a long, well-documented history as a wildlife management problem (Sanderson and Bellrose 1986). Since 1976, nontoxic shot zones have been implemented across the United States in an effort to reduce availability of lead pellets, and nationwide mandatory use of nontoxic shot for hunting waterfowl is scheduled to begin with the 1991-1992 waterfowl hunting season (U.S. Fish and Wildl. Serv. 1988). Although compliance with nontoxic shot regulations is high in some areas (Simpson 1989), controversy persists because some hunters and hunting organizations are not convinced that lead poisoning is a problem or that steel shot is the solution (Smith and Townsend 1981, Sanderson and Bellrose 1986). Furthermore, lead shot is still used in Canada (Schwab and Daury 1989). Because dwindling habitat has concentrated waterfowl and hunters onto smaller areas, decades of hunting have built up high densities of lead shot on some areas (Sanderson and Bellrose 1986, U.S. Fish and Wildl. Serv. 1988). Eliminating or reducing the lead shot available to birds is still an important aspect of waterfowl management.

We studied lead exposure in the Eastern Prairie Population of Canada geese (*Branta canadensis interior*), a population of 170,000-230,000 individuals (DeStefano 1989),

throughout their range and over 2 annual cycles. Our objectives were to document the prevalence of lead exposure in the population, identify specific sites of lead exposure at major migration and wintering areas, and monitor changes in rates of lead exposure over time and among areas.

### STUDY AREAS

During summer-winter 1986-1987 and 1987-1988, we conducted fieldwork on 5 areas in the Eastern Prairie Population range: Cape Churchill and vicinity, northern Manitoba; Oak Hammock Wildlife Management Area (WMA), southern Manitoba; Roseau River and Lac Qui Parle WMA's in Minnesota; and Swan Lake National Wildlife Refuge (NWR), northcentral Missouri (Fig. 1). These 5 areas were used by Eastern Prairie Population geese as breeding, migration, and wintering grounds (DeStefano 1989) (Table 1). The latter 4 areas attracted large numbers of waterfowl hunters. Central portions of these areas were refuges where hunting was prohibited; waterfowl hunting took place along perimeters and on surrounding private land. Use of lead shot was still permitted in Manitoba, but nontoxic shot requirements were in effect in Minnesota and Missouri (Table 1). Some spring waterfowl hunting took place on the breeding grounds at remote camps along the shore of Hudson Bay, but hunting pressure was extremely light and deposition of lead shot was minor (M. Gillespie, Manit. Dep. Nat. Resour., Winnipeg, unpubl. data).

### METHODS

#### *Field Methods*

Canada geese were captured and marked with leg bands and individually coded plastic neckbands as part

<sup>1</sup> Present address: P.O. Box 226, Philomath, OR 97370.

<sup>2</sup> Request reprints from this author.

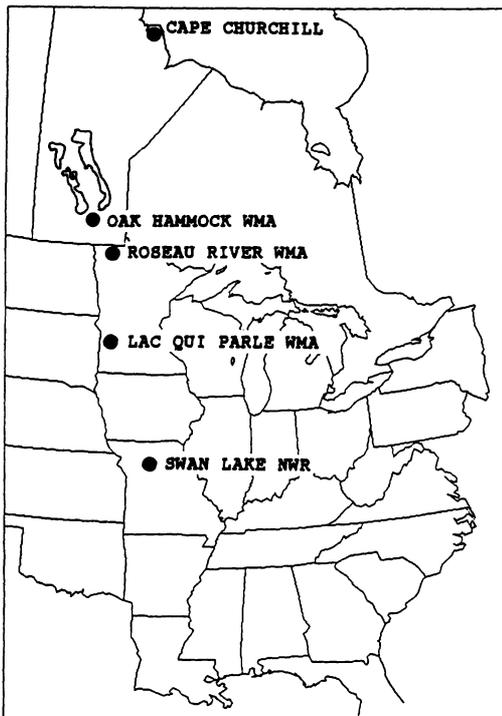


Fig. 1. Locations of breeding grounds (Cape Churchill), major migration areas (Oak Hammock Wildlife Management Area [WMA], Roseau River Wildlife Management Area, and Lac Qui Parle Wildlife Management Area), and wintering grounds (Swan Lake National Wildlife Refuge [NWR]) where Canada geese of the Eastern Prairie Population were captured and sampled for exposure to lead in 1986–1987 and 1987–1988.

of a cooperative research program conducted by the U.S. Fish and Wildlife Service, Canadian Wildlife Service, and states and provinces in the Mississippi Flyway. Helicopter drive-trapping (Timm and Bromley 1976) was used to capture molting adults and goslings on the breeding grounds, and rocket or cannon nets were used on migration and wintering areas.

A 1–2-ml blood sample was drawn from the jugular vein of each goose into a 2-ml vacutainer that contained the anticoagulant sodium heparin (American Scientific Products, McGaw Park, Ill.). Blood samples were shipped chilled or frozen to the National Wildlife Health Research Center in Madison, Wisconsin for lead assay. During both field seasons geese were sampled at Cape Churchill in late July–early August, Oak Hammock WMA in September–November, Roseau River WMA in October, and Lac Qui Parle WMA in October. In 1986–1987 geese were sampled at Swan Lake NWR during 3 periods: late October, December–January, and

February. In 1987–1988 we sampled at Swan Lake NWR during 2 periods: late October and January.

