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SPATIAL AND TEMPORAL VARIATION IN LEAD LEVELS RELATED TO BODY CONDITION IN THE MISSISSIPPI VALLEY POPULATION OF CANADA GEESE

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ABSTRACT: Concern over lead poisoning led to progressive prohibition of toxic shot to harvest waterfowl in the 1980's. Nevertheless, waterfowl remain susceptible to ingestion of lead shot because illegal use continues and spent shot persists in soil and wetland substrates. While mortality due to lead toxicosis has subsided, sublethal effects may still affect survival and reproduction. We measured liver lead levels and body condition in 732 Canada geese (*Branta canadensis interior*) during July 1984 to April 1989 in southern Illinois (USA), east-central Wisconsin (USA), and northern Ontario (Canada). Although we sampled only individuals that were visibly healthy, 55 of 732 (7.5%) geese had elevated liver lead levels (>2 ppm). Lead levels of 46 (6.3%) geese indicated subclinical poisoning (2–6 ppm) and 9 (1.2%) geese had lead levels indicative of clinical poisoning (>6 ppm). A greater proportion of juveniles (14.3%) had elevated lead levels than did adults (6.0%), but there was no difference between genders. Lead levels were highest in autumn and winter in southern Illinois, but were low during nesting and summer, despite legal use of lead shot in northern Ontario during our study. Lead poisoning (≥5% of the population) was still evident during all seasons in juveniles, and during autumn and winter in adults, 5 to 10 yr after toxic shot was banned from areas where we collected geese during migration and winter. Elevated lead levels did not affect total body mass, lipid reserves, or mineral levels of geese we collected. Protein levels also were unaffected below 10 ppm, but there was evidence of decline at higher concentrations. Thus, it seems unlikely that lead exposure currently affects survival or reproduction of Mississippi Valley Population (MVP) geese via body condition, although other sublethal effects cannot be discounted.

Key words: *Branta canadensis interior*, Canada geese, lead poisoning, Mississippi Valley population, sublethal effects, body condition.

INTRODUCTION

Lead poisoning has long been recognized as an indirect source of waterfowl hunting mortality (Adler, 1944; Bellrose, 1959). Although some duck species are more susceptible to lead poisoning, >3,000 Canada geese succumbed during die-offs in southern Illinois (USA) and east-central Wisconsin (USA) in 1977 and 1981 (Sanderson and Bellrose, 1986). Anderson and Havera (1989) reported smaller mortality events involving at least 40 to 450 Canada geese on southern Illinois wintering areas during 1978–1985.

Concern over mortality caused by exposure to ingested lead shot led to progressive implementation of non-toxic shot regulations during the 1980's. Use of lead shot to harvest waterfowl was first prohibited during 1980 in the Horicon Canada goose management zone in east-central Wisconsin, the primary autumn and spring migration staging area for Mississippi Valley Population (MVP) Canada geese. Lead shot was not prohibited statewide in Wisconsin until 1987. Non-toxic shot was first required during 1977 on state and federal public hunting areas in Alexander, Union, Jackson, and Williamson countries in southern Illinois where most MVP geese winter (Anderson and Havera, 1989). Prohibition of toxic shot was extended to private lands in the four county region of southern Illinois in 1985, and was eventually implemented throughout Illinois in 1991. Use of lead shot to harvest waterfowl was legal throughout our study in northern

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Ontario (Canada; Scheuhammer and Norris, 1995) where most MVP geese nest.

Several studies documented pellet ingestion rates and lead levels in blood or body tissues of Canada geese in the Mississippi flyway during the 1980's (Bellrose, 1959; Trainer and Hunt, 1965; Anderson and Havera, 1989; Wheeler, 1995). These studies examined already sick or moribund birds, harvested birds, or birds captured with baited traps thereby introducing sampling bias. Relatively little is known about lead concentrations in apparently healthy Canada geese, particularly after hunting, on spring staging areas, and during the breeding season. We obtained geese almost entirely by selectively shooting birds that were visibly healthy at the time of collection. Concurrent analyses of physiological condition (Gates, 1989; Wheeler et al., 1994) provided opportunity to examine relationships between liver lead concentrations and nutrient reserve levels of free-living Canada geese throughout their annual cycle.

