

LEAD POISONING AS A COMPONENT OF MORBIDITY AND MORTALITY IN CARCASSES OF EASTERN PRAIRIE POPULATION CANADA GEESE

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Abstract: The relative importance of lead poisoning as a cause of morbidity and mortality in Eastern Prairie Population Canada geese (*Branta canadensis*) was studied as part of a comprehensive study of lead exposure in this population. Necropsies were performed on 158 Canada geese found sick or dead during fall or winter at Oak Hammock Wildlife Management Area (WMA) in Manitoba, Lac qui Parle WMA in Minnesota, and Swan Lake National Wildlife Refuge and Fountain Grove WMA in Missouri from 1986 to 1988. Lead poisoning was the predominant diagnosis in geese examined from Oak Hammock (55%), where nontoxic (steel) shot is not required for waterfowl hunting. Lead poisoning was also the predominant diagnosis at Swan Lake/Fountain Grove (67%), where steel shot has been required since 1978, but only accounted for 11% of the mortality at Lac qui Parle, where steel shot has been required since 1980. Lead poisoning continues to occur in Canada geese on staging and wintering areas where use of steel shot is required, suggesting that, in some areas, lead poisoning will not be substantially reduced for many years. Further research is warranted to identify areas with persistent lead poisoning following the nationwide conversion to nontoxic shot, and to develop means for reducing lead poisoning at these sites.

Key words: *Branta canadensis*, Canada goose, lead poisoning.

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The Eastern Prairie Population (EPP) of Canada geese breeds in northern Manitoba, stages primarily at Oak Hammock Wildlife Management Area (WMA) in southern Manitoba and Lac qui Parle WMA in southwestern Minnesota, and winters near Swan Lake National Wildlife Refuge (NWR) and Fountain Grove WMA in northern Missouri. Studies of EPP Canada geese during the 1970's and early 1980's at Lac qui Parle (Bengtson 1984) and Swan Lake NWR (Humburg and Babcock 1982, Humburg et al. 1983) demonstrated the occurrence of lead poisoning as a mortality factor in this population, and influenced decisions requiring the use of nontoxic shot for waterfowl hunting on federal- and state-owned land in Minnesota and Missouri.

In 1986 we began an investigation of the magnitude and effects of lead pellet ingestion and lead poisoning in EPP Canada geese (DeStefano 1989, DeStefano et al. 1991). As part of this study, sick and dead Canada geese collected during 1986-88 at EPP staging and wintering areas in Manitoba, Minnesota, and Missouri were necropsied to determine causes of

morbidity and mortality. We present results of diagnostic examinations of these carcasses, and compare proportional mortality from lead poisoning from 1986 to 1988 among areas and to studies conducted before or during the early years of nontoxic shot requirements.

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STUDY AREAS

Oak Hammock WMA (Fig. 1) is a 3,500-ha wetland managed for waterfowl by the Manitoba Department of Natural Resources (DNR). Waterfowl hunting

occurs on privately owned agricultural land (primarily barley, wheat, and hay crops) surrounding Oak Hammock. Canada geese begin arriving in late August and generally remain until snow covers available waste grain, usually in early November. Peak Canada goose numbers occur in late September. Nontoxic shot has not been required for waterfowl hunting and voluntary use of nontoxic shot was low, based on examination of shot embedded in hunter-killed geese (DeStefano 1989).

Lac qui Parle WMA comprises 10,930-ha of lake and surrounding uplands managed for waterfowl by the Minnesota DNR (Fig. 1). Hunting occurs in the surrounding agricultural fields that are primarily in corn production. Canada geese use this area from September until December or January. Nontoxic shot has been required for waterfowl hunting in the

Lac qui Parle Steel Shot Zone since fall 1980. Additional legislation required the use of nontoxic shot for waterfowl hunting state-wide beginning in 1987.