Gizzards and livers from hunter-killed geese were collected at Oak Hammock, Lac Qui Parle, and Swan Lake to determine shot ingestion frequencies and liver lead concentrations. At each area,  $\geq 50$  paired gizzard and liver samples were collected each week of the hunting season from local commercial goose cleaning stations; additional samples were solicited from hunters. All were shipped frozen to the National Wildlife Health Research Center.

At Oak Hammock and Swan Lake, soil samples were collected after the hunting season to estimate quantity of shot available. A 40-cm  $\times$  40-cm sample plot 2.5 cm deep was taken at 50, 75, and 100 m on each of 3 or 5 transects radiating from the front of a randomly selected hunting pit or blind (Humburg and Babcock 1982). At Oak Hammock, 60 sample plots were collected from in front of 4 hunting pits in 1987. At Swan Lake, 138 plots from 10 blinds in 1986 and 120 plots from 8 blinds in 1987 were collected. Soil was washed through a 1.5-mm<sup>2</sup> wire mesh screen (standard window screen), and remaining particles were transferred to a white porcelain tray to be visually examined for shot.

#### Laboratory Methods

Blood samples were analyzed for lead concentration by atomic absorption spectrophotometry with a Perkin-Elmer model 2380 spectrophotometer set at a wavelength of 283.3 nm (Perkin-Elmer Analytical Instruments, Norwalk, Conn.) (Fernandez and Hilligoss 1982). Aliquots of 100 microliters of whole blood were prepared for assay by tenfold dilution in a solution containing 0.5% Triton X-100 (alkylaryl polyether alcohol; J. T. Baker Chem. Co., Phillipsburg, N.J.) and 0.2% ammonium dihydrogen phosphate. Partially clotted samples were sonicated to restore homogeneity before aliquots were taken. Lead standards of 0, 0.02, and 0.05 ppm, as well as a reagent blank and a spiked sample, were tested with each group of 30–35 samples and were used to determine lead concentrations in samples by linear regression. Lead concentrations are reported in ppm of whole blood.

Following analysis of elevated blood lead concentrations, we examined background blood lead concentrations (samples  $< 0.18$  ppm) for trends among areas and over time because elevated blood lead concentrations can return to background levels in birds that survive ingestion of lead shot (Longcore et al. 1974, Roscoe et al. 1979, Franson et al. 1986). We hypothesized that median background levels would increase as the season progressed, and some geese survived ingestion of lead shot. Because numbers of blood samples with background lead levels were large for each field season (see Table 2), years were treated separately to facilitate ranking procedures for Kruskal-Wallis tests (Daniel 1978:200–205).

Whole gizzards were X-rayed, and those showing radio opacities were checked for shot by opening the

Table 1. Summary of Canada goose use and waterfowl hunting regulations at 5 study areas in the Eastern Prairie Population range during 1986–1987 and 1987–1988. Months of use by geese are approximate and include peak use periods.

Area	Use	Time of use	Hunting season	Shot	First year nontoxic shot required
Cape Churchill, Manitoba	Breeding	Apr to early Sep	Some spring hunting at remote camps	Lead	None
Oak Hammock, Manitoba	Migration	Late Aug to late Oct	Late Sep to Nov; no afternoon hunting first half of season	Lead	None
Roseau River, Minnesota	Migration	Mid-Sep to mid-Oct	Early Oct to mid-Nov	Steel	1977
Lac Qui Parle, Minnesota	Migration	Mid-Sep to early Jan	Early Oct to early Nov	Steel	1980
Swan Lake, Missouri	Winter	Early Oct to early Mar	Early Nov to mid-Dec	Steel	1978

gizzard and rinsing the contents into a porcelain tray for visual examination. Shot was classified as lead or steel and ingested or shot-in (DeStefano 1989). When necessary, gizzard muscle was sectioned to retrieve embedded shot. Although X-raying whole gizzards can underestimate shot ingestion by up to 25–30% (Montalbano and Hines 1978, Anderson and Havera 1985), it allowed us to quickly process large numbers of gizzards in order to document shot ingestion, locate embedded shot, and to compare frequencies among areas.

Ground liver samples were oven-dried and ashed in a muffle furnace. Ashed samples were dissolved in a mixture of nitric and hydrochloric acids, digested on a hot plate to 3–5 ml volumes, and then diluted with distilled water to 12 ml and tested for lead by flame analysis on an atomic absorption spectrophotometer set at 217.0 nm wavelength (Boyer 1984, DeStefano 1989). Spiked samples and blank crucibles were also tested for quality control. Results were calculated by linear regression from a standard curve and reported in ppm wet and ppm dry weight. Dry weight results are more accurate than wet weights because variations due to moisture content are eliminated (Adrian and Stevens 1979). However, when proportions of goose livers with elevated lead concentrations were compared on a wet versus dry weight basis, no differences were detected (DeStefano 1989). We report both wet and dry weights to facilitate comparisons with other studies. Concentrations  $\geq 300$  ppm wet ( $\geq 1,000$  ppm dry) weight were eliminated from analysis because of possible contamination with lead shrapnel.