Lead concentrations in liver tissue are considered the best indicator of recent exposure to ingested shot in waterfowl (Bagley and Locke, 1967). Exposure to sublethal lead levels has not been directly compared with body condition in waterfowl, although lead exposure has been associated with weight loss (Hohman et al., 1990). Documentation of sublethal lead levels is particularly important since studies have documented reduced reproductive output (Buerger et al., 1986), anemia (O'Halloran et al., 1989), immunosuppression (Franson, 1986), cardiovascular degeneration (Karstad, 1971), and lesions in the central and peripheral nervous systems (Hunter and Wobeser, 1980) in birds with elevated tissue lead levels.

Our objectives were to (1) estimate proportions of MVP Canada geese exposed to elevated lead levels during the mid to late 1980's as non-toxic shot regulations were implemented; (2) determine annual, seasonal, and geographic variation in lead levels of MVP Canada geese throughout their life cycle; and (3) relate exposure to sublethal concentrations of lead to lipid, protein, and mineral reserves.

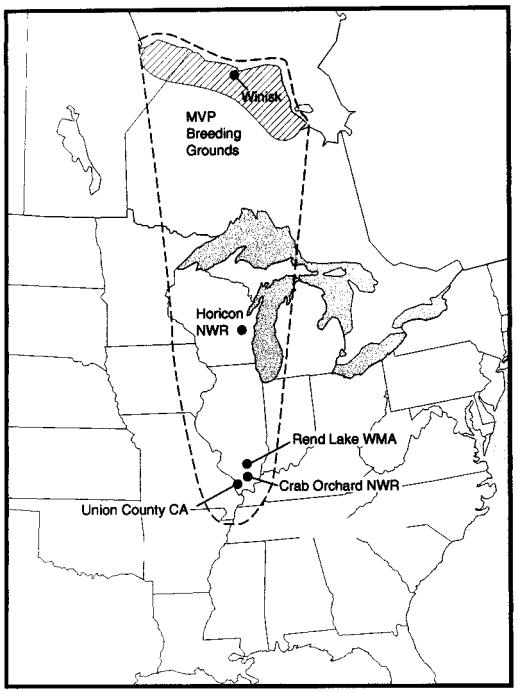


FIGURE 1. Annual range and migration corridor of the Mississippi Valley Population of Canada geese with locations where Canada geese were collected during July 1984 to April 1989 for analyses of lead concentrations in liver tissue and body condition.

STUDY AREAS AND METHODS

We collected geese during July 1984 to April 1989 at seasonal concentration areas throughout the geographic range of MVP Canada geese (Fig. 1). Most (50–80%) of the MVP stage in east-central Wisconsin during autumn, until cold weather and snow cover forces birds to migrate to wintering areas that are principally located in southern Illinois and western Kentucky (USA) (Tacha et al., 1991). A smaller proportion of MVP geese migrate directly to wintering areas from the breeding range in the Hudson Bay Lowlands of northern Ontario (Canada). Spring-migrating geese also stage in east-central Wisconsin during March to April before they migrate to breeding areas in early to mid-April.