Wintering grounds near Swan Lake NWR, Missouri, include waterfowl areas managed by both the U.S. Fish and Wildlife Service and the Missouri Department of Conservation (Fig. 1). Swan Lake NWR (4,318 ha) and Fountain Grove (2,557 ha), 8 km northwest of Swan Lake, are the major wintering sites for EPP geese (Vaught and Kirsch 1966). Crops grown on surrounding agricultural lands are primarily soybeans and corn. Hunting occurs at Fountain Grove and on the periphery of Swan Lake NWR. Geese are usually present between October and early March. Nontoxic shot first was required on hunted areas surrounding the refuge and at Fountain Grove in 1978, but was temporarily rescinded at Fountain Grove during 1980.

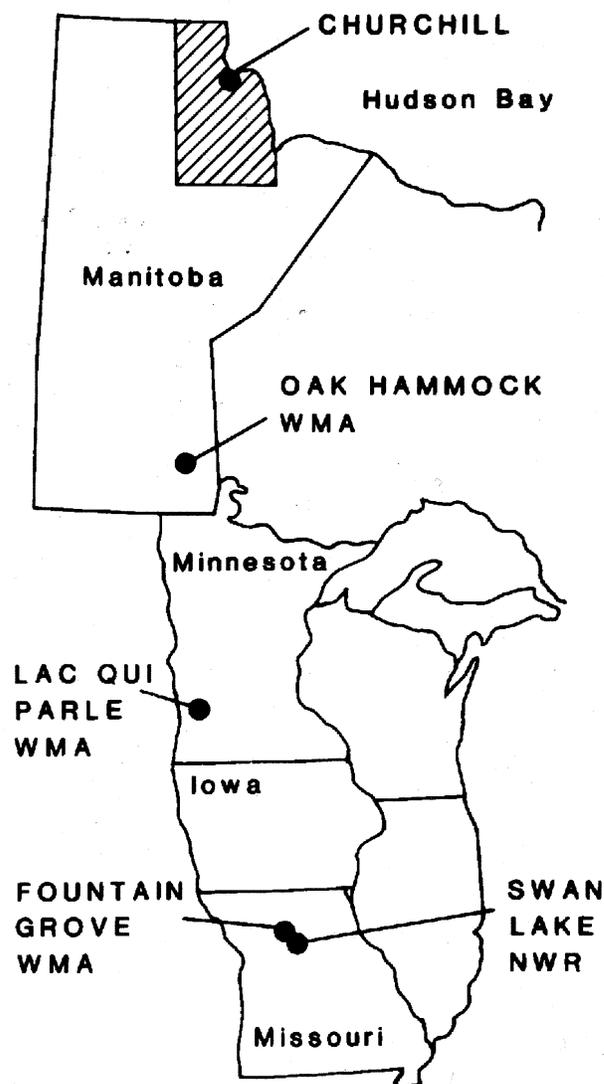


Figure 1. Carcass collection sites on fall staging and wintering areas of Eastern Prairie Population Canada geese during 1986-88. Stippled area indicates breeding range.

METHODS

Sick and dead Canada geese were collected during fall 1986 and 1987 at Oak Hammock WMA and Lac qui Parle WMA, and fall and winter 1987-88 at Swan Lake NWR and Fountain Grove. Collections were made on foot or by air boat in areas where geese were known to feed or roost, including open water, shorelines, areas of emergent vegetation, agricultural fields used for feeding, and upland areas adjacent to roosting or feeding sites. In 1987, Oak Hammock and Lac qui Parle were searched after the hunting season in hunted and nonhunted areas. In 1986, the 13 carcasses we examined from Oak Hammock were found incidental to other field activities. In 1987, over 200 carcasses were collected at Oak Hammock. Because of time and resource constraints, 56 intact carcasses in good postmortem condition were selected from these 200 carcasses for necropsy. Carcasses were selected to represent geese with obvious gunshot wounds, emaciated geese, and geese with normal pectoral development without gunshot injuries. At Swan Lake/Fountain Grove, sites utilized by geese and adjacent nonhunted areas were searched regularly (several times/week) during and after hunting season.

