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A COMPARISON OF BLOOD LEAD LEVELS IN BALD EAGLES FROM TWO REGIONS ON THE GREAT PLAINS OF NORTH AMERICA

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ABSTRACT: The connection between bald eagles (*Haliaeetus leucocephalus*) and the consumption of waterfowl, lead shotshell pellet ingestion, and subsequent lead exposure is well documented in the United States and is presumed also to be widespread in Canada. We compared blood lead concentrations in samples from bald eagles ranging in age from 0.5- to 1.5-yr-old at Galloway Bay, Saskatchewan, Canada (n = 97) during October–November, 1992–95 and Hauser Lake, Montana, USA (n = 81) during October–December, 1990–94, within the Great Plains region of North America. Abundant prey are available in the form of hunter-injured waterfowl in Saskatchewan and spawning salmon in Montana; both areas attract large numbers of mid-continent bald eagles during fall migration. Blood lead concentrations suggestive of recent lead exposure (>0.201 μg/ml) were found in 32% and 8% of eagles at Hauser Lake and Galloway Bay, respectively, when samples from each study area were analyzed independently at two laboratories. To determine if this difference was an artifact of interlaboratory variation, we determined a correction factor by reanalyzing 14 Saskatchewan blood samples at each laboratory and predicted blood lead concentrations from Hauser Lake had the samples been analyzed at the Canadian laboratory. Adjusted blood lead concentrations of samples from Hauser Lake indicated that 21% of eagles were recently exposed to lead, a proportion not significantly different from the proportion of the same exposure category at Galloway Bay. Our data do not support the supposition that a large proportion of bald eagles feeding on waterfowl in areas of high hunting pressure will be exposed to lead via consumption of lead shotshell pellets in waterfowl.

Key words: Bald eagle, blood lead, *Haliaeetus leucocephalus*, lead exposure, lead shotshell pellets, waterfowl.

INTRODUCTION

Concentrations of lead in blood often are used to monitor free-ranging avian populations for lead exposure (Mautino and Bell, 1987). The presumed major source of lead available to raptors is from bullet fragments or pellets that are either embedded in tissue or have been ingested by prey (e.g., Redig et al., 1980; Pattee and Hennes, 1983; Gill and Langelier, 1994). Bald eagles (*Haliaeetus leucocephalus*) often rely heavily on wounded birds or carrion likely to contain lead (Gerrard and Bortolotti, 1988), and thus are extremely vulnerable to lead poisoning (Pattee and Hennes, 1983). Lead exposure and lead toxicosis as a result of lead shotshell pellet ingestion have been documented previously in bald eagles (e.g., Mulhern et al., 1970; Hennes, 1985; Langelier et al., 1991). The deleterious impact of lead poisoning on the once endangered population of bald eagles in the conterminous United States is well documented, and was partially responsible for the implementation of the nationwide ban on the use of lead shotshell ammunition for hunting waterfowl (U.S. Fish and Wildlife Service, 1986).

Pattee and Hennes (1983) speculated that lead poisoning in bald eagles was like-

ly when large numbers of waterfowl are concentrated in a restricted area, hunters are active in these areas, and eagles gather in these same areas and feed on birds killed or injured by hunters. The greatest number of lead induced eagle mortalities has been documented to occur between the late fall and early winter months (Feierabend and Myers, 1984; Elliott et al., 1992). This time period also coincides with the advent of abundant food resources in the form of waterfowl which have been injured by hunters (Griffin et al., 1982).

Our study focused on the mid-continental population of bald eagles that nest in the Canadian boreal forest regions of Manitoba, Saskatchewan, Alberta, and the Northwest Territories and migrate south beginning in October to wintering regions throughout the western United States and Mississippi River basin (Gerrard and Bortolotti, 1988; McClelland et al., 1994). This study was developed *a posteriori* from a collaboration of two independent research projects to compare blood lead levels in bald eagles at two different sites, one where eagles have ample opportunity for feeding on injured waterfowl and the other where they feed on fish. Based on the supposition of Pattee and Hennes (1983), we suspected that the population of eagles feeding on waterfowl would exhibit a higher prevalence of lead exposure and higher median blood lead concentrations than would the piscivorous population.