Sample Collection

We collected geese mostly at Union County Conservation Area (CA; 37°23′N, 89°22′W) in

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southern Illinois, and within 10 km of Horicon National Wildlife Refuge (NWR; 48°28′N, 88°37′W) in east-central Wisconsin (Horicon Zone) during autumn (October–December), winter (January–February), and spring (March–April). Geese (n = 45) also were collected at Crab Orchard NWR (37°42′N, 89°03′W) and Rend Lake Wildlife Management Area (WMA; 38°03′N, 88°57′W) during autumn and winter in southern Illinois. We also collected geese during the 1985 and 1986 nesting seasons (May-June) ≤5 km from the Hudson Bay Coast, 12 km east of Winisk (Ontario; 55°15′N, 84°58′W; Fig. 1.). Wing-molting geese were collected near Winisk during July in 1984 and 1985, and near the mouth of the Little Shagamu River (55°52′N, 86°37′W), 130 km northwest of the Winisk study site in 1985. Geese collected 12 km east of Winisk after wing-molt (August-September) in 1985 were pooled with wing-molting birds to form a summer sample from northern Ontario. Geese were observed for 2–30 min before collection to identify age and sex (Caithamer et al. 1993). We collected most geese with rifles, although some were killed with shotgun over decoys in northern Ontario or were taken from baited swim-in traps and rocket nets in southern Illinois. Geese were not collected unless they were visibly healthy and behaved normally. Collected geese were weighed, aged, sexed, and then frozen in plastic bags within 8 hr after collection. Carcasses remained frozen 3 to 9 mo before they were thawed, reweighed, measured, and dissected.

Lead Levels

Approximately 10 g of liver tissue was excised from the distal end of the longest liver lobe and frozen. Liver samples were not taken when a penetrating wound to the liver was evident. A 1-g subsample of thawed and ground liver tissue was digested by microwave digestion (Gilman, 1988) and analyzed by Atomic Absorption Spectrophotometry on a Model 2380 spectrophotometer (Perkin-Elmer Corporation, Norwalk, Connecticut, USA) at the Wisconsin Department of Agriculture Trade and Consumer Protection Central Animal Health Laboratory (Madison, Wisconsin, USA). We considered that liver lead concentrations (wet mass) <2 ppm indicated background levels (Friend, 1985). Subclinical poisoning was indicated at 2–6 ppm, clinical poisoning at 6–15 ppm and severe clinical poisoning at >15 ppm (Pain, 1996).

Body Condition

Frozen carcasses were sectioned with a band saw, then ground three times through a commercial meat grinder. Carcass water was estimated from mass loss after drying homogenate to constant mass in a convection oven at 90–110 C (Kerr et al., 1982). Neutral lipids were determined by loss of mass extracting lipids from dried homogenate with petroleum ether (B. P. 33–56 C) on a Goldfisch fat extractor (Dobush et al., 1985). Mineral content was determined from ash residue after burning lean dry homogenate in a muffle furnace for 16–20 hr at 600 C.

Percentages of water, lipid, and ash were multiplied by masses of plucked ingesta-free carcasses without feet and bill to estimate carcass composition. Ash-free lean dry mass (AFLDM) was obtained by subtracting water, neutral lipid, and ash masses from carcass mass. AFLDM is mostly protein but includes other minor fractions (structural lipids, carbohydrates, and non-protein nitrogenous compounds) that are relatively constant in animal carcasses (Robbins, 1993). Gates (1989) provided a more detailed description of procedures used to determine carcass composition.

Data Analysis

We tested the normality of distributions of wet and dry mass concentrations of lead in liver tissue with the Univariate procedure of the Statistical Analysis System (SAS) (SAS, 1985). Wet and dry mass concentrations were highly skewed (14.2–14.5) and kurtotic (234.7–246.8) so we $\log_{10}$-transformed liver lead concentrations before statistical analyses. Transformation did not completely normalize the data (W = 0.93–0.94, $P < 0.001$), but skewness (1.1–1.2) and kurtosis (4.0–4.6) were markedly reduced. Although dry mass concentrations are considered more reliable (Adrian and Stevens, 1979), statistical comparisons based on wet and dry mass concentrations produced equivalent results. Consequently, we report only comparisons based on wet mass concentrations for consistency with previously published values. We used geometric means of liver lead concentrations (antilog$_{10}$ of means of transformed values) as a measure of central tendency and the ranges of untransformed wet mass concentrations as a measure of dispersion.