Sick geese (including geese with apparent gunshot injuries) were either collected by shotgun using nontoxic loads, or captured by hand and euthanized by cervical dislocation. Field necropsies were conducted by staff of the National Wildlife Health Research Center (NWHRC), or carcasses were placed in plastic bags and shipped chilled or frozen to the NWHRC for necropsy to determine cause of morbidity or death.

Table 1. Diagnoses for 158 Canada goose carcasses examined at major Eastern Prairie Population staging and wintering areas during 1986 and 1987.

Diagnosis	No. of carcasses (%)				
	Oak Hammock		Lac qui Parle		Swan Lake Fountain Grove
	1986	1987	1986	1987	1987-88
	(1-14 Nov)	(5 Sep-4 Nov)	(11-15 Nov)	(14 Oct-11 Nov)	(25 Oct-23 Feb)
Lead poisoning	9 (69)	29 (52)	3 (9)	4 (15)	18 (67)
Lead poisoning and gunshot	0 (0)	1 (2)	0 (0)	0 (0)	0 (0)
Gunshot ^a	2 (15)	23 (41)	12 (34)	15 (56)	7 (26)
Impaction ^b	0 (0)	0 (0)	17 (49)	6 (22)	0 (0)
Other ^c	2 (15)	1 (2)	0 (0)	0 (0)	2 (7)
Unknown	0 (0)	3 (5)	3 (9)	2 (7)	0 (0)
Total	13	57	35	27	27

^a Includes deaths directly from gunshot, debilitating gunshot wounds, and gunshot wounds resulting in peritonitis.

^b Impactions of undetermined etiology not associated with lead poisoning.

^c Includes predation ($n = 1$), visceral gout ($n = 1$), ulcerative gastritis of unknown etiology ($n = 1$), emaciation of unknown etiology ($n = 1$), and nonpredation trauma ($n = 1$).

Cause of morbidity or death was determined on the basis of lesions observed at necropsy and by appropriate laboratory analyses for each examined carcass. Gross lesions were recorded at necropsy. The gizzard of each bird was opened and manually examined to recover shot. Recovered shotgun pellets were identified as lead or steel (nontoxic) shot, and whether they had been ingested or "shot in."

The liver from each bird examined was assayed for lead by atomic absorption spectrophotometry (Model 2380, Perkin-Elmer Analytical Instruments, Norwalk, Conn.), as described by Boyer (1984). The lower limit of detection for lead was 0.22 ppm. Lead poisoning was diagnosed as a cause of morbidity or mortality if the wet-weight concentration of lead in the liver was ≥ 8.0 ppm, wet weight basis, and lesions consistent with lead toxicosis were present (Cook and Trainer 1966, Wobeser 1981, Friend 1987). Lead poisoning was suspected as contributing to morbidity or mortality when lead concentration in the liver was elevated (≥ 2.0 and < 8.0 ppm, wet weight basis) and lesions consistent with lead poisoning were present. Data from confirmed and suspected cases of lead poisoning are combined here.

Laboratory assays to determine other causes of morbidity or mortality involved standard virologic (Hitchner et al. 1980) and bacteriologic (Lennette et al. 1985) procedures on tissues with lesions suspected of being infectious in origin. In addition, livers from selected geese without gross evidence of infectious disease were cultured on blood agar plates (Lennette et al. 1985) and duck embryo fibroblast cells (Hitchner et al. 1980) to rule out the presence of several common avian bacterial or viral infections.

Proportional mortality due to lead poisoning (number of cases of lead poisoning/number examined) among necropsied carcasses were compared between areas and time of year using chi-square (χ^2) contingency tables. Student's t test was used to determine differences in numbers of ingested lead shot in gizzards. A significance level of $P \leq 0.05$ was used for both tests.

RESULTS

We examined 158 Canada goose carcasses (Table 1). The cause of morbidity or death was determined for 150 (95%) of the 158 carcasses necropsied (Table 1). Diagnoses could not be made on the basis of necropsy and laboratory studies conducted on the other 8 carcasses, but these birds had no evidence of gunshot wounds or lead poisoning.