### Terminology and Statistical Analysis

Although a blood lead concentration of 0.20 ppm has been used as an indication of recent lead exposure in waterfowl (Dieter et al. 1976, Finley et al. 1976,

Dieter and Finley 1978, Friend 1985), 0.18 ppm more adequately reflected recent exposure to lead in Eastern Prairie Population Canada geese (see DeStefano 1989). Therefore, blood lead concentrations  $< 0.18$  ppm indicated that geese were unexposed or had background levels of lead, while samples with  $\geq 0.18$  ppm lead indicated recent exposure and were considered elevated. For livers, the terms unexposed and elevated indicated lead concentrations of  $< 2.0$  and  $\geq 2.0$  ppm wet weight ( $< 8.0$  and  $\geq 8.0$  ppm dry weight), respectively (Friend 1985).

Data from samples assayed for lead were not normally distributed because most samples had undetectable ( $< 0.02$  ppm) or very low levels of lead. Data transformations would not normalize distributions, so nonparametric statistical methods were used (Daniel 1978). Significance level was  $P \leq 0.05$ .

## RESULTS

### Blood Lead Concentrations

We collected blood from 7,433 Canada geese in the Eastern Prairie Population range during July–February 1986–1987 and July–January 1987–1988 (Table 2). Differences between years in the proportion of geese with elevated blood lead concentrations were found in only 2 of 6 comparisons (Table 2); therefore, we combined data from both years to simplify analyses.

Geese sampled on the breeding grounds near Cape Churchill had almost exclusively low

Table 2. Number and percent of Canada geese with elevated blood lead concentrations ( $\geq 0.18$  ppm) captured in Eastern Prairie Population range, summer–winter 1986–1987 and 1987–1988.

Area <sup>a</sup>	1986–1987			1987–1988			Combined		
	n	No. elevated	%	n	No. elevated	%	n	No. elevated	%
CC	1,642	5	0.3	944	4	0.4	2,586	9	0.3
OH	352	16	4.5	638	59	9.2 <sup>b</sup>	990	75	7.6
RR	95	8	8.4	85	10	11.8	180	18	10.0
LQP	586	7	1.2	554	12	2.2	1,140	19	1.7
SL1	333	12	3.6	1,011	54	5.3	1,344	66	4.9
SL2	501	35	7.0	287	44	15.3 <sup>b</sup>	788	79	10.0
SL3	405	21	5.2				405	21	5.2

<sup>a</sup> Areas: CC = Cape Churchill, Manitoba; OH = Oak Hammock Wildlife Management Area, Manitoba; RR = Roseau River Wildlife Management Area, Minnesota; LQP = Lac Qui Parle Wildlife Management Area, Minnesota; and SL1–SL3 = Swan Lake National Wildlife Refuge, Missouri in late fall, midwinter, and late winter, respectively.

<sup>b</sup> Difference in proportion of lead-exposed geese found between years ( $\chi^2 = 7.16$ , 1 df,  $P = 0.008$ ; and  $\chi^2 = 14.09$ , 1 df,  $P = < 0.001$  for OH and SL2, respectively).

background concentrations of lead well below 0.18 ppm (Table 2). The 9 elevated samples were from adults captured throughout the breeding grounds, not at 1 specific location.

An increased proportion ( $\chi^2 \geq 163.1$ , 1 df,  $P < 0.001$ ) of geese were exposed to lead at Oak Hammock in comparison to Cape Churchill (Table 2). However, none of the geese sampled during the first 2 weeks of capture at Oak Hammock showed exposure to lead ( $n = 136$ ). During the second and third 2-week sampling periods, 8.9% ( $n = 436$ ) and 8.6% ( $n = 418$ ), respectively, of all geese captured were exposed to lead.

At Roseau River, the percentage of all geese sampled that showed recent exposure to lead was similar to that found at Oak Hammock, but it was higher ( $\chi^2 = 39.6$ , 1 df,  $P < 0.001$ ) than the proportion of lead-exposed geese found at Lac Qui Parle (Table 2). Evaluation of the timing of lead exposure at either Roseau River or Lac Qui Parle was not possible because blood sampling took  $< 2$  weeks.

Blood samples were first collected at Swan Lake in October, before hunting began (SL1 in Table 2). At this time, the proportion of geese exposed to lead was about 5%. Lead exposure doubled during the midwinter period (SL2) after hunting ended ( $\chi^2 = 20.5$ , 1 df,  $P < 0.001$ ). Lead exposure returned to about 5%

when geese were captured and sampled for a third time (SL3) in late winter (Feb) 1987 ( $\chi^2 = 8.16$ , 1 df,  $P = 0.005$ , Table 2).

Differences in medians of background blood lead concentrations among areas were detected in 1986–1987 and 1987–1988 (Kruskal-Wallis test [Daniel 1978:200–205], both field seasons  $H \geq 537$ , 6 df,  $P < 0.01$ ). Multiple comparisons (Daniel 1978:211–214) revealed differences between all pairs except Oak Hammock and Roseau River in both 1986 and 1987, and Roseau River and Lac Qui Parle in both 1986 and 1987. Median background blood lead concentrations tended to increase in later sampling periods (Fig. 2).