We compared lead concentrations ($\log_{10}$-transformed) in liver tissue among years, seasons, and locations with 1- and 2- factor analyses of variance (ANOVA). Separate ANOVA models were used to compare lead concentrations among (1) seasons (autumn, winter, spring migration, nesting, summer) across and within locations and years; (2) years (1984–85 versus 1985–86) during autumn, winter, spring migration, and nesting; (3) years (1985-1988) during

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spring migration within adult females; and (4) locations (southern Illinois versus east-central Wisconsin) during autumns 1984 and 1985.

Analyses were conducted with adults and juveniles separated and pooled. Tests were conducted with the General Linear Models (GLM) procedure of SAS. We used the Ryan-Einot-Gabriel-Welsch multiple range test (option REGWQ of the SAS GLM procedure) to separate differences among group means when global *F*-tests from 1-factor ANOVA models were significant ($P \leq 0.05$). Pairwise differences among main and simple effect least-squares means were tested using the GLM LSMEANS and PDIFF options because cell sample sizes were unbalanced (Milliken and Johnson, 1984).

Proportions of geese with background versus above background lead concentrations in liver tissue also were compared among locations, seasons, years, and age classes. With data sets partitioned identically to ANOVA models, we compared dose levels among age classes, years, locations, and/or seasons using *G*-tests in the SAS frequency (FREQ) procedure.

We also examined the relationship between liver lead concentrations and individual variation in body condition after removing differences attributable to years, seasons, locations, and sex-age classes. Residual values were output from a means model where all combinations of years, seasons, locations, and sex-age classes were represented with their own data cells (Milliken and Johnson, 1984). We regressed residual lipid, AFLDM, and ash values on $\log_{10}$-transformed wet mass lead concentrations with SYSTAT (Wilkinson et al., 1996). Second order quadratic equations were fit separately for geese with lead concentrations at or below versus above background levels.

RESULTS

Lead Concentrations

We determined lead concentrations in liver tissues of 732 Canada geese collected in Illinois, Wisconsin, and Ontario during July 1984 to April 1989. Only 55 geese (7.5%) had elevated liver lead levels (Fig. 2). Liver lead levels of nine (1.2%) indicated clinical or severe clinical poisoning.

Geometric mean (GM) lead levels (range) were 0.63 ppm (0.10–40.00) in 341 adult females, 0.69 ppm (0.10-9.80) in 244 adult males, 0.90 (0.18–11.00) in 62 juvenile females, and 0.79 ppm (0.08–54.00) in 85 juvenile males. Lead levels differed ($F_{1,728} = 13.48$, $P < 0.01$) between adults

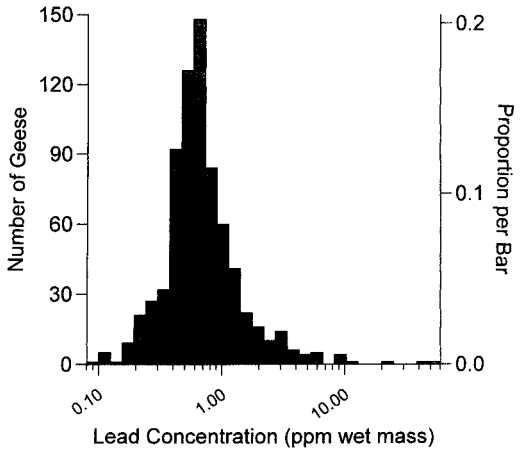


FIGURE 2. Frequency distribution ($\log_{10}$ scale) of lead levels in liver tissue of 732 Canada geese collected in southern Illinois, east-central Wisconsin, and northern Ontario during July 1984 to April 1989.