MATERIALS AND METHODS

We examined bald eagles for lead exposure at migratory stopover areas located at Galloway Bay (Saskatchewan, Canada; 50°48′N, 108°27′W), and Hauser Lake (Montana, USA; 46°41′N, 111°49′W). Galloway Bay, situated on a major migration corridor for mid-continent white-fronted geese (*Anser albifrons*), Canada geese (*Branta canadensis*), snow geese (*Chen caerulescens*) and sandhill cranes (*Grus canadensis*), is a 60 km^2 portion of the larger Lake Diefenbaker impoundment of the South Saskatchewan River, consisting of shallow bays and sandbars favourable for staging waterfowl. Up to 700,000 geese and cranes stage in this area between mid-September and late November (Roy, 1996). In addition to the large congregations of waterfowl, large numbers of migrant bald eagles also use the area, where up to 123 eagles were recorded in a single count (M. J. R. Miller, unpubl. data). The area also is used extensively in the autumn for goose hunting. Hauser Lake is a 23 km long impoundment of the upper Missouri River in west-central Montana. In the autumn, up to 300 bald eagles temporarily stopover in the area to feed on dead and spawning kokanee salmon (*Oncorhynchus nerka*) which were introduced in 1978 (Restani, 1997).

Because this was an opportunistic comparison of independent research projects, the methods used to capture eagles, assess food habits and to determine blood lead concentrations were different, and are described below. Eagles were trapped from the beginning of October to mid-November, 1992–95 at Galloway Bay, and from mid-October to mid-December, 1990–94 at Hauser Lake. Eagles were trapped using padded, weakened leg-hold traps with offset jaws (Victor coil-spring #3, Victor double-spring #3N, and Victor long-spring #4 traps, Oneida Victor Trap Corporation, Niagara Falls, Ontario, Canada) following the modified Lockhart method described by Harmata (1984). At Galloway Bay, traps were placed on small islands around staked-down waterfowl carcasses free of lead shotshell pellets that were obtained either from a culling operation of an urban population of Canada geese or that had been collected in the study area and subsequently fluoroscoped to assure the absence of lead shotshell pellets. Traps at Hauser Lake were placed around salmon carcasses staked in shallow water along shorelines. Eagles were aged according to plumage characteristics suggested by McCollough (1989).

For Saskatchewan eagles, 8 to 10 ml of blood was collected from the brachial vein using a 21-gauge winged hypodermic needle (Venisystems "Butterfly-21," Abbott Laboratories Ltd., St. Laurent, Québec, Canada) attached to a 12 cc syringe that had been flushed with heparin (Hepalean-heparin sodium injection, Organon Teknika Inc., Toronto, Ontario, Canada). Care was taken to gently roll the syringe along its longitudinal axis during blood sampling to allow for adequate mixing of blood with the anticoagulant. Blood was then transferred to labelled, nitric acid-rinsed cryovials (Nunc Inc., Rochester, New York, USA). Vials were then placed in liquid nitrogen for transport and storage.

At Hauser Lake, 3 to 8 ml of blood drawn from the brachial vein of each bird was placed in sodium heparinized Vacutainers (Becton-Dickinson, Rutherford, New Jersey, USA).

Whole blood was frozen on ice within 1 hr of collection, and later stored in −20 C freezers.

Lead concentrations (μg/ml) in blood samples from Saskatchewan were determined by graphite furnace atomic absorption spectrophotometry (GFAAS) at the National Wildlife Research Centre (NWRC) of Environment Canada (Hull, Québec, Canada) using a 3030B spectrophotometer equipped with HGA-300 graphite furnace and an AS-40 autosampler (Perkin-Elmer Ltd., Norwalk, Connecticut, USA). Pyrolytically coated graphite tubes with L-vov platform were used in conjunction with solid pyrolytic graphite platforms (Perkin-Elmer Ltd., Norwalk, Connecticut, USA). Argon was used as the purge gas.