#### Shot in Gizzards

Percentages of gizzards from hunter-killed geese with  $\geq 1$  ingested lead or steel pellet were similar between years at Oak Hammock, Lac Qui Parle, and Swan Lake so years were combined (Table 3). No steel pellets were found in gizzards from Oak Hammock, while very few lead pellets were found in gizzards from Lac Qui Parle. Swan Lake had the highest combined lead and steel ingestion frequency of the 3 areas—6.2%. The ratio of lead to steel shot at Swan Lake was 0.7:1.

We retrieved pellets that had been shot into

gizzard muscle or had penetrated into the gizzard chamber (Table 3). Gizzards with shot-in lead pellets predominated at Oak Hammock. At Lac Qui Parle and Swan Lake, 33% and 25%, respectively, of all shot-in pellets were lead.

#### Liver Lead Concentrations

At Oak Hammock and Lac Qui Parle, there were no differences between years in the proportion of livers with elevated concentrations of lead; only Swan Lake showed a difference between years (Table 4). The proportion of livers with elevated lead concentrations was higher at Oak Hammock than at Lac Qui Parle in both years (combined data,  $\chi^2 = 9.41$ , 1 df,  $P = 0.003$ ). We have no explanation for the high percentage of livers with elevated lead concentrations at Swan Lake in 1986.

#### Shot in Soil

At Oak Hammock, 8 lead and 0 steel shot were found in soil sample plots, or 8,333 (SE = 4,501) lead pellets per ha. At Swan Lake in 1986, 29 lead and 17 steel shot were found, or 12,589 (SE = 3,724) lead and 8,095 (SE = 2,218) steel per ha. In 1987, 28 lead and 17 steel shot were found, or 14,583 (SE = 3,608) lead and 7,292 (SE = 1,716) steel per ha. Lead to steel ratio averaged 1:0.6 for both years at Swan Lake.

### DISCUSSION

We may have underestimated the prevalence of lead exposure in Eastern Prairie Population Canada geese because elevated lead concentrations in blood can decline to background levels (i.e., <0.18 ppm) over time in birds that survive lead shot ingestion (Longcore et al. 1974, Roscoe et al. 1979, Franson et al. 1986). In addition, some geese died of lead poisoning after ingesting lead shot, and were unavailable for blood sampling (Brand et al. 1992). However, we used the blood assay tech-

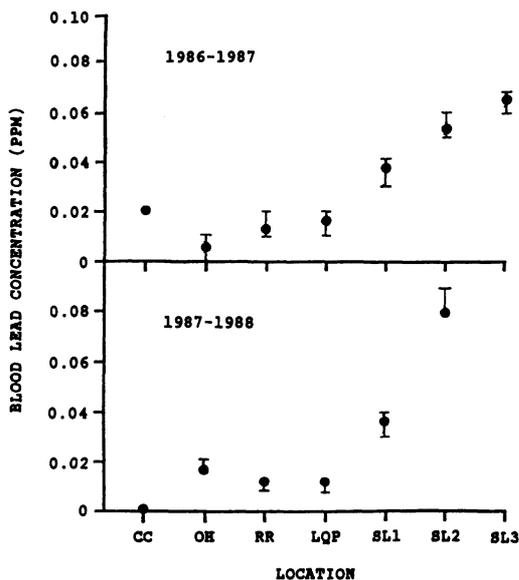


Fig. 2. Trends in median background blood lead concentrations (<0.18 ppm lead) and 95% confidence intervals for the median for blood samples collected from Canada geese on Eastern Prairie Population breeding, migration, and wintering areas, 1986-1987 and 1987-1988. (Location: CC = Cape Churchill, Manitoba; OH = Oak Hammock Wildlife Management Area, Manitoba; RR = Roseau River Wildlife Management Area, Minnesota; LQP = Lac Qui Parle Wildlife Management Area, Minnesota; and SL1-SL3 = Swan Lake National Wildlife Refuge, Missouri during fall, mid-winter, and late winter, respectively.)

nique as our main criteria for evaluating lead exposure in the population because it is the most sensitive measure of lead intoxication available and allows sampling of large numbers of live birds throughout the year (Anderson and Havera 1985, Roscoe 1986, Friend 1987). Ingested shot in gizzards and lead concentrations in livers are less useful indices because large samples are available only during hunting seasons, whereas most shot ingestion occurs after hunting has ended (Sanderson and Bellrose 1986, Friend 1987). Additional biases associated with estimating lead exposure based on shot ingestion have been discussed by many authors (Jordan and Bellrose 1951, Bellrose 1959, Dieter and Finley 1978, Montalbano and

Table 3. Number and percentage of gizzards with  $\geq 1$  ingested or shot-in lead or steel shotgun pellets collected from hunter-killed Canada geese at 3 locations in Eastern Prairie Population range during fall 1986 and 1987.

Area*	n	Ingested				Shot-in			
		No. with lead shot	%	No. with steel shot	%	No. with lead shot	%	No. with steel shot	%
OH	1,029	26	2.5	0		91	8.8	3	0.3
LQP	516	3	0.6	13	2.5	15	2.9	31	6.0
SL	590	15	2.5	22	3.7	13	2.2	40	6.8

\* Areas: OH = Oak Hammock Wildlife Management Area, Manitoba; LQP = Lac Qui Parle Wildlife Management Area, Minnesota; SL = Swan Lake National Wildlife Refuge, Missouri.