(GM = 0.66 ppm) and juveniles (GM = 0.84 ppm), but were similar ($F_{1,728} = 0.06$, $P = 0.81$) in males (GM = 0.72) and females (GM = 0.67). Age group differences were similar in males and females ($F_{1,728} = 3.04$, $P = 0.082$) so we pooled sexes for further comparisons. A larger ($G_1 = 10.45$, $P = 0.001$) proportion of juveniles (14.3%) had elevated lead levels compared to adults (5.8%).

Lead levels varied among seasons with pooled age classes ($F_{4,644} \leq 13.05$, $P < 0.01$). Seasonal differences also occurred for adults ($F_{4,498} = 10.66$, $P < 0.01$), and were nearly significant for juveniles ($F_{4,141} = 2.42$, $P = 0.051$). Mean lead levels were highest in winter, lowest in spring and summer, and intermediate during autumn and nesting (Table 1). Percentages of adults with elevated lead levels ranged from 2.1% during nesting to 11.8% during winter, and for juveniles ranged from 8.3% during summer to 20.0% during winter (Fig. 3). Proportions of geese with elevated lead levels did not vary among seasons within age classes ($G_4 \leq 7.18$, $P \geq 0.127$), but seasonal differences were nearly significant with age-classes combined ($G_4 = 9.43$, $P = 0.051$).

We collected geese in two consecutive years (1984 and 1985) during autumn,

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TABLE 1. Geometric mean (range) *n* concentrations of lead (ppm wet mass) in liver tissues of adult and juvenile Canada geese collected July 1984 to April 1986 in east-central Wisconsin (autumn and spring), southern Illinois (autumn and winter), and northern Ontario (summer).

Season	Adult		Juvenile		All ages	
Autumn	0.67 (0.11–40.00)	179 AB[a]	0.76 (0.18–9.00)	65 A	0.69 (0.11–40.00)	244 BC
Winter	0.84 (0.17–5.70)	102 A	1.14 (0.37–54.00)	40 A	0.92 (0.17–54.00)	142 A
Spring	0.55 (0.10–9.80)	133 C	0.70 (0.30–3.10)	21 A	0.56 (0.10–9.80)	154 D
Nesting	0.80 (0.30–8.50)	47 AB	0.96 (0.57–3.80)	12 A	0.83 (0.30–8.50)	59 AB
Summer	0.61 (0.29–2.80)	42 BC	0.51 (0.08–2.40)	8 A	0.60 (0.08–2.80)	50 CD

[a] Column means sharing the same letter were not significantly different ($P > 0.05$, Ryan-Einot-Gabriel-Welsch multiple range test).

winter, spring migration, and nesting seasons. Lead levels were higher ($F_{1,591} \geq 56.55$, $P < 0.01$) in 1984 (GM = 0.93 ppm, range = 0.10–54.00) than in 1985 (0.58 ppm, range = 0.10–11.00). Lead levels differed among years within all seasons in adults, but only during winter in juveniles (Table 2). Liver lead concentrations exceeded background in 15.5% of geese in 1984, compared to only 3.1% in 1985 ($G_1 = 29.78$, $P < 0.01$). Lead concentrations exceeded background in 10.5% (autumn) to 23.3% (winter) of adults during 1984 (Fig 3), compared to 0.0% (autumn, nesting) to 3.4% (winter) of adults during 1985. Lead levels were elevated in 0.0% (spring, nesting) to 31.5% (winter) of juveniles during 1984, compared to 7.7% (spring) to 14.3% (nesting) of juveniles during 1985.