One-hundred μl of whole blood were pipetted from the field-sample cryovials into a 1500 μl micro centrifuge tube (Eppendorf-Nethelar-Hinz Gmbh, Hamburg, Germany) containing 400 μl of a diluent solution containing 0.2% diammonium hydrogenphosphate ($NH_4H_2PO_4$) (Lot. #861272, Fisher Scientific, Nepean, Ontario, Canada), and 0.5% Triton X-100 (Lot. #76C-0343, Sigma-Aldrich, Oakville, Ontario, Canada). The tube was then capped and vortexed for 10 sec. Blank solutions of 0.5% HNO_3 (FW.63.01—Analyzed Reagent for Trace Metal Analysis, J. T. Baker, Mississauga, Ontario, Canada), 0.2% $NH_4H_2PO_4$ and 0.5% Triton X-100 were prepared and analyzed to verify the absence of lead in those solutions.

Standards were prepared in 0.5% HNO_3 with 0.2% $NH_4H_2PO_4$ as the matrix modifier. Accuracy was determined by analyzing blood samples supplemented (spiked) with 10 and 20 μg/l lead (Lead Reference Solution, Lot. #903563-24, cat.93/RS, Fisher Scientific, Nepean, Ontario, Canada). Recovery of spiked samples ranged between 89 and 102%. Analytical precision was measured by analyzing 10 samples in duplicate and seven samples in triplicate. The standard deviation of duplicate analyses was <10%. The practical detection limit (DL) was 0.010 μg/ml.

Blood samples from Montana were analyzed for lead at the Montana State University (MSU; Agricultural Experiment Station Analytical Laboratory, Bozeman, Montana, USA) using an AA40 spectrophotometer with a GTA96 carbon furnace and a PS55 autosampler using pyrolytically coated tubes and platforms (Varian Ltd., Palo Alto, California, USA). Samples were thawed and vortexed for 15 sec. Blood (0.5 to 1.5 ml) was drawn from the vials with a Pasteur pipette, and transferred to a 120 ml, hot acid cleaned, Teflon® microwave vessel. Eight ml of Trace Metal grade 66% Nitric Acid (Cat. #A2004X-212, Fisher Scientific, Pittsburgh, Pennsylvania, USA) was added to the samples. The vessels were sealed in a capping station and allowed to predigest overnight. The samples were digested in a microwave oven with pressure control. The samples were then transferred by rinsing with double deionized water and made to volume in 25 ml volumetric flasks. Standards were prepared in 20% nitric acid. One per cent H_3PO_4 was used as a matrix modifier and added to both standards and samples. Reagent blanks were included in each string of ≤10, and showed no measurable interference. The in-house liver quality control and blood spike samples were run with each set of ≤10 samples. Recovery of samples spiked with 25 μg/l inorganic lead ranged between 106 and 121%. A Fisher Scientific certified reference lead standard solution (Cat. #SL21-100) was diluted and used to make standards (0.0–50.0 μg/l) and the spiking solution. Analytical precision was measured by analyzing 11 samples in duplicate on different days. The standard deviation of duplicate analyses ranged from 0.00 to 20%. All but two duplicates had <8% standard deviation. Detection limits varied during the years, decreasing from 0.1 μg/ml in 1990 to 0.025 μg/ml for analysis of blood collected from 1992–94.

Fourteen blood samples representing the range of lead concentrations from Saskatchewan were used in an interlaboratory calibration. Approximately 1 ml of blood was removed from each of the sample cryovials for analysis at NWRC and remainder was sent to MSU for analysis using the previously described technique. Accuracy of the MSU analysis was determined by analyzing spiked samples (25 μg/l lead). Recovery of spiked samples ranged between 92 and 116%, based on three measurements of each sample. Precision in the 14 blood samples was measured by analyzing three samples in duplicate and one sample in triplicate. Standard deviation of the three duplicate analyses ranged from 2 to 15%.

Following Wiemeyer et al. (1989), we assigned a value equal to one-half of the laboratory detection limit (DL) to samples in which lead was not detected. Blood lead levels were highly skewed to the right and could not be normalized using appropriate transformations; therefore, nonparametric statistical techniques were used for analysis (Siegel and Castellan, 1988). A one-tailed Wilcoxon-Mann-Whitney test was used to compare the medians of blood lead concentrations between the two areas. Chi-square analysis with Yates' correction for continuity was used to compare frequency of occurrence of elevated blood lead concentrations. Statistical significance was assigned at $P \leq 0.05$.