Hines 1978, Anderson and Havera 1985, Sanderson and Bellrose 1986). Despite potential biases, we believe that blood assays, supported by data on shot ingestion and liver lead concentrations, allowed us to make comparisons among areas and to develop a synopsis of lead exposure in the Eastern Prairie Population.

The Eastern Prairie Population breeding grounds are remote, inaccessible to most hunters, and essentially free of spent lead shot. Accordingly,  $<0.5\%$  of geese from Cape Churchill showed recent exposure to lead. Furthermore, because all geese near Cape Churchill were away from a source of lead shot for 2–4 months, we believe that elevated blood lead concentrations in geese that survived ingestion of lead shot on migration and wintering grounds had dropped to background levels on the breeding grounds. Therefore, the fall flight of geese in the Eastern Prairie Population began with a population of “clean” birds (i.e., virtually all geese had  $<0.18$  ppm blood lead).

After leaving northern Manitoba, the geese encountered a source of lead shot around Oak Hammock WMA in southern Manitoba (8,333 [SE = 4,501] shot/ha in soil near hunting pits). A delay of about 2 weeks in detecting lead exposure in blood samples indicated that Oak Hammock was probably the first major source of lead shot for Eastern Prairie Population geese. Geese were often seen feeding in hunted fields during periods of low hunting pressure and when hunting closed at noon (DeStefano 1989). Annual tilling may have decreased shot availability (Fredrickson et al. 1977, Esslinger and Klimstra 1983), but new lead pellets were added every year. Although density of shot in soil at Oak Hammock was not as high as that reported in the literature for other areas (Bellrose 1959, Humburg and Babcock 1982, Fisher et al. 1986a, Windingstad and Hinds 1987, Anderson and Havera 1989), Szymczak and Adrian (1978) found that 7,512 lead pellets/ha was high enough to cause a major lead poisoning die-off of Canada geese in Colorado.

Table 4. Number and percent of livers with elevated lead concentrations ( $\geq 2.0$  ppm lead wet weight [ $\geq 8.0$  ppm dry weight]) collected from hunter-killed Canada geese at 3 locations in Eastern Prairie Population range during fall 1986 and 1987.

Area*	1986–1987			1987–1988			Combined		
	n	No. elevated	%	n	No. elevated	%	n	No. elevated	%
OH	741	35	4.7	255	6	2.4	996	41	4.1
LQP	276	3	1.1	227	3	1.3	503	6	1.2
SL	193	56	29.0	221	11	5.0 <sup>b</sup>	414	67	16.2

\* Areas: OH = Oak Hammock Wildlife Management Area, Manitoba; LQP = Lac Qui Parle Wildlife Management Area, Minnesota; SL = Swan Lake National Wildlife Refuge, Missouri.

<sup>b</sup> Difference in proportion of livers with elevated lead concentrations found between years ( $\chi^2 = 43.9$ , 1 df,  $P < 0.001$ ).

Our contention that Oak Hammock was a major source for lead exposure is supported by the fact that >50% of moribund and dead geese collected at Oak Hammock were victims of lead poisoning and had ingested an average of 19 pellets (Brand et al. 1992).

A much higher percentage of geese showed exposure to lead at Roseau River than at Lac Qui Parle, even though both areas were nontoxic shot zones. We attribute this difference to the close proximity of Roseau River to Oak Hammock and other lead shot areas in Manitoba. Geese that arrived at Lac Qui Parle early in the fall, thereby minimizing time spent at Oak Hammock or Roseau River, decreased their potential for lead exposure. Low frequencies of lead concentrations in tissues, lead pellet ingestion, and carcasses diagnosed as having lead poisoning (Brand et al. 1992) at Lac Qui Parle supported this assumption.

Humburg and Babcock (1982) reported that the Swan Lake Steel Shot Zone decreased the magnitude of lead poisoning in Missouri. We concur, but a persistent, localized source of lead has reduced its effectiveness as a nontoxic shot zone. Lead shot in soil samples was present at a rate of  $1.8\times$  that of steel, even though Swan Lake has been a steel-shot zone since 1978. We attribute the predominance of lead shot to >20 years of hunting from permanent blinds before Swan Lake became a steel shot zone (Vaught and Kirsch 1966); to the higher decay rate of steel, which rusts, compared to lead, which is relatively inert (Fisher et al. 1986b); and to the possibility that lead shot is illegally used.

Bengtson (1984) found that 7–9% of seasonal deposition of shot at Lac Qui Parle was illegally used lead shot, and Simpson (1989) reported that noncompliance with nontoxic shot regulations in South Dakota was about 20%. Another index to violation of steel shot regulations is the number of shot-in lead pellets found in gizzards from geese killed in steel shot zones (Simpson 1989). At Lac Qui Parle and Swan Lake, lead shot composed 33% and 25%, re-

spectively, of the shot-in pellets. It is possible that these estimates are biased high because some geese, previously wounded by hunters at other locations, probably could survive with shotgun pellets embedded in their gizzard muscle. Nevertheless, these figures reflect violation of steel shot regulations from these 2 areas. At Oak Hammock, where nontoxic shot was not required, lead shot accounted for 97% of all shot-in pellets.