The longest time span over which we observed lead levels in a single season was during springs 1985–1988 when we collected only adult females during spring migration in east-central Wisconsin. Lead levels varied annually ($F_{3,156} = 8.91$, $P < 0.001$), declining from 0.63 ppm (range 0.20–3.10) in 1985 to 0.43 ppm (range 0.10–1.80) in 1986 ($P < 0.05$). Similarly

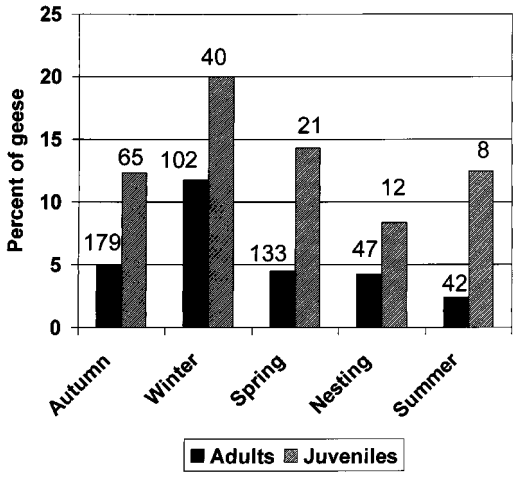


FIGURE 3. Proportions of adult and juvenile Canada geese with background versus elevated (≥ 2 ppm) wet mass concentrations of lead in liver tissue in east-central Wisconsin, southern Illinois, and northern Ontario during July 1984 to September 1986. Sample sizes appear above each bar.

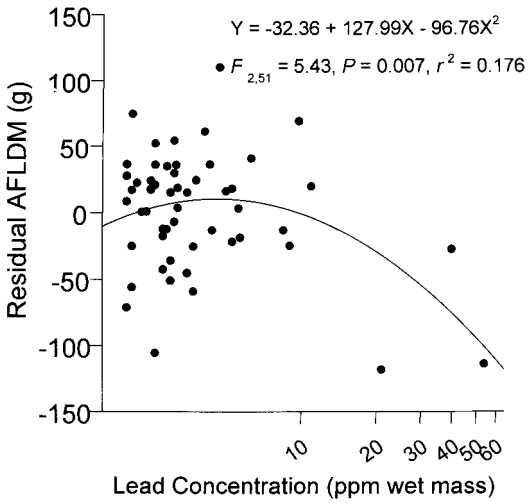


FIGURE 4. Regression of residual (deviation of individuals from their respective annual, seasonal, location, and age-group cell means) ash-free lean dry masses (AFLDM) on lead levels in liver tissue of 53 Canada geese collected in southern Illinois, east-central Wisconsin, and northern Ontario with lead levels that exceeded background (>2 ppm wet mass) during July 1984 to April 1989.

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TABLE 2. Geometric mean (range) *n* concentrations of lead (ppm wet mass) in liver tissues of adult and juvenile Canada geese collected October 1984 to June 1985 in southern Illinois, east-central Wisconsin, and northern Ontario.

Season	Year	Adult		Juvenile		All ages	
Autumn	1984	0.80 (0.11–40.00)	86	0.83 (0.18–6.30)	30	0.81 (0.11–40.00)	116
Autumn	1985	0.57 (0.18–1.90)	93	0.71 (0.19–9.00)	35	0.60 (0.18–9.00)	128
	1984 vs. 1985	$P < 0.01$		$P = 0.450$		$P = 0.001$	
Winter	1984	1.27 (0.50–5.70)	43	1.89 (0.45–54.00)	19	1.43 (0.45–54.00)	62
Winter	1985	0.62 (0.17–3.00)	59	0.78 (0.37–11.00)	21	0.65 (0.17–11.00)	80
	1984 vs. 1985	$P < 0.001$		$P = 0.001$		$P = 0.001$	
Spring	1984	0.66 (0.10–3.50)	39	0.76 (0.30–2.00)	8	0.67 (0.10–3.50)	47
Spring	1985	0.44 (0.10–9.80)	94	0.66 (0.35–3.10)	13	0.46 (0.10–9.80)	107
	1984 vs. 1985	$P < 0.001$		$P = 0.718$		$P = 0.002$	
Nesting	1984	1.27 (0.30–8.50)	15	1.02 (0.70–1.30)	5	1.21 (0.30–8.50)	20
Nesting	1985	0.65 (0.40–1.90)	32	0.92 (0.57–3.80)	7	0.69 (0.40–3.80)	39
	1984 vs. 1985	$P < 0.001$		$P = 0.840$		$P = 0.003$	