Lead poisoning, the most frequently diagnosed condition, occurred in 63 (40%) of 158 carcasses examined (Table 1). One of these 63 geese also had been crippled by gunshot. Thirty-two (51%) of the 63 lead-poisoned geese were found dead, and 31 were moribund when collected. Lesions consistent with lead poisoning were observed in all geese examined with liver lead concentrations ≥ 8 ppm, wet weight ($n = 60$). In addition, elevated liver lead concentrations were found in 3 geese (2.31, 4.97, and 7.71 ppm, wet weight) that had lesions consistent with lead poisoning; no other causes of morbidity or mortality were found in these geese. We suspect these birds were moribund from lead toxicosis.

Among the 65 lead-poisoned geese (including the 3 suspected cases of lead toxicosis), gross lesions characteristic of lead poisoning were recorded at the following frequencies: atrophy of pectoral muscles and reduction of subcutaneous and internal fat deposits (71%), bile staining of gizzard pads and mucosa (47%), engorgement of gall bladder with thick dark-green bile (46%), proventricular impaction (38%), liver atrophy (30%), esophageal impaction (29%), bile staining of liver (21%), gizzard impaction (18%), apparent myocardial necrosis (18%), bile-staining of feathers around the cloaca (10%), flaccid heart muscle (8%), intestinal atrophy (8%), excess pericardial fluid (6%), submandibular edema (4%), and pale muscles suggestive of anemia (4%).

Ingested lead pellets were found in 52 (83%) of the 63 lead-poisoned geese (mean number of pellets = 9.5, SE \pm 1.8, range = 0 to 80). The mean number of ingested lead pellets in lead-poisoned geese was different at Oak Hammock between 1986 and 1987 (3.1 ± 0.6 vs. 18.2 ± 3.3 , respectively; $t = -4.5$, $P < 0.01$); at Lac qui Parle, these means were not different between years (1.7 ± 0.7 vs. 2.8 ± 1.1 ; $t = -0.76$, $P > 0.10$), although the sample size was small. At Swan Lake and Fountain Grove, the mean number of ingested lead pellets during 1987-88 was 1.0 ± 0.4 . The ratio of ingested steel pellets to lead pellets was 0:557 at Oak Hammock, 0:17 at Lac qui Parle, and 1:18 at Swan Lake/Fountain Grove.

Proportional morbidity and mortality from lead poisoning were not significantly different between 1986 and 1987 at Oak Hammock and at Lac qui Parle ($\chi^2 = 1.30$ and 0.59 , respectively; 1 df, $P \geq 0.20$ and 0.40 , respectively). Data were thus combined over years for each of these 2 sites, and proportional morbidity and mortality compared between areas. Swan Lake/Fountain Grove had the highest proportional mortality from lead poisoning (67%) and Lac qui Parle had the lowest (11%). The proportions were similar at both Oak Hammock and Swan Lake/Fountain Grove (55% and 67%, respectively; $\chi^2 = 1.07$, 1 df, $P \geq 0.25$). The proportional mortality at Lac qui Parle (11%) was lower than either Oak Hammock or Swan Lake/Fountain Grove ($\chi^2 = 21.02$ and 22.97 , respectively; 1 df, $P \leq 0.01$).

Gunshot-related morbidity or death was diagnosed in 59 (37%) of the 158 carcasses. Gunshot-related diagnoses included deaths directly from gunshot ($n = 11$) and gunshot wounds resulting in debilitation ($n = 36$) or peritonitis ($n = 12$). Twenty-three deaths at Lac qui Parle were apparently caused by severe impaction of the lower esophagus with grasses, sedges, and other vegetation. Mucosal tissue in the area of impaction was necrotic and may have lead directly to death, or death may have been

due to resulting secondary infection or to emaciation. There was no evidence of lead poisoning in these birds, and the cause of impaction remains under investigation.

DISCUSSION

Lead poisoning was the most frequently diagnosed cause of morbidity and mortality in this study, accounting for 40% of the 158 carcasses examined. If gunshot-related deaths are excluded, lead poisoning accounted for 64% of the remaining mortality. Because only intact carcasses were examined, our sample may underrepresent predation. However, predation rates on healthy Canada geese on staging and wintering areas appears low (Owen 1980). Carnivorous birds and mammals, particularly bald eagles (*Haliaeetus leucocephalus*), are commonly seen preying on sick or debilitated birds, or scavenging carcasses.