Geese probably encountered sources of lead shot at several locations before reaching Swan Lake; thus the potential for lead exposure increased as geese moved southward. An increase in background blood lead concentrations as the season progressed revealed that some geese reached Swan Lake already exposed to lead. Prevalence of elevated lead concentrations in blood samples collected when birds first arrived at Swan Lake also indicated that some lead exposure occurred before birds reached Missouri.

However, spent lead shot was available near hunting blinds along the boundary of Swan Lake NWR, and crop plantings around these blinds brought geese to this source of lead shot after the close of the hunting season. Movements and distribution of geese around Swan Lake showed that crops near blinds were used heavily for feeding during midwinter (DeStefano 1989). Although nontoxic shot has been used at Swan Lake since 1978, several more years must pass before lead shot in soil is reduced to low densities despite ongoing cultivation of the soil around blinds.

#### MANAGEMENT IMPLICATIONS

The most effective step in rectifying the lead poisoning problem in the United States has been taken: nationwide, mandatory use of nontoxic shot for hunting migratory waterfowl beginning in 1991 (U.S. Fish and Wildl. Serv. 1988). Based on our research, the Manitoba Department of Natural Resources has decided to establish an area around Oak Hammock

WMA as the first nontoxic shot zone in Manitoba, beginning with the 1991 hunting season. Additional nontoxic shot zones will be established in other provinces, but to date there are no plans for a nationwide nontoxic shot regulation in Canada (M. Gillespie, unpubl. data). However, lead exposure and lead poisoning can still occur >10 years after steel shot zones are established, as found at Swan Lake. Localized lead poisoning problems could be alleviated by continuing annual soil tillage near permanent hunting blinds to reduce lead shot availability and by discontinuing crop plantings immediately around blinds to discourage goose use of these areas.

### SUMMARY

We monitored lead exposure in Eastern Prairie Population Canada geese during summer–winter, 1986–1987 and 1987–1988 at 5 areas. Blood lead concentrations in geese trapped during summer at Cape Churchill, Manitoba were below levels indicative of recent lead exposure (0.18 ppm). Geese exposed to lead ( $\geq 0.18$  ppm blood lead) increased to 7.6% at Oak Hammock Wildlife Management Area (WMA), southern Manitoba, where lead shot was still in use, and to 10.0% at Roseau River WMA, northern Minnesota, when fall-staging geese were close to a source of lead shot in Manitoba. Proportion of birds exposed to lead dropped to <2% at Lac Qui Parle WMA, Minnesota, a steel shot zone since 1980. On the wintering grounds at Swan Lake National Wildlife Refuge in Missouri, 4.9% of all geese showed exposure to lead before the hunting season. Lead exposure rose to 10.0% after hunting ended and then decreased to 5.2% in late winter. Incidence of lead shot in gizzards and concentrations of lead in livers supported blood assay data. Soil samples indicated that lead shot continues to be available to geese at Swan Lake, even though this area was established as a nontoxic shot zone in 1978. Steel shot zones have

reduced lead exposure in the Eastern Prairie Population, but lead shot persists in the environment and continues to account for lead exposure and mortality in Eastern Prairie Population Canada geese.

*Acknowledgments.*—Funding was provided by the U.S. Fish and Wildlife Service and administered through the Wisconsin Cooperative Wildlife Research Unit. Research was conducted through the National Wildlife Health Research Center, Madison, Wisconsin in cooperation with the Manitoba Department of Natural Resources, Minnesota Department of Natural Resources, and Missouri Department of Conservation. We thank M. P. Conrad, B. K. Whaley, and G. W. Ball in Manitoba; M. C. Zicus, J. DiMatteo, K. Bonnema, and J. Anderson in Minnesota; L. M. Mechlin, J. E. Austin, M. Jackson, B. Burton, L. Burton, K. A. McMullen, J. Frye, and P. Thomsen in Missouri; and numerous others for invaluable field support. T. Eberhardt of the Minnesota Department of Natural Resources collected all blood samples at Roseau River WMA. We also appreciate assistance from S. J. Madsen, M. Smith, T. Forster, B. Roubicek, J. Coluccy, B. Allen, J. C. Franson, R. M. Windingstad, owners of goose cleaning stations, and goose hunters throughout the flyway. M. D. Samuel, J. R. Cary, G. W. Swenson, and E. A. Kihm provided statistical and computer analyses consultation. M. G. Hornocker, E. D. Ables, K. P. Reese, and D. R. Johnson of the University of Idaho–Moscow offered advice throughout the study. We thank S. J. Madsen, J. C. Franson, K. Cook, S. B. White, and an anonymous referee for reviews of the manuscript.