The four categories of blood lead concentra-

TABLE 1. Summary of blood lead concentrations (μg/ml, wet weight) in bald eagles captured between October through November 1992–95, Galloway Bay (GB; South Saskatchewan River, Saskatchewan) and October through mid-December 1990–94, Hauser Lake (HL; Montana).

Study site	Laboratory[a]	Number sampled	Number with detectable lead[b]	Blood lead concentration		
				Median	Minimum	Maximum
GB	NWRC	97[c]	63	0.020[e]	ND	0.585
HL	MSU	81[d]	65	0.120	ND	2.100
HL	MSU_{NWRC}	81	53	0.035	ND	2.080

[a] NWRC = National Wildlife Research Center, MSU = Montana State University, MSU_{NWRC} = $MSU_{adjusted\ to\ NWRC}$, as described in text.

[b] Detection limit (DL) for NWRC and MSU_{NWRC} was 0.01 μg/ml; DL for MSU was 0.025 μg/ml.

[c] Age class composition – age in years: 0.5 (n = 62); 1.5 (n = 35).

[d] Age class composition – age in years: 0.5 (n = 58); 1.5 (n = 23).

[e] No difference in median blood lead concentration detected between years (1992–95) (Kruskal-Wallis 1-way ANOVA, $KW_{corrected\ for\ ties}$ = 4.680, df = 3, P = 0.197).

tions on a wet weight basis suggested by Redig et al. (1983) were used to indicate levels of exposure: blood lead concentrations <0.200 μg/ml were considered background levels; concentrations of 0.201 to 0.600 μg/ml indicative of recent, acute exposure; concentrations in the range of 0.601 to 1.0 μg/ml indicative of chronic, clinical exposure; and concentrations >1.0 μg/ml indicative of acute clinical exposure.

Prey remains consisting of whole or partially consumed carcasses were salvaged underneath eagle roost trees and elsewhere throughout Galloway Bay. Mersmann et al. (1992) indicated that analysis of prey remains often overrepresented birds and large bony fish in the diet and understated the frequency of large items which may not have been brought back to roosts where they would have been collected. Observations of Bald Eagles foraging within the study areas were made incidental to field work at Galloway Bay using 22× spotting scope or 7× binoculars and during set observation times at Hauser Lake using 20–60× spotting scope or 10× binoculars. When eagles were observed consuming a prey item, the prey item was identified either at a distance through a spotting scope or, at Galloway Bay, by retrieving the item after the eagle(s) had ceased feeding or until flushed by an observer. Although labour intensive when compared to analysis of regurgitates or prey remains, Mersmann et al. (1992) determined that this method of determining food habits was the least biased technique.

All research methods employed during this study were approved by the Animal Care Committee (University of Saskatchewan, Saskatoon, Saskatchewan, on behalf of the Canadian Council on Animal Care, Ottawa, Ontario, Canada), and the Institutional Animal Care and Use Committee of Montana State University (Bozeman, Montana, USA) and Utah State University (Logan, Utah, USA).

RESULTS

One hundred and seventy-eight eagles of two age classes were sampled (Galloway Bay, n = 97; Hauser Lake, n = 81). Although 67% of samples were from eagles within their first year, blood lead data were pooled across age classes for analysis (Table 1). A significantly greater proportion of bald eagles at Hauser Lake had elevated lead levels than at Galloway Bay (χ^2 = 13.490, P = 0.0002) (Table 2). The difference between median blood lead levels (unadjusted for interlaboratory variation) from eagles at Hauser Lake and Galloway Bay also was significant (Wilcoxon-Mann-Whitney, $Z_{corrected\ for\ ties}$ = −7.263, P < 0.0001) (Table 1).