The proportional mortality rate from lead poisoning was greatest at Swan Lake NWR and Fountain Grove WMA (67%), where nontoxic shot has been required for waterfowl hunting since 1978, but not significantly different from Oak Hammock WMA (55%), where lead shot was allowed. The low proportional mortality rate from lead poisoning at Lac qui Parle WMA (11%) is partially explained by the large number of geese that died from esophageal impaction from a yet-unidentified cause. No significant disease outbreaks were found during these 2 yr at either Oak Hammock or Swan Lake/Fountain Grove. We emphasize that proportional mortality reflects the relative importance of lead poisoning to other causes of morbidity and mortality in our sample, but not the actual magnitude of lead poisoning. In addition, methods of carcass collection (viz., search effort, hunted vs. nonhunted areas, during vs. after hunting season) varied between sites, and may have introduced biases in the likelihood of finding geese sick or dying from various causes. Thus, comparisons of proportional mortality between areas must be interpreted with caution. However, the proportional mortality from lead poisoning parallels the prevalence of lead exposure in geese at these 3 sites (DeStefano et al. 1991); where the percent of hunter-killed birds with ≥ 1 ingested shot in the gizzard was 2.5% at Oak Hammock and Swan Lake and 0.6% at Lac qui Parle. In addition, elevated blood lead concentrations were found in 7.6% and 7.5% of live-trapped geese at Oak Hammock and Swan Lake, respectively, and 1.7% of geese from Lac qui Parle during these same years.

We did not attempt to determine the total amount of mortality at any of the study sites. However, we believe that the morbidity and death due to lead poisoning was greatest at Oak Hammock. Sick and dead geese were plentiful throughout the fall at Oak Hammock, and up to 200 carcasses were easily collected in 1987; during 1986, only carcasses found incidental to other field activities were collected at Oak Hammock. In contrast, intensive searches were necessary at Lac qui Parle and Swan Lake to obtain the 89 intact carcasses that were found. Large populations of scavengers, particularly bald eagles and coyotes (*Canis latrans*), were present at study sites in Minnesota and Missouri. Although coyotes, red foxes (*Vulpes vulpes*), and avian scavengers were present at Oak Hammock, their removal of the large numbers of sick and dead geese was unlikely (Humburg and Babcock 1982, Sanderson and Bellrose 1986:19). In addition, many of the carcasses were found on islands inaccessible to scavenging mammals. The large number of ingested lead pellets in geese from Oak Hammock compared to Lac qui Parle and Swan Lake also suggests that geese were at greater risk of ingesting lead pellets and dying from lead poisoning.

Bengtson (1984) examined crippled, moribund, and dead Canada geese collected during 15 October to 15 December from 1978 to 1982 at Lac qui Parle WMA. Nontoxic shot regulations in the Lac qui Parle Steel Shot Zone began in fall 1980. There was a marked decrease in the percentage of birds with >6.0 ppm lead (wet weight) in liver tissue from 63% to

<10% between 1978 and 1980-82 (Table 2). Although the diagnosis of lead poisoning as a cause of morbidity or death in these birds could not be made without accompanying pathological data, lead concentrations above 6.0 ppm in liver tissue suggested these birds may have been suffering from lead toxicosis. Bengtson (1984) suggested this decrease in apparent lead poisoning was partly due to the conversion to nontoxic shot. He also reported the lack of correlation between tissue lead levels and lesions characteristic of lead poisoning. Observed lesions included a high prevalence of impactions and breast muscle atrophy, without corresponding elevated or toxic tissue lead levels. These data suggest that the impaction syndrome of unknown etiology found during 1986 and 1987 at Lac qui Parle also occurred during the early 1980's. The decrease in percentage of moribund and dead birds with >6.0 ppm lead in the liver between 1978 and 1980 to 1982 reported by Bengtson (1984) may have been related to the occurrence of this impaction syndrome during the latter years rather than conversion to nontoxic shot. Further, because esophageal impaction and muscle atrophy are not exclusive for lead poisoning, these observations underscore the need for thorough necropsy with supporting laboratory studies to determine causes of morbidity and mortality. Gross lesions alone are insufficient and can lead to misinterpretation of mortality data.