### LITERATURE CITED

- ADRIAN, W. J., AND M. L. STEVENS. 1979. Wet versus dry weights for heavy metal toxicity determinations in duck liver. *J. Wildl. Dis.* 15:125–126.
- ANDERSON, W. L., AND S. P. HAVERA. 1985. Blood lead, protoporphyrin, and ingested shot for detecting lead poisoning in waterfowl. *Wildl. Soc. Bull.* 13:26–31.
- , AND ———. 1989. Lead poisoning in Illinois

- waterfowl (1977–1988) and the implementation of nontoxic shot regulations. *Ill. Nat. Hist. Surv. Biol. Notes* 133. 37pp.
- BELLROSE, F. C. 1959. Lead poisoning as a mortality factor in waterfowl populations. *Ill. Nat. Hist. Surv. Bull.* 27:232–288.
- BENGTSON, F. L. 1984. Studies of lead toxicity in bald eagles at the Lac Qui Parle Wildlife Refuge. M.S. Thesis, Univ. Minnesota, St. Paul. 95pp.
- BOYER, K. W. 1984. Metals and other elements at trace levels in foods. Pages 444–476 in S. Williams, ed. *Official methods of analysis of the Association of Official Analytical Chemists*. Fourteenth ed. Assoc. Off. Anal. Chem., Inc., Arlington, Va.
- BRAND, C. J., S. DEStEFANO, AND J. C. FRANSON. 1992. Lead poisoning as a component of nonhunting mortality in Eastern Prairie Population Canada geese. In D. H. Rusch, B. D. Sullivan, and M. D. Samuel, eds. *Proc. International Canada Goose Symposium*, Milwaukee, Wis. In Press.
- DANIEL, W. W. 1978. Applied nonparametric statistics. Houghton Mifflin Co., Boston, Mass. 510pp.
- DEStEFANO, S. 1989. Ecological relationships of lead exposure in Canada geese of the Eastern Prairie Population. Ph.D. Thesis, Univ. Idaho, Moscow. 97pp.
- DIETER, M. P., AND M. T. FINLEY. 1978. Erythrocyte delta-aminolevulinic acid dehydratase activity in mallard ducks: duration of inhibition after lead shot dosage. *J. Wildl. Manage.* 42:621–625.
- , M. C. PERRY, AND B. M. MULHERN. 1976. Lead and PCB's in canvasback ducks: relationship between enzyme levels and residues in blood. *Arch. Environ. Contam. and Toxicol.* 5:1–13.
- ESSLINGER, C. G., AND W. D. KLIMSTRA. 1983. Lead shot incidence on a public goose hunting area in southern Illinois. *Wildl. Soc. Bull.* 11:166–169.
- FERNANDEZ, F. J., AND D. HILLIGOSS. 1982. An improved graphite furnace method for the determination of lead in blood using matrix modification and the I'vov platform. *Atomic Spectroscopy* 3:130–131.
- FINLEY, M. T., M. P. DIETER, AND L. N. LOCKE. 1976. Sublethal effects of chronic lead ingestion in mallard ducks. *J. Toxicol. and Environ. Health* 1:929–937.
- FISHER, F. M., JR., S. L. HALL, W. R. WILDER, B. C. ROBINSON, AND D. S. LOBPRIES. 1986a. An analysis of spent shot in upper Texas coastal waterfowl wintering habitat. Pages 50–54 in J. S. Feierabend and A. B. Russell, eds. *Lead poisoning in wild waterfowl, a workshop*. Natl. Wildl. Fed., Washington, D.C.
- , ———, C. D. STUTZENBAKER, AND D. S. LOBPRIES. 1986b. An evaluation of spent shot ingestion in Texas wintering waterfowl by X-ray radiography. Pages 18–26 in J. S. Feierabend and A. B. Russell, eds. *Lead poisoning in wild waterfowl, a workshop*. Natl. Wildl. Fed., Washington, D.C.
- FRANSON, J. C., G. M. HARAMIS, M. C. PERRY, AND J. F. MOORE. 1986. Blood protoporphyrin for detecting lead exposure in canvasbacks. Pages 32–37 in J. S. Feierabend and A. B. Russell, eds. *Lead poisoning in wild waterfowl, a workshop*. Natl. Wildl. Fed., Washington, D.C.
- FREDRICKSON, L. H., T. S. BASKETT, G. K. BRAKHAGE, AND V. C. CRAVENS. 1977. Evaluating cultivation near duck blinds to reduce lead poisoning hazard. *J. Wildl. Manage.* 41:624–631.
- FRIEND, M. 1985. Interpretation of criteria commonly used to determine lead poisoning problem areas. *U.S. Fish and Wildl. Serv. Leaflet*. 2. 4pp.
- . 1987. Lead poisoning. Pages 175–189 in M. Friend, ed. *Field guide to wildlife diseases*. Vol. 1. U.S. Fish and Wildl. Serv. Resour. Publ. 167.
- HUMBURG, D. D., AND K. M. BABCOCK. 1982. Lead poisoning and lead/steel shot: Missouri studies and a historical perspective. *Mo. Dep. Conserv. Tech. Rep. Terrestrial Ser.* 10. 23pp.
- JORDAN, J. S., AND F. C. BELLROSE. 1951. Lead poisoning in wild waterfowl. *Ill. Nat. Hist. Surv. Biol. Notes* 26. 27pp.
- LONGCORE, J. R., L. N. LOCKE, G. E. BAGLEY, AND R. ANDREWS. 1974. Significance of lead residues in mallard tissues. *U.S. Fish and Wildl. Serv. Spec. Sci. Rep.-Wildl.* 182. 24pp.
- MONTALBANO, F., AND T. C. HINES. 1978. An improved X-ray technique for investigating ingestion of lead by waterfowl. *Proc. Annu. Conf. Southeast. Assoc. Fish and Wildl. Agencies* 32:364–368.
- ROSCOE, D. E. 1986. Diagnostic and survey techniques for lead poisoning in waterfowl. Pages 27–32 in J. S. Feierabend and A. B. Russell, eds. *Lead poisoning in wild waterfowl, a workshop*. Natl. Wildl. Fed., Washington, D.C.
- , S. W. NIELSEN, A. A. LAMOLA, AND D. ZUCKERMAN. 1979. A simple, quantitative test for erythrocytic protoporphyrin in lead-poisoned ducks. *J. Wildl. Dis.* 15:127–136.
- SANDERSON, G. C., AND F. C. BELLROSE. 1986. A review of the problem of lead poisoning in waterfowl. *Ill. Nat. Hist. Surv. Spec. Publ.* 4. 34pp.
- SCHWAB, F. E., AND R. W. DAURY. 1989. Incidence of ingested lead shot in Nova Scotia waterfowl. *Wildl. Soc. Bull.* 17:237–240.
- SIMPSON, S. G. 1989. Compliance by waterfowl hunters with nontoxic shot regulations in central South Dakota. *Wildl. Soc. Bull.* 17:245–248.
- SMITH, R. L., AND T. W. TOWNSEND. 1981. Attitudes of Ohio hunters toward steel shot. *Wildl. Soc. Bull.* 9:4–7.
- SZYMCZAK, M. R., AND W. J. ADRIAN. 1978. Lead poisoning in Canada geese in southeast Colorado. *J. Wildl. Manage.* 42:299–306.
- TIMM, D. E., AND R. G. BROMLEY. 1976. Driving Canada geese by helicopter. *Wildl. Soc. Bull.* 4:180–181.
- U.S. FISH AND WILDLIFE SERVICE. 1988. Final supplemental environmental impact statement: issuance of annual regulations permitting the sport