Lead in the 14 blood samples analyzed at both MSU and NWRC were significantly different (Wilcoxon signed ranks, T^+ = 103, P = 0.0004). Therefore, to derive an adjustment factor, we regressed blood lead concentrations from samples analyzed at MSU on fractions of the same samples analyzed at NWRC using Kendall's robust line-fit nonparametric regression in lieu of parametric regression techniques (Sokal and Rohlf, 1995) (Fig. 1). The regression equation was MSU analyzed$_{[blood\ Pb]}$ = 0.968 NWRC analyzed$_{[blood\ Pb]}$ + 0.0864,

TABLE 2. Lead exposure categories (Redig et al., 1983) for autumn migrant bald eagles captured at Galloway Bay (Saskatchewan) and Hauser Lake (Montana). Adjusted blood lead values for samples from Hauser Lake were determined from a regression equation derived from 14 samples from Galloway Bay re-analyzed at the National Wildlife Health Centre of Environment Canada and Montana State University.

Blood lead concentration exposure category (μg/ml)	Hauser Lake				Galloway Bay	
	Original data		Adjusted to NWRC		Original data	
	(*n*)	(%)	(*n*)	(%)	(*n*)	(%)
<0.200	55	(68)	66	(82)	89	(92)
0.201–0.600	24	(30)	13	(16)	8	(8)
0.601–1.000	0	(0)	0	(2)	0	(0)
>1.000	2	(3)	2	(3)	0	(0)
Total	81		81		97	

to which there was a significant correlation (Kendall's rank order correlation coefficient, $T_{\text{corrected for ties}} = 0.595$, $P = 0.0031$). By modifying the above equation to Hauser Lake$_{[\text{blood Pb adjusted to NWRC}]}$ = (Hauser Lake$_{[\text{blood Pb at MSU}]}$ − 0.0864)/0.986, Hauser Lake blood lead concentrations were then adjusted to values that we predicted would have resulted had they been analyzed at NWRC. Adjusted Hauser Lake samples with negative values or values < 0.010 μg/ml were assigned a concentration of 0.005 μg/ml.

Agreement between the uncorrected and adjusted Hauser Lake data sets were quite close (Fig. 2). There was a marginally non-significant difference in the proportion of eagles at Hauser Lake with blood lead concentrations >0.201 μg/ml after adjusting values compared to before adjusting values ($\chi^2 = 3.377$, $P = 0.066$) (Table 2). After adjustment, the difference between the proportion of bald eagles with blood lead concentrations >0.201 μg/ml at Hauser Lake and Galloway Bay approached significance ($\chi^2 = 2.546$, $P = 0.11$) (Table 2). Adjusted median blood

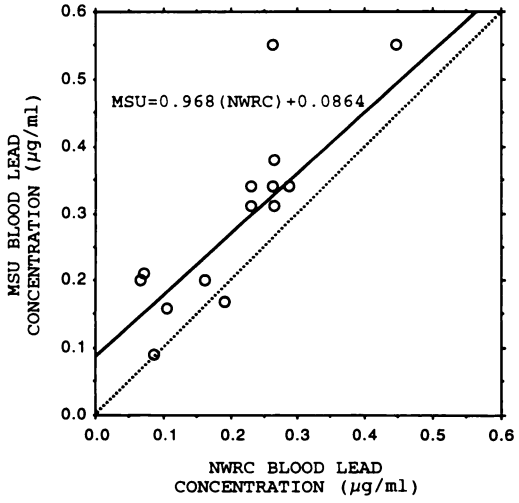

FIGURE 1. Kendall's robust line-fit regression of 14 blood samples obtained from Galloway Bay, Saskatchewan, analyzed at Montana State University (MSU) on portions of the same samples analyzed at the National Wildlife Research Centre (NWRC). The hatched line indicates a 1:1 relationship.

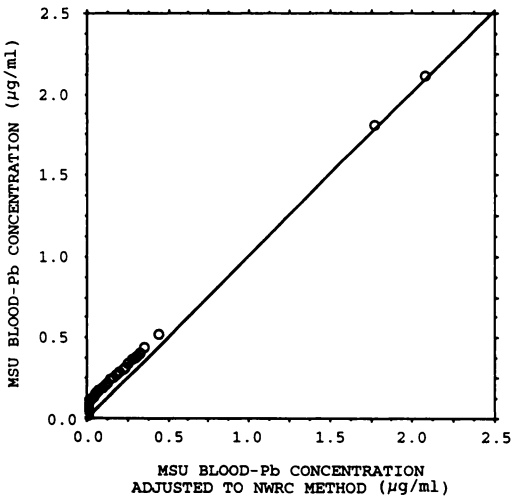

FIGURE 2. Relationship between adjusted and uncorrected Hauser Lake blood lead concentrations. Solid line indicates a 1:1 relationship. See legend of Figure 1 for abbreviations.

lead levels at Hauser Lake were higher than those at Galloway Bay although the difference only approached significance (Wilcoxon-Mann-Whitney, $Z_{\text{corrected for ties}} = -1.617$, $P = 0.053$) (Table 1).