low lead levels ($P > 0.05$) were observed in 1987 (GM-0.54, range = 0.25–0.97), but lead levels increased ($P < 0.05$) to 0.76 ppm (range 0.10–5.60) in 1988, to a similar level to 1985. Background lead levels were exceeded in 10.0–14.3% of adult females in 1985 and 1988, compared to only 0–2% in 1986 and 1987 ($G_3 = 14.27$, $P < 0.01$).

Lead levels averaged higher in adults from southern Illinois (GM = 0.79 ppm, range 0.18–40.00) than in adults from east-central Wisconsin (GM = 0.57 ppm, range 0.11–8.50) during autumns 1984-1985 ($F_{1,175} = 12.71$, $P < 0.01$). No differences between locations were found in juveniles ($F_{1,61} = 0.16$, $P = 0.69$). Lead concentrations were higher ($P < 0.01$) in adults from southern Illinois (GM = 1.04 ppm, range 0.21–40.00) than in adults from east-central Wisconsin (GM = 0.63 ppm, range = 0.11–8.50), but we found no differences between east-central Wisconsin (GM = 0.52 ppm, range = 0.22–1.00) and southern Illinois (GM = 0.62, range = 0.18–1.90) among adults in 1985.

Six of 42 (14.3%) adults and 3 of 19 (15.8%) juveniles exceeded background lead levels in southern Illinois, compared to 3 of 44 (6.8%) adults and 1 of 11 (9.1%) juveniles in east-central Wisconsin during autumn 1984. No adults exceeded background levels at either location in autumn 1985, but 3 of 19 (15.8%) juveniles in east-central Wisconsin and 1 of 16 (6.2%) juveniles in southern Illinois exceeded background lead levels during autumn 1985.

Body Condition Effects

We found no differences in residual body mass, lipid or AFLDM between geese with liver lead concentrations that were above versus below background levels ($F_{1,729} \leq 0.76$, $P \geq 0.38$). However, residual AFLDM declined ($F_{2,51} = 5.43$, $P < 0.01$) with wet mass concentrations of lead among geese that had elevated lead levels in liver tissue (Fig. 4). Liver lead levels explained 17.6% of variation in residual AFLDM, but the statistical relationship was highly leveraged by our 2 most extreme observations of lead levels (40–54 ppm). The curvilinear trend indicated that AFLDM was not depressed until geese exceeded 10 ppm wet mass of lead in liver tissue. Furthermore, we found no relationship between lead levels and residual lipid or ash values ($F_{2,52} \leq 0.58$, $P \geq 0.63$) among geese with elevated lead concentrations. We also found no relationship of liver lead levels with body mass, lipid, AFLDM, or ash residuals among geese with lead concentrations at or below background ($F_{2,671} \leq 0.05$, $P \geq 0.95$).

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DISCUSSION

Our sample represented an apparently normal, healthy cross-section of the MVP interior Canada geese. Any sampling bias would have excluded sick or moribund birds not behaving normally at time of collection, so our estimates of lead concentrations may have been slightly conservative for the entire population. We also collected geese almost entirely within zones where toxic shot had been prohibited for 5 to 8 yr in east-central Wisconsin and for 8 to 10 yr in southern Illinois. Liver lead levels generally reflect recent exposure to ingested lead (Pain, 1996). The primary sources of lead exposure in our study likely included non-compliant hunters, (Simpson, 1989) residual shot in soil or wetland substrates (Esslinger and Klimstra, 1983), or lead shot legally discharged outside of non-toxic shot zones where geese possibly fed before they were collected. We believe the latter source to be the least important source of lead because radiotelemetry data indicated that daily movements of geese we studied were largely restricted to areas where use of toxic shot to harvest waterfowl was prohibited (Caithamer, 1989).