Humburg and Babcock (1982) and Humburg et al. (1983) used several sampling methods to identify mortality factors in Canada geese at Swan Lake and

Table 2. Percentage of crippled, moribund, and dead Canada geese with lead poisoning collected at Lac qui Parle WMA, Minnesota (1978 to 1982), and Swan Lake NWR and Fountain Grove WMA, Missouri (1977 to 1981). Data adapted from Bengtson (1984), Humburg and Babcock (1982), and Humburg et al. (1983).

Location	No. examined	No. (%) lead poisoning	No. (%) non-lead poisoning
Lac qui Parle^a			
1978	302	190 (63)	112 (37)
1979	161	38 (24)	123 (76)
1980	90	2 (2)	88 (98)
1981	150	5 (3)	145 (97)
1982	93	6 (6)	87 (94)
Swan Lake/Fountain Grove^b			
1977	24	15 (62)	9 (38)
1978	54	14 (26)	40 (74)
1979	25	3 (12)	22 (88)
1980	149	16 (11)	133 (89) ^c
1981	57	3 (5)	54 (95) ^d

^a Geese with >6.0 ppm, wet weight, lead in liver tissue were considered to be suffering from lead poisoning, as discussed in Bengtson (1984), Humburg and Babcock (1982) and Humburg et al (1983).

^b Diagnoses of lead poisoning were made by the University of Missouri Diagnostic Laboratory (Columbia, Mo.); criteria used may be different than those used in the present study.

^c Includes 88 (59%) gunshot, 31 (21%) avian cholera, 8 (5%) aspergillosis, and 6 (4%) impaction.

^d Includes 50 (88%) gunshot, 3 (5%) aspergillosis, and 1 (2%) impaction.

Fountain Grove from 1977 to 1981, during the period of conversion to steel shot regulations in 1978. Lead poisoning decreased from 62% of the carcasses examined in 1977 to 5% by 1981 (Table 2). Proportional mortality from lead poisoning during years of steel shot requirements was considerably lower than that found at Swan Lake and Fountain Grove in this study; however, the data from 1980 and 1981 were collected both during and after hunting seasons, whereas 24 of the 27 carcasses we examined were found after the hunting season. In addition, Humburg et al. (1983) found temporal differences in these sources of mortality, and other diseases occurred at Swan Lake/Fountain Grove during their survey (avian cholera, aspergillosis, and impaction) that were not found in 1987-88.

Despite our inability to estimate the actual magnitude of lead poisoning mortality at staging and wintering areas, lead poisoning evidently still occurs, and in the absence of other concurrent epizootics, can comprise a large proportion of nonhunting mortality. Spent shot in the vicinity of hunting blinds at Swan Lake NWR in 1986 and 1987 showed mean lead shot densities of 13,122 and 14,500/ha, respectively, and a ratio of 1 lead shot:0.6 steel shot in the top 5 cm of soil (DeStefano et al. 1991). This suggests that many years will be required before the density of lead shot and the ratio of lead to steel shot declines sufficiently to markedly reduce the frequency of lead shot ingestion at hunting areas within EPP range. Fredrickson et al. (1977) and Esslinger and Klimstra (1983) found that shot available to waterfowl in hunted agricultural fields is significantly reduced by tillage. However, the area around blinds at Swan Lake NWR had been tilled regularly, suggesting that some shot may be returned to upper soil levels through tillage, as suggested by Esslinger and Klimstra (1983). Manipulation of wetland or agricultural lands may be required to enhance the disappearance of lead shot from certain "hotspots" if we are to hasten the reduction of lead poisoning mortality. Noncompliance with steel shot requirements may also contribute to the continued presence of lead shot near hunting blinds (DeStefano 1989).

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