Despite a prediction of a positive association, temporal increases in blood lead concentrations were not observed for all years combined at Hauser Lake for both uncorrected and adjusted data (Kendall's rank order correlation coefficient, $T_{\text{corrected for ties}} = -0.083$, $P = 0.14$ and $T_{\text{corrected for ties}} = -0.0611$, $P = 0.21$, uncorrected and adjusted, respectively). However, a significant positive correlation was detected at Galloway Bay for all years combined (Kendall's rank order correlation coefficient, $T_{\text{corrected for ties}} = 0.133$, $P = 0.027$).

TABLE 3. Prey use by bald eagles at Hauser Lake (HL; Montana) and Galloway Bay (GB; Saskatchewan), as determined by analysis of prey remains and direct observation.

Prey class	Direct observation[a]		Prey remains[b,c]
	HL	GB	GB
Waterfowl[d]	—	41	945
Other avian species	—	—	19
Mammal	—	1	5
Fish	3,800	3	7
Unknown	—	13	13
Total	3,800	58	989

[a] The number of observations of eagles feeding on a particular prey item.

[b] Each individual prey item found was considered a separate event.

[c] Items were identified using skeletal and prepared specimens from the University of Saskatchewan and the Royal Ontario Museum, Toronto, Ontario, Canada.

[d] Anatidae.

DISCUSSION

Subramanian (1987) compared blood lead concentrations determined by two atomic absorption spectrophotometry (AAS) methods similar to those that were used by MSU and NWRC, nitric acid deproteinization and $NH_4H_2PO_4$-Triton X-100, respectively, and concluded the two were comparable and provided accurate and precise results when used under optimized experimental conditions. Nevertheless, we did find consistent differences between the two laboratories.

The interpretation, comparison and the conclusions drawn from avian toxicological data are dependent on several factors, including the analytical methodology, sensitivity of equipment, and the level of detection limits (Pain et al., 1993a). Quality control, both external and internal to the analytical laboratory, is critical to the production of both precise and accurate data (National Research Council, 1993), and can circumvent several sources of error including lead contamination of equipment or a departure from the assumed linearity of calibration curves (Boone et al., 1979). A reduction in the biases associated with interlaboratory variation can be accomplished analyzing industry standard solutions, or by establishing a correction factor between collaborating laboratories, or both (Keppler et al., 1970). Comparisons to standards are commonplace; however, reported interlaboratory calibration is rare (National Research Council, 1993). By calculating a correction factor between our two collaborating laboratories and providing details of analytical methods, we present the reader with additional information from which to base interpretation and conclusions drawn from the data (Pain et al., 1993a).

Based on the work of Pattee and Hennes (1983), we predicted *a priori* that median blood lead concentrations and the prevalence of elevated blood lead concentrations would be higher at Galloway Bay, an area where eagles feed almost exclusively on a hunted population of waterfowl (Table 3). However, results were contrary to our prediction.

We must first acknowledge that our sampling methods may be inherently biased. For example, eagles that have died from or have been debilitated by lead poisoning may have eluded capture due to behavioral or physiological disturbances, and would also have been effectively removed from the population. It also has been suggested that birds with sufficiently

elevated blood-lead may suffer from impaired hunting ability and may scavenge to a greater extent and/or be less selective in their choice of prey (Redig et al., 1983). For example, Pain et al. (1993b) found significantly more highly lead-exposed marsh harriers in carrion-baited traps than in mist nets, suggesting a bias of the former for more heavily contaminated birds.