We found elevated liver lead levels in nearly 8% of MVP Canada geese during 1984–1988. Elevated lead levels in >5% of individuals is considered an "excessive" level of lead poisoning in waterfowl populations (U.S. Department of Interior, 1986). Juveniles exceeded this threshold in all seasons, and adults met or exceeded the threshold during autumn and winter. DeStefano et al. (1991) reported similar proportions (<1–10%) of geese with elevated blood lead levels (≥0.18 ppm) across the geographic range of Eastern Prairie Population Canada geese during 1986–1988. We found, as did DeStefano et al. (1992), that juvenile Canada geese had higher lead levels than adults on winter and migration areas, but lead levels did not differ between adult males and females. DeStefano et al. (1992) attributed the difference between age classes to higher rates of food intake in juveniles.

Grain-dominated diets enhance digestive absorption of ingested lead (Sanderson and Bellrose, 1986). The high lead levels that we observed during winter in southern Illinois might be attributable to diets that consisted of >80% corn and soybeans during 1985 and 1986 (Gates, 1989). Geese consumed more corn in east-central Wisconsin than in southern Illinois, but lead levels differed little between southern Illinois and east-central Wisconsin during autumns 1984 and 1985.

Interestingly, lead levels were lowest in geese from northern Ontario where use of lead shot remained legal. DeStefano et al. (1991) also found the lowest incidence of elevated blood lead levels in Canada geese from Cape Churchill in northern Manitoba (Canada). Fewer hunters, shorter hunting seasons, different vegetation communities and soils, and forage-dominated diets may diminish risk of exposure to toxic shot in the Hudson Bay lowlands (Canada).

Anderson and Havera (1989) found that 24% of MVP geese harvested on public hunting areas in southern Illinois had elevated blood lead levels (≥2 ppm) during the 1981–1983 hunting seasons. We observed smaller proportions of geese with elevated liver lead levels during autumns 1984–1985 in east-central Wisconsin (6%) and southern Illinois (8%) and during winters 1985–1986 in southern Illinois (14%). Liver lead levels also declined between 1984–1985 and 1985–1986. However, if exposure to ingested lead declined in MVP Canada geese during the early and mid 1980's as these studies suggest, the decline was not evident in adult females that we collected in east-central Wisconsin during springs 1985–1988.

We expect that lead levels in MVP Canada have declined somewhat since the 1980's with more widespread prohibition of toxic shot. Our expectation also assumes that spent lead shot has become less available to geese over time, and that hunters have become more compliant with toxic shot regulations. However, use of toxic

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shot remains legal for harvest of upland game and for trap and skeet shooting in areas where geese often feed, so exposure to toxic shot remains a health risk for MVP Canada geese and other waterfowl populations.

Reported incidences of large die-offs due to lead poisoning declined after the early 1980's, (Anderson and Havera, 1989; Wheeler, 1995) indicating that localized implementation of toxic shot regulations was effective. However, documented cases of lead toxicosis persist, although they seem to have become more sporadic and involve fewer individuals than in the past. Nevertheless, more recent estimates of lead levels in Canada geese would be useful in evaluating the effectiveness of statewide toxic shot bans and whether geese continue to be exposed to lead shot that persists in soil. Our results provide a baseline for such comparisons.

Although mortality due to lead poisoning has diminished over time, sublethal effects persist as geese will continue to be exposed to toxic shot. We found that body condition was not adversely affected by liver lead levels <10 ppm. There was weak evidence that protein levels were affected above 10 ppm, but few of our birds had liver lead levels this high. Other sublethal effects such as diminished immunological function and disease resistance (Rocke and Samuel, 1991), neurological dysfunction (Dieter and Finley, 1979), and reduced survival (Samuel et al., 1992) found in other waterfowl cannot be discounted for Canada geese.

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