- hunting of migratory birds. U.S. Dep. Inter., Washington, D.C. 340pp.
- VAUGHT, R. W., AND L. M. KIRSCH. 1966. Canada geese of the Eastern Prairie Population, with special reference to the Swan Lake flock. Mo. Dep. Conserv. Tech. Bull. 3. 91pp.
- WINDINGSTAD, R. M., AND L. S. HINDS III. 1987. Lead poisoning in Canada geese on Plum Island, Massachusetts. *J. Wildl. Dis.* 23:438-442.

Received 23 April 1990.

Accepted 20 September 1990.



*Wildl. Soc. Bull.* 19:32-35, 1991

## AN EVALUATION OF TWO HAZING METHODS FOR URBAN CANADA GEESE

ELIZABETH AGUILERA,<sup>1</sup> *Department of Fishery and Wildlife Biology, Colorado State University, Fort Collins, CO 80523*

RICHARD L. KNIGHT,<sup>2</sup> *Department of Fishery and Wildlife Biology, Colorado State University, Fort Collins, CO 80523*

JOHN L. CUMMINGS, *APHIS-USDA, Denver Wildlife Research Center, Denver Federal Center, Denver, CO 80225*

The Hi-Line population of Canada geese (*Branta canadensis*) that winters east of the Continental Divide has increased substantially in recent years (Szymczak 1975.) This increase has been accompanied by the development of resident urban populations of geese that are augmented during the winter months by migratory birds. Conflicts between geese and people have been reported and, at present, effective goose control methods have not been developed (e.g., Conover 1989).

The most common techniques to reduce use of areas by geese include loud noises (e.g., fire-crackers and exploders), chasing, harassing with dogs, swan decoys, wires or lines to discourage geese from ponds, and shooting (Conover and Chasko 1985). Of these methods, at least 2 appear to be applicable to urban situations. First, loud noises, such as those created by screamer shells, cause geese to fly. Second, tapes

of geese alarm or distress calls may also cause geese to abandon an area (Mott and Timbrook 1988). Although these methods may be appropriate in urban areas, their usefulness has not been tested. Hence, in this study, we evaluated the effectiveness of goose calls and screamer shells to disperse wintering Canada geese in Fort Collins, Colorado.

### METHODS

We conducted the study at 10 parks and recreation areas within Fort Collins, Colorado between November 1988 and February 1989. Sites were separated by at least 4 km, ranged from 0.6 to 1.0 ha in size, and 6 of 10 were associated with ponds. City government had received complaints about Canada geese from land-owners for each site, although no form of goose harassment had been conducted at any of the sites prior to our study.

We randomly assigned a treatment of either goose calls or "screamer" shells to each site, creating 5 replications of each treatment. One replication of each treatment was conducted during December, and 4 replications of each treatment were conducted during January and February.

Two goose-call tapes were used. The first contained the alarm call of 1 goose and a chorus of 25 disturbed geese as they took flight (Mott and Timbrook 1988). The second tape contained the distress call of a single

<sup>1</sup> Present address: ICA, Palmira, A.A. 233, Palmire, Colombia, South America.

<sup>2</sup> Request reprints from Richard L. Knight.