When compared to other areas, lead exposure at Galloway Bay was quite low despite the apparent potential for high exposure. For example, Hennes (1985) found that bald eagles at a heavily hunted waterfowl refuge in Minnesota had median blood lead values 29 times higher than those at Galloway Bay. Geometric mean blood lead concentrations reported for populations of migratory and resident bald eagles in the western United States ranged from 0.072 to 0.32 μg/ml (Wiemeyer et al., 1989; Harmata and Restani, 1995); the upper value 16 times higher than the geometric mean reported for Galloway Bay (0.02 μg/ml). However, these comparisons must be examined in the context of the methods used to quantify blood lead concentrations and the time of year when sampled.

After adjustment for differences between the two laboratories, Hauser Lake blood lead concentrations were not significantly different from the Galloway Bay concentrations, suggesting that differences in laboratory technique may partially explain the apparent discrepancy between the predicted and observed results. Nevertheless, the lack of differences between the two study areas were opposite to what we had predicted. We offer several reasons which may account for this discrepancy.

First, eagles may endure continual exposure to lead shotshell pellets in latitudes further south of Galloway Bay before arrival at Hauser Lake, resulting in incremental increases in blood lead concentrations during migration. Alternatively, there may be a reliance by eagles on other food sources apart from lead contaminated waterfowl at Galloway Bay, which would reduce the potential for lead shotshell pellet intake; conversely, other local sources of lead may be available at Hauser Lake apart from lead shotshell pellets which would augment the potential for lead exposure. And finally, differences due to storage and handling techniques may have produced falsely elevated or decreased blood lead concentrations.

In general, blood lead concentrations increase rapidly and peak within a week following lead shotshell pellet ingestion (Pain and Rattner, 1988), and depending on the initial amount absorbed, lead concentrations may remain elevated from several weeks to several months following ingestion (Pain, 1996). Previous exposure to lead at locations other than where sampling occurs has been suggested to influence lead exposure prevalence in raptors for a particular area (Wiemeyer et al., 1989; Kramer and Redig, 1997; Pain et al., 1997); this may have occurred at Hauser Lake.

Pattee et al. (1981) suggested that raptors were able to survive occasional exposure to lead shotshell pellets, as it is the continual ingestion and egestion of these pellets that leads to overt lead toxicosis. Blood lead concentrations in bald eagles (Hennes, 1985) and marsh harriers (Pain et al., 1997) have been shown to increase with the progression or cessation of the autumn and winter hunting seasons. Bald eagles at Hauser Lake were sampled on average 3.5 wk after those at Galloway Bay. Thus, it is possible that the higher than predicted lead levels in Hauser Lake eagles was attributable to the fact that they were sampled later in the fall than at Galloway Bay. Migrating eagles can travel on average 150 to 200 km per day (Stalmaster, 1987); thus, movements between migratory stopovers may take only a few days. Therefore, elevated blood lead levels resulting from ingested lead obtained in areas to the north of Hauser Lake may not have sufficiently decreased prior to sampling in Montana, and would have remained above background concentrations,

also resulting in a greater than predicted prevalence of lead exposed eagles. The significant correlation between Julian Date and blood lead concentration may be able to explain the greater than predicted lead exposure at Hauser Lake.

At Galloway Bay, eagles remain for only a relatively short period of time, and only during the early portion of fall migration (late September to mid-November). Minimum estimates of post-capture residency times at Galloway Bay as determined by radio-telemetry ranged from 1 to 25 days with a mean of 8.2 days (n = 31, SE = ±1.2 days) (M. J. R. Miller, unpubl. data). Using similar techniques to estimate minimum residency time of eagles at Hauser Lake, Restani (1997) determined a mean duration of 12.0 days (n = 55, SE = ±1.6 days) with a range of 1 to 45 days. Relative to Hauser Lake, the residency times of eagles at Galloway Bay were only slightly shorter. If Pattee et al. (1981) are correct in their assumption that only chronic ingestion and egestion of lead shotshell pellets results in high lead exposure in raptors, then the short and rapid migration of bald eagles through the southern portion of the Canadian prairies each fall should result in only a small proportion of eagles in the southern prairies of Canada including Galloway Bay, being exposed to lead shotshell pellets or high levels of lead.

Another factor that can potentially limit or increase lead exposure is the undetected reliance on food items less or more likely to contain lead shotshell pellets. For example, at Galloway Bay, eagles used waterfowl dying from avian cholera (a source not likely to contain more lead shotshell pellets than waterfowl killed or injured by hunters) extensively as an alternate food source in 1992 and 1995. Median blood lead concentrations from 1992–95 did not however, yield any significant differences, suggesting other factors besides reliance on cacasses of waterfowl that succumbed to avian cholera (Table 1).

Also, other sources of lead in addition to lead shotshell pellets may be important in contributing to lead exposure in eagles. For example, fishing sinkers are known to contribute to lead toxicosis in common loons (*Gavia immer*) in North America (Scheuhammer and Norris, 1995). Although ingestion of lead fishing sinkers has not been adequately documented in raptors (Locke and Thomas, 1996), the potential for their ingestion exists at Hauser Lake, where eagles feed exclusively on fish. Although direct observation of eagles feeding is the least biased technique used to obtain food habits data (Mersmann et al., 1992), eagles may have additionally fed on other unknown lead contaminated prey items outside of the observational areas, resulting in greater than expected exposure prevalence based on diet (Table 3). Lead fragments from rifle bullets embedded in game animals also may provide a source of lead to foraging raptors (Bloom et al., 1989). Harmata and Restani (1995) suggested that wintering and spring migrant eagles may ingest lead ammunition fragments by feeding on hunter-killed ground squirrels (*Spermophilus* spp.). Eagles were observed feeding on deer carcasses (*Odocoileus* spp.) at Galloway Bay (M. J. R. Miller, pers. obs.), but no attempts were made to determine the presence of lead ammunition fragments.

The final confounding factor which may constitute falsely elevated or decreased blood lead concentrations, are differences due to storage and handling techniques. Factors such as the temperature and duration of storage, the type of container material, and laboratory contamination have been implied as causative agents leading to either artificial increases or decreases in target contaminant concentrations (e.g., Unger and Green, 1977; Méranger et al., 1981). Therefore, we cannot ignore the possibility that the different sample storage and handling techniques used at Galloway Bay and Hauser Lake may have altered blood lead concentrations.

MANAGEMENT IMPLICATIONS

Our data do not support the paradigm that a large proportion of bald eagles feeding on waterfowl in areas of high hunting pressure will be exposed to lead via consumption of lead shotshell pellets in waterfowl on the Canadian prairies. Nevertheless, given the large seasonal movements of bald eagles throughout the North American interior, the potential for waterfowl to carry tissue-embedded lead shotshell pellets throughout their flyways, and the vulnerability of bald eagles to lead poisoning, the possibility of lead exposure to the mid-continental migratory population of bald eagles will continue despite the 1991 nationwide ban on lead shot for hunting waterfowl in the United States (Langelier et al., 1991; Harmata and Restani, 1995). The nationwide restriction on the use of lead shotshell ammunition for hunting migratory birds over waterbodies in Canada commencing in September 1999 may however, begin to further reduce the occurence of primary and secondary lead exposure to waterfowl and raptors, respectively, in North America.

Management strategies for mitigating lead exposure in free-ranging avian populations have focused on reducing the availability of lead shotshell ammunition (U.S. Fish and Wildlife Service, 1986). However, it remains unclear to what extent other sources of lead including fragments from lead rifle bullets, lead fishing sinkers, and lead shotshell pellets in upland game birds may contribute to lead exposure in eagles and other raptors. Demonstrating the importance of other sources of lead, Kramer and Redig (1997) indicated that despite the elimination of lead shotshell pellets for waterfowl hunting, the prevalence of lead exposure in bald eagles has not changed in the midwestern United States since the implementation of the 1991 nationwide ban. This issue must be resolved in order to accurately assess the effect on raptors of banning lead shot for waterfowl hunting in both the United States and Canada.

The necessity for long-term conservation of transborder migratory raptor species, such as the bald eagle, was discussed by McClelland et al. (1994). Most current bald eagle management strategies encourage high reproductive rates, although modelling has shown that extinction rates were more closely related to the survival of individuals already in the population (Grier, 1980). Therefore, survival of migrants is a critical factor in bald eagle population dynamics. Collaboration between government investigators and agencies on toxicological monitoring of transborder migrants such as in this study, in addition to the use of interlaboratory calibration exercises, is strongly encouraged.

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